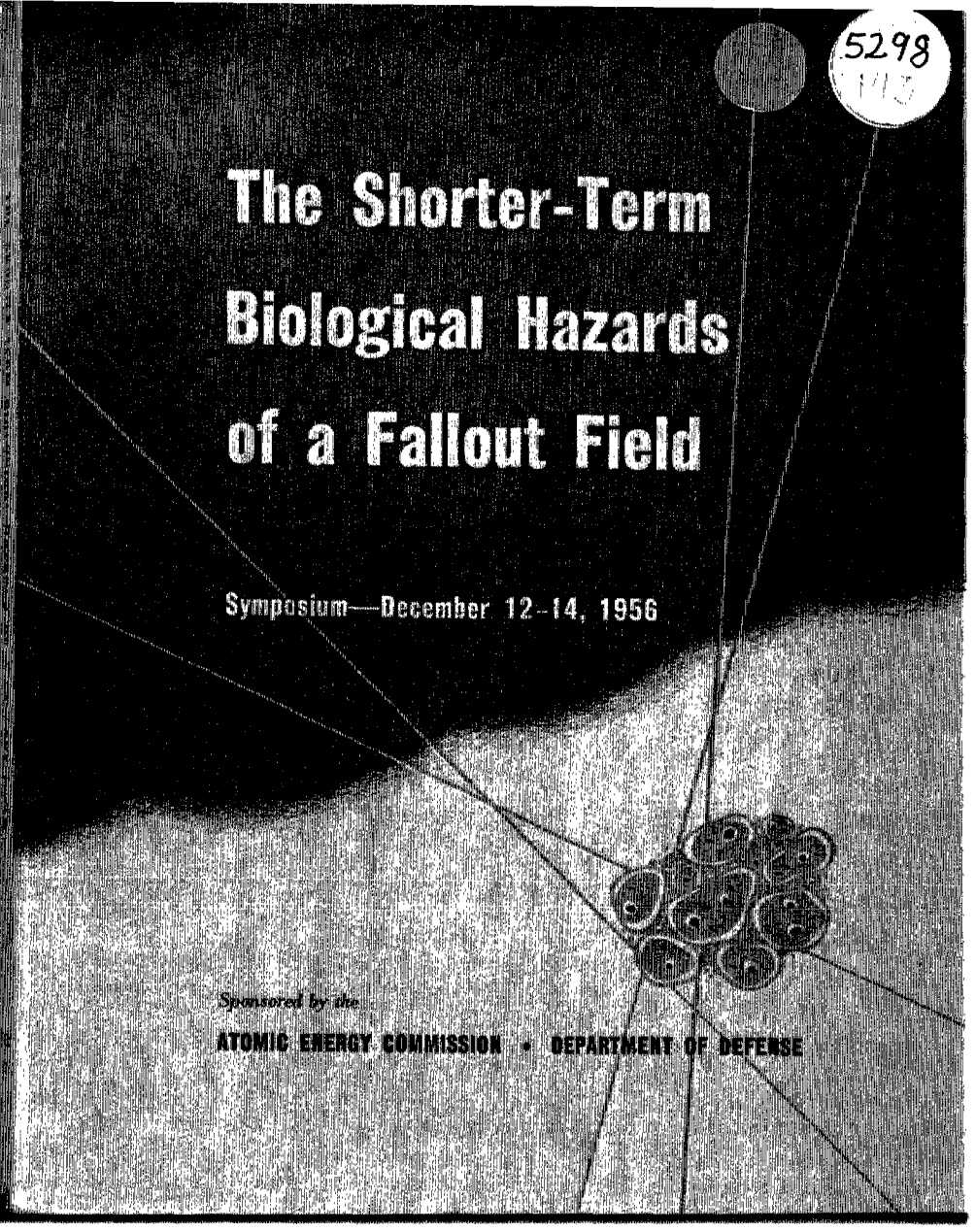
The Shorter-Term Biological Hazards of a Fallout Field

Symposium—December 12-14, 1956



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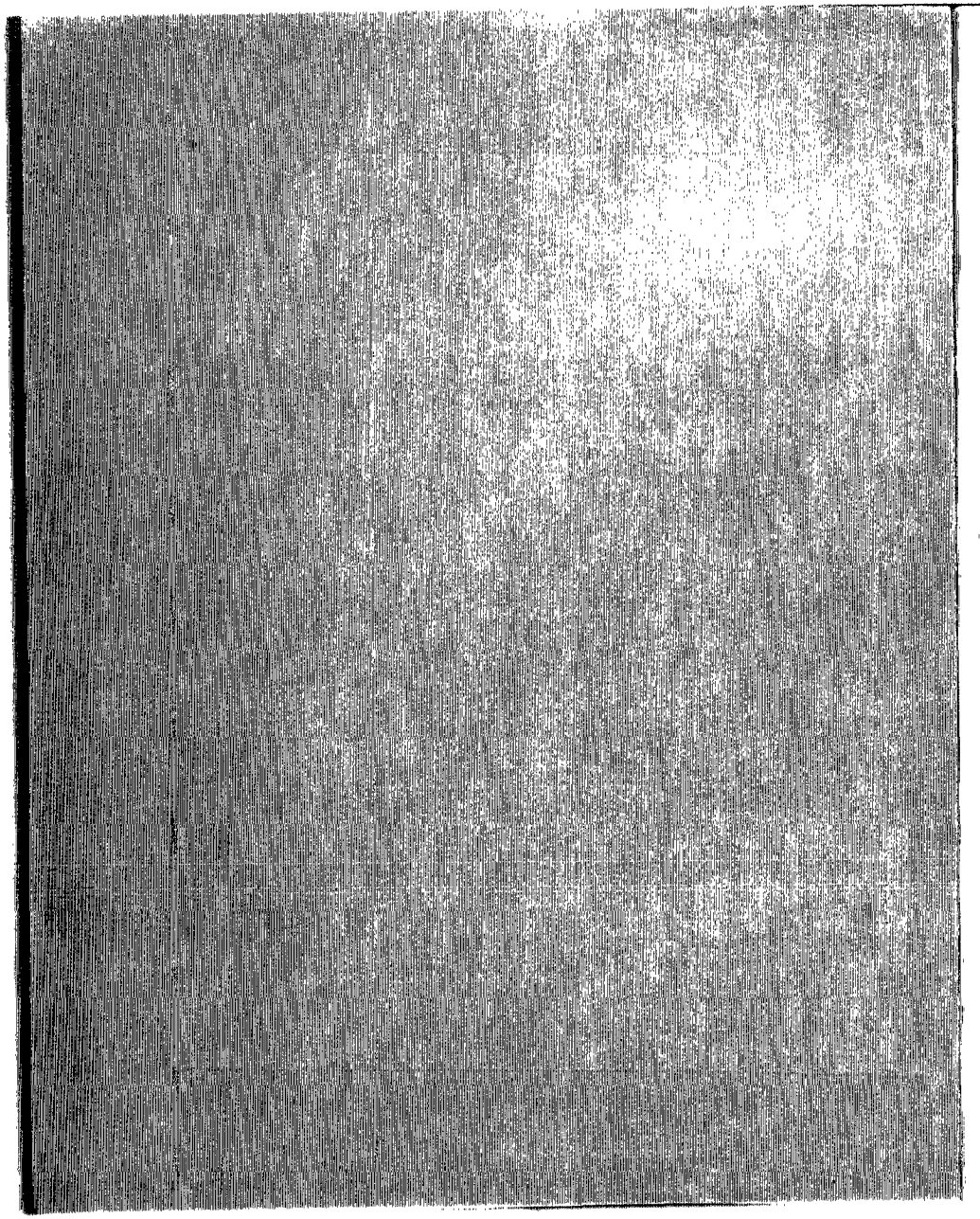

Symposium

The Shorter-Term Biological Hazards of a Fallout Field

Washington, D. C. · December 12–14, 1956

Edited by

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INTRODUCTION

On December 12, 13, and 14, 1956, a *Symposium on the Shorter-Term Biological Hazards of a Fallout Field* was held at the Pentagon, Washington, D. C., under the joint sponsorship of the Atomic Energy Commission and the Department of Defense. The purposes were to review the basic information related to the more immediate effects of fallout, both biological and physical, laboratory and field, and to suggest new research approaches to the many unresolved problems.

The papers were presented under five topic headings:

1. *Decay Constants, Weathering and Shielding*
   Chairman, Dr. Louis B. Werner, U. S. Naval Radiological Defense Laboratory
2. *Gamma Energy Spectra and Geometry Factor*
   Chairman, Dr. Eugene P. Cronkite, Brookhaven National Laboratory
3. *External Beta Radiation*
   Chairman, Lt. Col. James T. Brennen, Walter Reed Army Medical Center
4. *Biological Repair Factor*
   Chairman, Dr. Nathaniel I. Berlin, National Institutes of Health
5. *Internal Emitters*
   Chairman, Dr. Wright H. Langham, Los Alamos Scientific Laboratory

Whatever success the Symposium may have achieved was due to the efforts of the chairmen, speakers and discussants. Appreciation is expressed especially to Colonel Roy D. Maxwell and Commander Thomas E. Shea, Jr., Armed Forces Special Weapons Project, and to Mr. George T. Anton of the Atomic Energy Commission, for their able assistance in planning and conducting the Symposium, and to Mrs. Violet M. McCarthy of the Atomic Energy Commission for her invaluable secretarial assistance.

Topic I

Decay Constants, Weathering, and Shielding

METEOROLOGY—FALLOUT AND WEATHERING

By Lester Machta and Kenneth M. Nagler

U. S. Weather Bureau, Washington, D. C.

INTRODUCTION

Meteorology plays two roles in the study of the biological effects of nuclear radiations on man. First, winds and rain govern the transport of the fission products to man's environment. Second, after settling on the ground, the fallout particles can have their effects modified by rain washing and wind erosion. It is the purpose of this paper to discuss both roles. Research in the Weather Bureau has been devoted largely to the first problem, namely predicting the fallout. Accordingly, in the absence of first-hand research, the discussion of weathering will be more general.

TRANSPORT

There are two aspects of the problem of predicting dosages of radioactivity on the ground. In the first place, the initial distribution of radioactivity in the stabilized atomic cloud on various particle sizes, at different altitudes must be given. Then, with this distribution as the starting point, the particles are tracked downward according to their settling velocity and horizontally according to the winds.

In theory, it might be possible to deduce the distribution of radioactive particle sizes and their specific activities in the atomic cloud from the explosion kinetics, thermodynamics, and available scavenging agents, but in practice, it is necessary to rely on the findings from previous nuclear explosions.

Figure 1 shows, in principle, how this is accomplished. From considerations of the settling speed of the particles and the winds, it is a straightforward process to obtain the locus of points at which particles from a given altitude will fall. These are the radial lines on the chart. Further, from the same information, it is also possible to derive the locus on the ground of particles of the same size (or, really, fall rate), also shown on the figure and labelled according to their diameters in microns. The heavy line shows the path that the 100-micron particle takes in falling from 40,000 feet to the ground. The heavy dashed lines are isolines of observed radiation intensities, in milliroentgens per hour 12 hours after the burst.

Although the actual procedure is more complicated because of the finite lateral width of the cloud, the theory of producing a model of cloud radioactivity can be illustrated from this figure. The procedure is that of associating the amount of radioactivity at a given range of cloud altitude and particle size with the corresponding radiation intensity on the ground. For example, the particles in the shaded area (those between 87 and 100 microns in diameter which were initially between 30,000 and 35,000 feet) have caused an average dose rate of about 50 mr/hr. It is to be noted that this mapping procedure bypasses the determination of the number of radioactive particles and their specific activities. In fact, since the radiation intensity lines used in this type of analysis are obtained for Nevada tests by monitoring the ground with conventional hand radiation-measuring instruments (or, less frequently, by aircraft surveillance), the effect of shielding due to rough terrain is already included in any forecast derived from such information. This technique of preparing forecasts of radiation intensities from cloud models is now used by

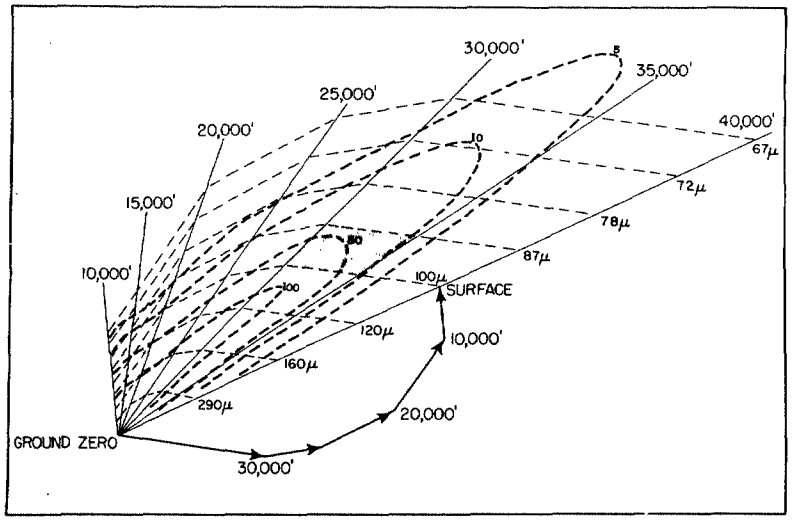

FIGURE 1.—*Hypothetical Fallout Plot.*

practically all groups engaged in this procedure: The Weather Bureau, the Los Alamos Scientific Laboratories, the Rand Corporation, the University of California Radiation Laboratory, the Naval Radiological Defense Laboratory, and others.

The result of the preceding analysis yields a model of a cloud from a specific explosion: A given yield, fission-fusion ratio, and type of burst. The best information, that from Nevada tests, is limited largely to comparatively low-yield weapons tested on towers. A scaling formula is required to refer the dosages to other yields and heights of burst, but such scaling relationships are not yet well understood.

It does not appear to be appropriate to provide the details of each of the models of radioactivity created by different organizations. Rather certain general results will be given, flavored by the Weather Bureau studies of Nevada tests.

The bulk of the radioactivity in the fallout comes from the mushroom head. The ratio of such mushroom to stem material in Nevada bursts is roughly 3 to 1, but this distribution seems quite variable even on shots of similar yield. There probably is a smaller proportion of stem material in bursts in the megaton range. Models proposed by various groups studying fallout have differed greatly in the proportions of activity in the mushroom top and stem. The particle sizes in the stem are relatively larger, that is, the radioactivity is mainly attached to particles greater than about 200 microns. Further, the lower down in the stem, the larger the particles appear to be.

The activity in the mushroom is also nonuniformly distributed in the vertical. From first principles, one may argue that the thorough turbulent mixing in the mushroom will make the amount of radioactivity per unit mass of air uniform throughout this part of the cloud.

Since the air density decreases with altitude, the amount of radioactivity in a given volume of the cloud is much larger near the bottom than at the top. This appears to be borne out by actual fallout data with the one additonal fact that the peak activity seems to be located on somewhat larger particles near the bottom of the mushroom cloud than near the top. In general, both in Nevada and the Pacific, the particle size with the greatest amount of mushroom radioactivity is between 100 and 150μ in diameter with a specific gravity of about 2.5.

APPLICATIONS

Aside from demonstrating how a fallout intensity field is predicted, there are other features which may be of interest in this symposium. For one thing, it is comparatively easy to estimate the time of arrival of fallout, which is necessary in estimating the cumulative dose from a dose-rate measurement. Also, if there is fractionation in the nuclear cloud as a function of altitude or particle size, then it is possible to provide estimates of the heights of origin and the particle size in a given part of the radiation field. As indicated by particle size measurements, meteorological predictions do yield approximately the correct particle sizes; but along with the activity on the predicted particle sizes, there is a disturbingly large fraction of activity on particles too small (even less than 5 microns) to have a significant settling velocity.

EXAMPLES

It may be of interest to consider a typical prediction of fallout in the Nevada Test Site. In Figure 2 the predicted fallout in milliroentgens per hour 12 hours after the burst is shown as the solid lines. The thin dashed lines are the observed after-the-fact fallout isolines in the same units. This case shows the verification of the fallout pattern, using a wind forecast made 2 hours before shot time and a Weather Bureau model of radioactivity. Such forecasts are used by the test management in making the decision whether or not to fire. On Figure 3, the shot time winds are used. A comparatively small decrease in wind velocities has made the fallout pattern shorter and wider than the H–2 forecast. Finally, as shown in Figure 4, a more refined treatment of the wind has been attempted. The time and space changes of the wind along the paths of the falling particles have been incorporated. It is evident that the eastward turning of the fallout pattern in northern Nevada, missed in the previous static wind cases, is better accounted for in this figure.

In Figure 5 the pronounced effect of the wind structure on the fallout pattern is shown. Not only the bearing of the fallout pattern, but also the shape is controlled by winds. Shown in the upper left corner (from a paper by Dr. Gordon Dunning) is a set of idealized dosage lines for the CASTLE BRAVO event, together with the winds which carried the particles eastward. The remaining three figures are the authors' estimate of what the same isolines would look like in different wind situations. In the lower left is a typical winter case of strong west winds, with an elongated fingerlike configuration. In the lower right is a case of light winds, changing from east to west. Note the marked difference in patterns. The upper right, a case of southerly low-level winds and moderately strong upper westerly winds, shows the stem fallout bulging northward somewhat in comparison with the lower left-hand case.

SCAVENGING BY PRECIPITATION

The previous discussions and examples of fallout have considered only the effects of gravity and wind. It should be pointed out that if the airborne debris passes into an area of rain or snow, a very different radiation pattern on the ground may result. There is no good data on the quantitative effects of precipitation on close-in fallout, but it has been observed that most of the radioactivity remote from the test site is brought down by rain.

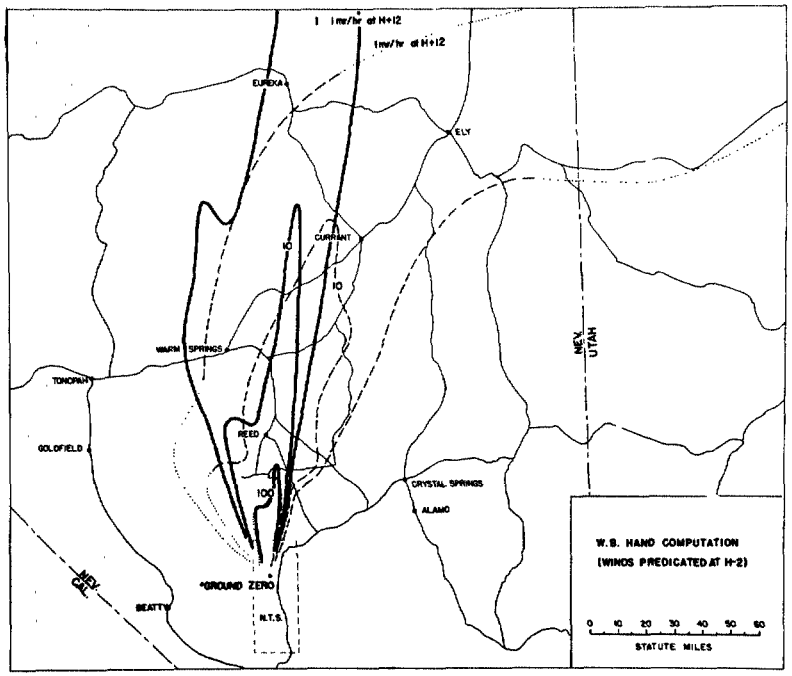

FIGURE 2.—*Sample Fallout Computation—Predicted Winds.*

WEATHERING

The radioactive particles deposited on the ground may be transported or modified in their effect on man by the action of wind and precipitation. Three types of weathering can be imagined:

1. Particles can be washed away.
2. Particles can be blown away.
3. Particles can be covered.

In the first case, particles embedded in rainwater or snow melt can be washed into the ground or carried away by runoff. Light rain or the initial part of even heavy rain tends to soak into the ground—carrying some particles with it. Once the air space in the soil is filled with water, most of the additional rainfall will run off along the surface and into streams—carrying along more of the radioactive particles.

To this must be added the action of raindrops in dislodging the particles by their striking force. With light winds or on level ground this is unimportant but in strong winds or on slopes with as little as 10 percent grade, there will be significant transport.

This qualitative picture combined with the tremendous variability in rainfall can differ

with time and location through a very large range.

The movement of various particles by the action of wind is shown in Figure 6 (from a paper by G. R. Hilst of the Hanford Atomic Products Operation). The table shows that the particles most easily moved by wind are those in the range of 50–500 microns in diameter. A wind of only about 10 knots (measured at the meter level) is sufficient to pick up particles in the 60 to 130-micron level (A. Sundborg, *Geografiska Annaler* XXXVII (1955)), so it is apparent that the important particle sizes as far as radioactivity is concerned are easily moved by moderate winds. Smaller particles may be eroded by a process known as saltation, in which the impact of larger, erodible, particles jars them loose and allows the wind to carry them away.

The particles which are lifted by the wind will, of course, settle elsewhere. However, in general, the importance of this dispersal process is the diluting of the higher radiation intensities. Furthermore, particles initially or subsequently settling into crevices will be the ones most apt to remain. Radiation from these

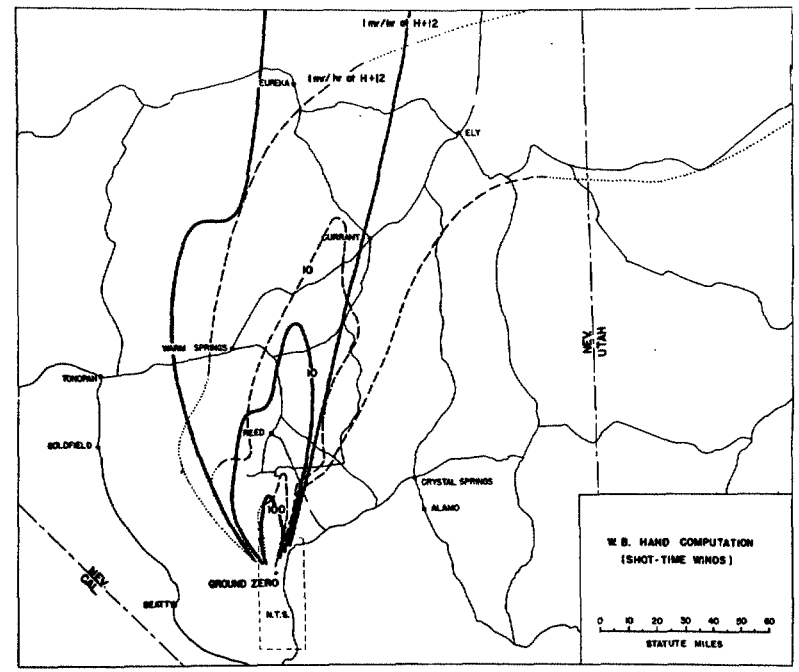

FIGURE 3.—*Sample Fallout Computation—Observed Winds at Test Site.*

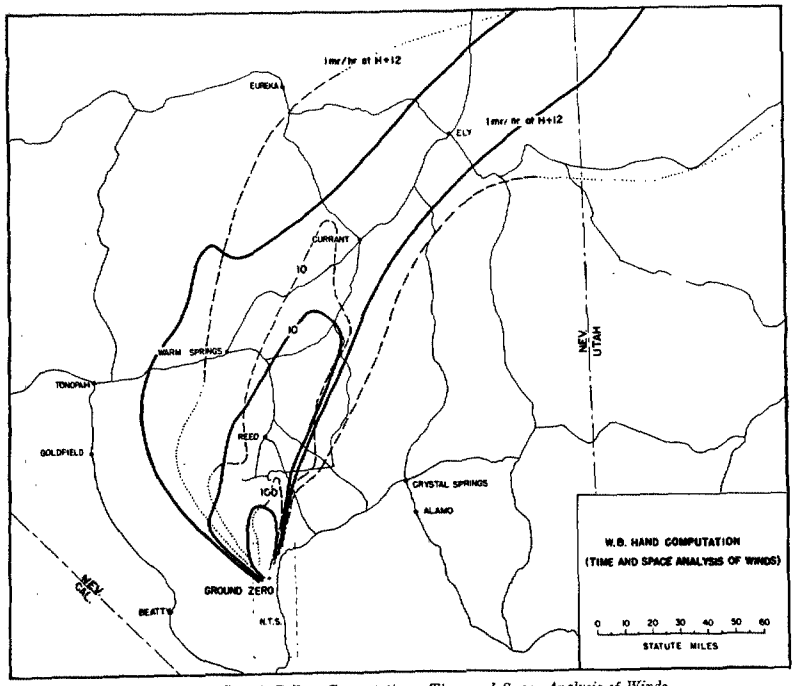

FIGURE 4.—*Sample Fallout Computation—Time and Space Analysis of Winds.*

particles will be partially shielded by the surrounding soil.

Like rainfall, wind speeds show marked variability, so that the erosion of particles by wind action likewise varies over a large range.

Finally, fallout particles may be covered by wind-blown sand with a resulting decrease in radiation. Also, it should be noted that snow cover has a shielding effect.

In conclusion, it might be well to emphasize the very great variability from time to time and place to place in the effects of weathering on deposited radioactive particles. Any "average weathering effect" must be used with caution.

DISCUSSION

K. M. Nagler, U. S. Weather Bureau

Dr. MITCHELL (Rand Corporation). I would like to know why the particles of less than 20 microns are considered non-erodible.

Mr. NAGLER. This concept that large particles are more easily blown away than smaller ones seems unlikely at first thought, but it has been verified experimentally. The explanation, I believe, lies in the way that the wind speed decreases very close to the surface over which it passes. With moderate wind speeds at a few inches above ground, the force of the wind is still strong enough 100 or 200 microns above the surface to move a particle which is large enough to extend into that layer, but it is normally so much weaker just above the surface that it is unable to move a 20 micron particle.

Dr. BORG (Brookhaven Laboratory). Would you care to comment on the importance of different soils in altering the particle distribution that the model that you propose deals with, and the resultant change in the fallout patterns?

Mr. NAGLER. This is something which is really not very well known. The distribution of activity on coral from a Pacific atoll does not seem to be greatly different from that on Nevada sand, but it seems probable that the rubble of a bombed city would lead to quite a different distribution of radioactivity and particle size.

Mr. SPENCER (Bureau of Standards). I have two questions. Have you actually carried out calculations of the time distribution of the fallout?

Mr. NAGLER. Yes.

Mr. SPENCER. Have you determined what percentage of the dose delivered is delivered while the particles are actually falling?

Mr. NAGLER. No, not specifically, but radiation from particles still airborne has been observed. In some Pacific tests, a considerable part of the dose received at some locations was due to particles that were settling very slowly or, essentially, just drifting past in the trade winds. The measurements I have seen from Nevada tests have not indicated that this is an important phenomenon there.

Mr. SPENCER. One other question. Have you plans or have you actually carried out any

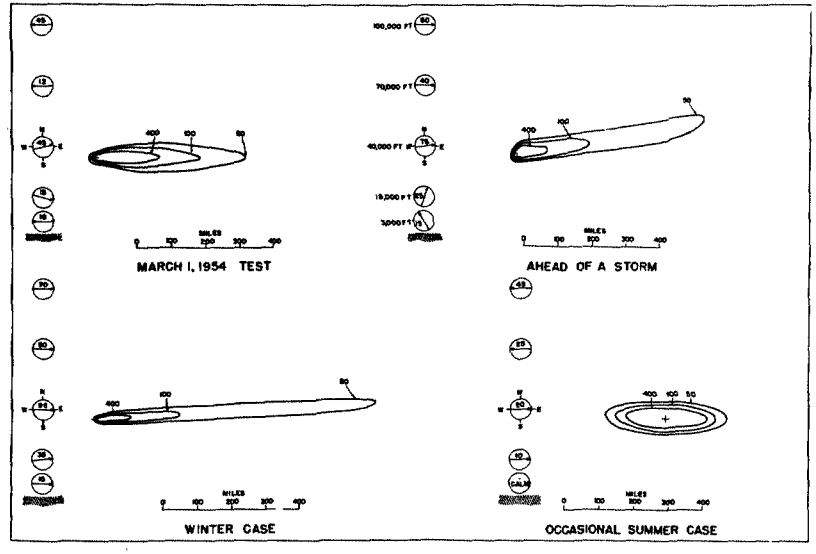

FIGURE 5.—*The Effect of Wind Structure on Fallout Patterns.*

SOIL ERODIBILITY AS A FUNCTION OF PARTICLE SIZE

Diameter (microns)	Relative erodibility
Less than 20	Non-erodible except at wind speeds greater than 50 mph, 6 inches above ground.
0 - 50	Difficult to erode.
50 - 500	Highly erodible.
500 - 1000	Difficult to erode.
More than 1000	Non-erodible except at wind speeds greater than 50 mph, 6 inches above ground.

FIGURE 6.—*Soil Erodibility as a Function of Particle Size.*

studies of local irregularities as they affect the fallout pattern?

Mr. NAGLER. The question of irregularities in the fallout pattern?

Mr. SPENCER. In the ground contour as they affect the fallout pattern.

Mr. NAGLER. We know that these irregularities exist, but to my knowledge, there has been no good quantitative study of them. The observed Nevada fallout pattern which I showed is probably oversimplified, since most of the monitoring runs are made in fairly broad, flat valleys. Some features of rougher terrain must act like snow fences and cause an irregular piling up of the radioactive particles.

Dr. WERNER. Are there any other questions from the audience?

Dr. STANNARD (University of Rochester). Could you give us some very average figures for the fraction of activity on particle sizes too small to settle out?

Mr. NAGLER. The fraction not settling out is quite dependent on the type of burst. We can get an idea of this fraction by considering the measurements of what fraction settles out. For Nevada tower bursts perhaps 5 to 20 percent of the total radioactivity falls out within the first 200 miles or so. For a surface explosion, where a great many more large particles are formed, a much higher percentage may fall down, perhaps as much as 80 percent, within this distance. For an air burst, this percentage falling down is almost negligible. In each case some of the remaining activity reaches ground in a few days, but much is on particles with no significant settling velocity.

Dr. WERNER. Are there any further questions? I would like to ask a question, if I could. What would you expect the effect would be of weathering on redistribution on a rather large-scale fallout field and also on structures?

Mr. NAGLER. On a large scale field the general effect would be to diminish the fallout in the places where it was most dangerous. Weathering would not have a concentrating effect normally. It would tend to distribute it and bring small amounts to other places

which had not been affected. I don't think this would be a very dangerous effect. As far as structures are concerned, the airborne particles would gradually infiltrate into homes and so forth, much as dust does. I don't know quantitatively how important an effect this would be. I would not imagine that it would be too great an effect.

Dr. WERNER. As I recall at Redwing, there were some effects of this sort noticed on the ships that were out in the fallout pattern. Passing through a rainstorm did have quite a significant effect in reducing the level of radiation. However, here there was a convenient waste disposal tank available which would not be available in the case of land installations. For land installation, I would think that perhaps the intensity would not be appreciably decreased by weathering processes. I wonder if you would comment on that.

Mr. NAGLER. I can cite an example of this. We drove in very close to the remains of one tower—that from the explosion on May 5, 1955—just a few days after the test. The levels of radioactivity were rather low on the asphalt pad almost underneath the tower itself. There had been rather strong winds. I would suspect that from smooth surfaces like city streets and buildings the wind erosion of these particles will be rather large. On rough terrain and in vegetation it would be rather small. It must be a tremendously variable thing. It would also depend upon how damp the ground was. On very dry ground, particles may be picked up more easily.

Dr. WERNER. Perhaps we have time for one other question.

Dr. NEWCOMBE (USNRDL). Do you have any information on the possible screening effect of vegetation in determining the amount of fallout on the ground? I have in mind availability to the animals on the floor of a forest, for instance, as contrasted with a desert area or grassland area.

Mr. NAGLER. I believe that Mr. Larson of UCLA has had some data on that, that the leaves of plants do tend to selectively collect small particles, due to the rough structure, the tiny hairs on the leaves, and so forth. So there is actually a collecting mechanism which is probably important in some types of vegetation in intercepting and holding these particles, making them more available to the animals. This is an important effect. In Nevada we don't have the best place for measuring the effect on foliage, but we feel this is an important effect.

Dr. WERNER. Thank you. I believe there is one thing that impresses those of us who have been concerned with the matter of predicting fallout field that Mr. Nagler has been discussing and that is the variability. Even under best conditions as you can see where the impute data is determined, one can still expect rather large variations.

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RADIATION PROTECTION WITHIN A STANDARD HOUSING STRUCTURE

By Robert T. Graveson

Health and Safety Laboratory New York Operations Office, U. S. Atomic Energy Commission

INTRODUCTION

The gamma radiation field measured at a point above a contaminated area is the sum of the individual radiation contributions from incremental surface areas located at various distances from the observer. A relatively small, clean area may be expected to reduce the total intensity by eliminating the close-in contribution. In practical application, a building which does not offer shielding by its walls will still reduce the total dose to an observer due to the existence of this clean area, although the roof may carry activity.

Relatively uniform fallout contamination was present in the vicinity of a one-story building. The gamma measurements made inside this building indicated that an appreciable reduction in radiation intensity may be expected within standard housing structures.

PROCEDURES

The building in Figure 1 is located on a level field. It is located 50 feet from an adjacent building, and its back faces a small earth mound situated 20 feet from the rear wall. The building was constructed of corrugated aluminum siding, approximately $\frac{5}{32}$-inch thick on a concrete slab floor. Two sides are bordered by a porch.

The gamma radiation field in the vicinity resulted from fallout, and the measurements were made 6 days after the shot. A total of 2 inches of rain had fallen in several hard showers subsequent to the cessation of active fallout. It is believed that the roof had been either partially, or completely, cleaned by the rain action.

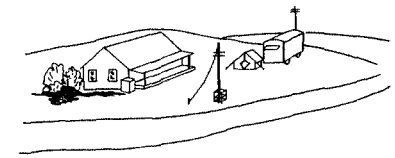

Figure 1.—*Location of Building.*

All measurements were made with a scintillation detector, HASL Type TH-3-C. This unit uses a sodium iodide crystal which has a nonlinear energy response, Figure 2, and is not roentgen equivalent. However, the readings were all made within a short time interval, eliminating energy dependence on the changing gamma ray spectra of the fission products.

DISCUSSION

The radiation readings, at 3.6 feet from the surface, were plotted on a plan view of the building, Figure 3. Readings were taken in the open doorways and behind the adjacent walls, and indicate that there was little effective wall absorption. The section through the building, Figure 4, shows that the outside activity appears constant except in the vicinity of a rain ditch where a sharp increase is noted. The distribution of readings across the open porch

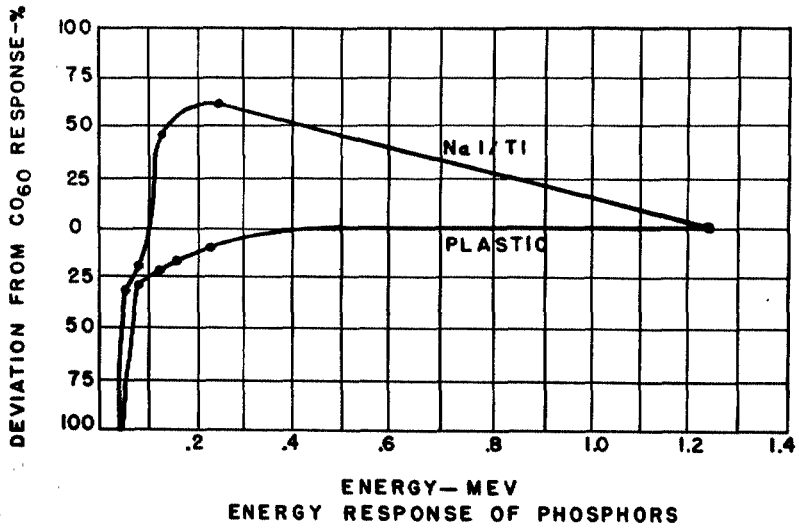

FIGURE 2.—*Energy—Mev, Energy Response of Phosphors.*

appears to confirm the absence of shielding by the walls.

Vertical profiles of radiation intensity vs. height were taken both inside and outside the building.

TABLE 1.—RADIATION—HEIGHT PROFILE

H (feet)	Station 1 (mr/hr)	Station 2 (mr/hr)	Station 3 (mr/hr)	Station 4 (mr/hr)
0.1	20.0	0.6	1.0	
1.0	15.0	1.5	1.2	0.05
3.6	12.0	2.7	2.1	.9
5.6	10.5	5.0	4.0	2.5
7.2	10.0	6.0		3.8

These profiles are plotted in Figure 5. The outside profile agrees with tentative calculations of the response from a large uniform source. The inside profiles do not remain constant though this might be expected, since the slant distance to the active source area does not change appreciably for the height range measured. The increase in intensity might be due to contamination on the roof, however, it is much more likely that a more complete field of view was obtained into irregularities of the outside surface.

CONCLUSIONS

An appreciable reduction in radiation intensity was noted near the center of the building. An occupant might receive between 1/10 and 1/20 of the radiation intensity that would be experienced in open field. The effect of ground unevenness allows even greater reduction in the intensity at points close to the floor. The radiation intensity which would be encountered in a basement would almost certainly be considerably less than indicated here. Thus, by observing precautions, occupants of standard houses may secure considerable protection from fallout, without any modification of these structures.

DISCUSSION

Robert T. Graveson

Mr. HOLLAND (AEC): Doesn't the falling off of the outward curve offer the explanation that as you get higher, you get into irregularities?

Mr. GRAVESON. No. When we are over a uniformly contaminated plane without a clean area between us a high percentage of that dose comes from a very close circle. Therefore even at these heights we are looking into irregularities. As soon as the slant distance becomes relatively large with respect to the height we are looking edgewise at little irregularities of the surface.

Mr. SHAPIRO (NRDL). I would like to comment that both experimental data and theoretical studies at our laboratory indicate what the

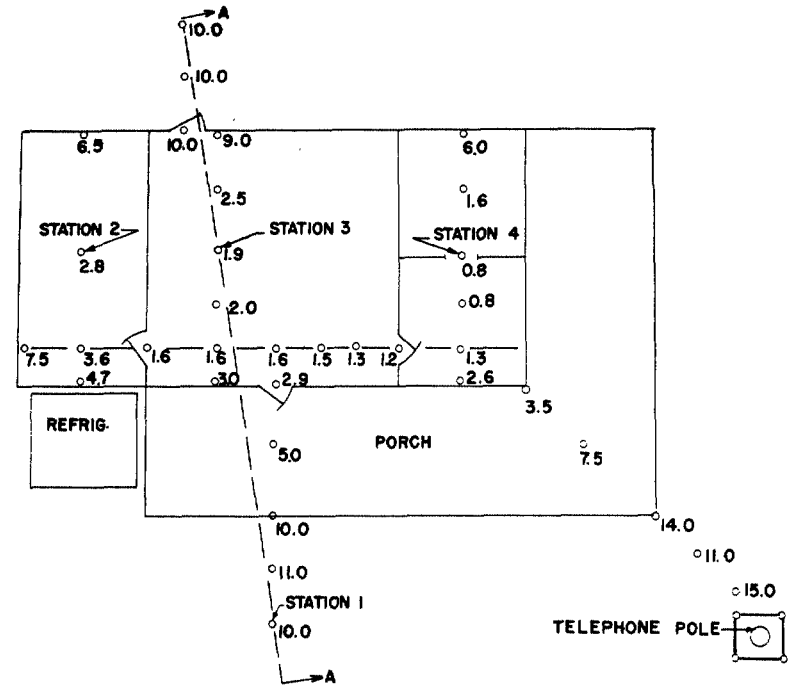

FIGURE 3.—*Plan View.*

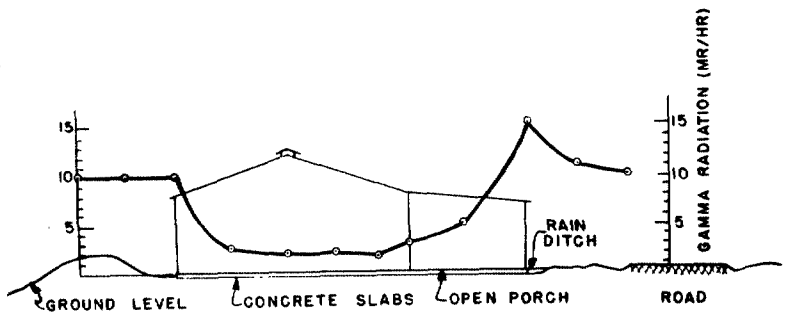

FIGURE 4.—*Section View.*

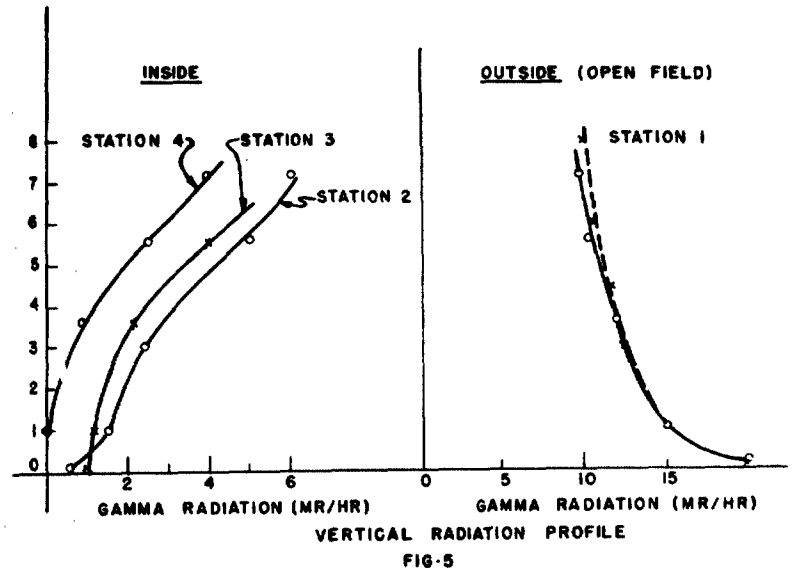

FIGURE 5.—*Vertical Radiation Profile.*

speaker said seems to be quite correct, in that surface roughness affects readings in this way. We have observed that sometimes the reading will rise to as large a height as 30 feet before beginning to fall if the surface is rough enough. The shape of the curves he showed seem to be in agreement with our experimental and theoretical work.

Mr. GRAVESON. This is approached by a thick slab calculation, such as you get over water, where the material is distributed in depth, and then any slant distances are highly preferentially absorbed and the source seems to be almost a monodirectional plane source.

Mr. HOLLAND (AEC). On the other hand, you have an increase of something of the order of 4 or 5 mr per hour in the indoor curve. It seems as though this would have to be reflected. This would be added onto the doses outdoors as you went up and would give you a departure from the theoretical curve, wouldn't it?

Mr. GRAVESON. I am sorry I didn't quite follow what you meant by your question. Would you rephrase it?

Mr. HOLLAND. I assume that the theoretical curve for the decrease of dose rate with height does not include this effect of irregularities?

Mr. GRAVESON. It as such does not. It is a true plane calculation. Therefore, the irregularities could not be serious to give this type of result. The small irregularities could, and it is possible did contribute.

Mr. HOLLAND. If they were of the magnitude shown which is of the order of several—I don't remember the exact magnitude—many r per hour increase, it seems to me they would have shown up on that curve, and particularly since those readings were taken indoors the same effect would probably be larger if existed outdoors. In other words, I am trying to point to the other indication. There must have been contamination on the roof.

Mr. GRAVESON. On the other hand, if we had contamination on the roof, which would match these readings, the contamination density would have been 3 or 5 times higher than the contamination density on the ground. I am not in a very strong position here. I am just saying I am presenting some measurements which I think are very interesting.

Dr. WERNER. Are there any further comments on this?

Mr. RECHEN (Public Health Service). I have a feeling on this that what was missing was a theoretical curve for the indoor type of measurement where you have an uncontaminated slab under the measuring instrument.

Mr. GRAVESON. The theoretical curve we derived was based on the simplest case, that of a smooth plane. This neglected unevenness of the surface. When the major radiation contribution is from outside a clean area, this is not applicable. The Theory for thick slab source is under examination.

THE APPLICATION OF AUTOMATIC WASHDOWN TO PITCHED ROOFS

By A. J. Breslin

Health and Safety Laboratory, New York Operations Office, United States Atomic Energy Commission

The classical approach to estimating the dosage which would accrue to the occupants of a building in a fallout situation is to compute the additive contributions to dose from contamination on the surrounding plane and the building roof. These dose contributions are functions of the geometry and the attenuation properties of the structure. One can conceive of many combinations of these variables in which the relative proportion of the dose originating from the roof would vary over a wide range. However, in typical one-story residences and industrial buildings of moderate size, the variability of this proportion ranges from 20 to 60 percent. The relative roof contribution is greater within a basement than at grade.

Continuous removal of roof contamination during a fallout event would result in an important reduction in dose to persons within a building. This method of dose reduction by itself would not necessarily render building occupants safe from harmful radiation in severe fallout but when applied in combination with measures to reduce the radiation emitted from the plane source, the overall protection could be made quite effective. When measures are taken which reduce the plane source component the roof component assumes a governing significance. Thus applying such a measure in conjunction with others can result in effective overall protection.

It has been suggested that continuous roof decontamination might be accomplished with an automatic washdown system. Such washdown systems have been used successfully on warships. Tests of their effectiveness against simulated fallout and actual field experience have demonstrated that efficiencies approaching 99 percent are achieved. On the other hand, consideration of the effectiveness of washdown on roof surfaces has been primarily one of conjecture. Not only are there uncertainties regarding removal efficiencies but legitimate questions may also be raised concerning water supply, contaminant disposal, effectiveness relative to other countermeasures, etc. One can conceive of circumstances in practice where these questions could be resolved making washdown feasible, provided that the washdown mechanism *per se* is effective.

Certainly a fundamental question is whether or not a water film will transport masses of particulate matter over a sloped surface. The magnitude of contaminant deposition in an event producing lethal dose rates may be milligrams to hundreds of grams per square foot. Large particles have been found in fallout of this intensity. Particle diameters covering a range of 150 to 400 μ with a mean of 260 μ were found on the *Fukuryu Maru*. Analyses of particle distribution in fallout collected on the outer Marshall Islands during Pacific tests indicate size medians of about 80 to 100 microns. It would seem, therefore, that in roof washdown the problem is one of mass transport rather than actual compound formation or simple adsorption which have been shown to be important contamination mechanisms in small scale laboratory tests.

At the Health and Safety Laboratory a pilot experiment was conducted to determine whether

masses of insoluble particulates can be removed from sloped surfaces by water films. The tests were of an exploratory nature and only simulated roofing surfaces were used; the basic objective was simply to test the capability of a water film in moving the contaminant.

A 4′ x 4′ panel was mounted on a tilting easel and set at an angle of 14° (3″ rise in 12″). 1″ x 1″ ribs fixed to the panel in the direction of slope divided the panel into 4 sections of equal area. Test surfaces were mounted within the 4 sections.

Two methods of applying wash water were tested. The first was by means of a header, or distribution pipe, mounted across the upper end of the sloped panel and perforated so as to deliver a distributed water film to 2 of the 4 test sections. The second method of application was by a garden spray nozzle fixed in position above the panel and adjusted to spray two adjacent test sections. Water delivery rates were a function of the characteristics of the delivery systems. They ranged from 0.3 to 1.0 gal/min/lineal foot. The total wash water used per panel in each test run was collected in a funnel placed under the lower edge and emptying into a jar.

The contaminant was simulated by calcium carbonate dust with a particle size range of 44 μ to 150 μ. This material was dusted onto the panel from a 4-foot long shaker held several feet above the surface. During dusting, the shaker was moved back and forth over the panel in the direction of slope to effect uniform deposition.

The tests were conducted in the following manner. The wash water was turned on prior to dusting. A measured charge of dust was shaken onto the 4′ x 4′ panel as uniformly as possible, both in surface distribution and time. The rate of deposition was about 0.3 to 0.5 gm/min/ft^2 and the total deposition was very close to 1 gm/ft^2 in each case. When the shaker charge was exhausted, the water was turned off. The collected wash water was filtered and the solid content weighed. The residual solids on the wetted test sections were carefully removed in 1-foot increments and weighed. The dust on the unwetted test panels was similarly removed and weighed. The latter measurements were used as material balance checks against the measurements obtained from the wetted panels and to determine uniformity of dust deposition.

The results of the tests appear on the following table.

SUMMARY OF WASHDOWN TESTS

Type of surface	Type of washdown	Water Rate gpm/ft of roof width	Avg. removal efficiency %
1. Smooth aluminum	Header		[1] 52.6
2. Smooth aluminum treated with Aerosol O. T.	Header	1.0	[1] 80.4
3. Smooth aluminum treated with Aerosol O. T.	Spray	.3	[1] 97.8
4. Corroded aluminum	Header	.6	[1] 99.6
5. Aluminum painted with flat white alkyd	Header	.6	[1] 97.2
6. Simulated gravel surface	Header	1.0	48.0
7. Simulated gravel surface	Spray	.9	32.5

[1] Average of two values.

The limitations of this series of tests are obvious. However, the simple objective of demonstrating the ability of water to transport sizeable masses of particulates was realized. The results are sufficiently encouraging to justify further investigation.

Certain behavior characteristics exhibited by the washdown system during the tests were noted. On reasonably smooth surfaces, the contaminant was effectively removed wherever the water film was maintained. In test number 1, the film divided into individual rivulets about half way down the slope. The paths of the rivulets were relatively fixed and as a consequence, portions of the test sections were unwetted and uncleaned. As a result, relatively poor efficiencies were obtained. The pretreatment with the wetting agent in the second test was an effort to promote more uniform wetting and was partially successful as indicated by the removal efficiency. However, the spray used in the third test successfully wetted the entire surface and the removal efficiency was again correspondingly improved. The corroded and painted surfaces were conducive to a uniform water film; hence, good efficiencies were realized with the header distribution system. The assumption was made that the spray would perform at least as well as the header on these surfaces and therefore was not tested. From the standpoint of practical application, it is difficult to imagine that even a smooth metal roof would be devoid of surface irregularities and it would appear that a spray would be necessary to achieve the required uniformity of water distribution.

As one might expect, removal from the coarse irregularity of a gravel surface is less effective. Since gravel surfaced roofs are normally flat or only gently sloped, performance may be expected to be poorer than indicated in these tests.

A second question concerning mass transport is associated with a roof washdown system. This involves the capability of a water flow to move the contamination collected in a roof gutter. This aspect was tested qualitatively. Water was passed through a slightly inclined 4-inch diameter cylinder. $CaCO_3$ dust was discharged into the water stream in the cylinder from a vibrating feeder at an arbitrary rate of about 80 gm/min. This was done at water flow rates of 4 and 9 gal/min. As determined visually, all of the dust was transported along the cylinder and out from the end for as long as the feed was continued.

Again the limitations of the test are obvious and need not be enumerated. On the other hand, it has been demonstrated that reasonably heavy amounts of insoluble particulates can be flushed through a gutter. As in the surface washdown tests, the results of this experiment may be taken as justification for further experimentation.

Topic II

Gamma Energy Spectra and Geometry Factor

SHORT LIVED FISSION PRODUCT GAMMA RADIATION

By W. Zobel and T. A. Love

Oak Ridge National Laboratory, Oak Ridge, Tennessee

Fission-product gamma-rays are defined as those gamma-rays emitted by fission-product nuclei, either primary or their daughters, at times measurably later than the fission event. Most of the available information about these gamma-rays is the result of radiochemical experiments which, by their nature, tend to discriminate against short-lived activity. As one is generally interested in the gross fission-product gamma-ray spectrum one has had to construct such a spectrum from the known emitters since experimental evidence was available only for times in excess of 17 hours after the fission event.[1,2] One would expect this synthetic spectrum to be in error at short times after fission, up to perhaps an hour, due to lack of information on nuclides of short half-life. Experiments were therefore undertaken at ORNL to measure the gross fission-product gamma-ray spectrum at short times, i. e., starting at about 1 second, after fission. This paper will present preliminary results obtained so far.

To investigate the energy spectrum and time behavior of the gross fission-product gamma-ray mixture we exposed small samples of highly enriched uranium for short periods in the ORNL Graphite Reactor and withdrew them rapidly to a position in front of the spectrometer. Sample sizes varied from about 2 mg to about 32 mg and exposure times varied from about 0.7 second to about 64 seconds. The experimental arrangement allowed us to measure either the time behavior of different energy groups, or detailed energy spectra.

The experimental results are summarized in Figures 1, 2, and 3 for the two phases of the experiments. It should be emphasized that these results are preliminary only, based on a rather crude analysis of the data which is currently being refined.

The time behavior of 6 energy groups, covering the range from 0.28 Mev to 5.0 Mev, is shown in Figure 1 for times after fission between 1.25 seconds and 1,600 seconds. These curves were integrated to obtain the number of photons/fission and the energy/fission carried off by fission-product gamma-rays in the time range and energy range mentioned above. The results are shown in Table 1.

Table 1

Photon energy range (Mev)	Number of photons/fission	Average energy (Mev)	Energy/fission (Mev)
0.28–0.51	0. 747	0. 395	0. 295
0.51–1.12	1. 225	. 815	. 998
1.12–1.62	. 452	1. 37	. 619
1.62–2.30	. 235	1. 96	. 461
2.3–3.5	. 198	2. 9	. 575
3.5–5.0	. 067	4. 25	. 285
Total	2. 924		3. 233

Detailed energy spectra taken at 10 different times after fission are presented in Figures 2 and 3. The peaks shown represent merely an attempt by the authors to indicate some of the fine structure. No errors have been computed on the experimental points at this time, so that this fine structure is still somewhat uncertain. It should be noted, however, that peaks tend to appear on successive curves, thus lending some credence to their existence. The

[1] D. H. Peirson, AERE–EL/R–155 (Nov. 6, 1954).
[2] Germagnoli and Mongini, *Energia Nucleare* 3, 32 (1956).

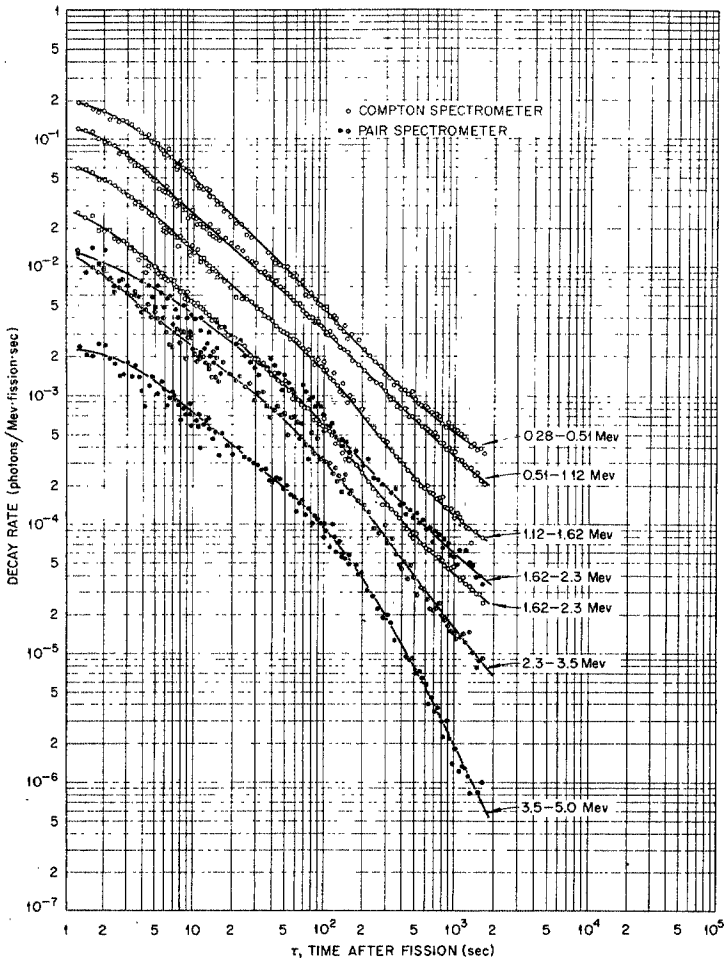

FIGURE 1.—*Fission-product gamma-ray decay rates as a function of time after fission for six photon energy groups.*

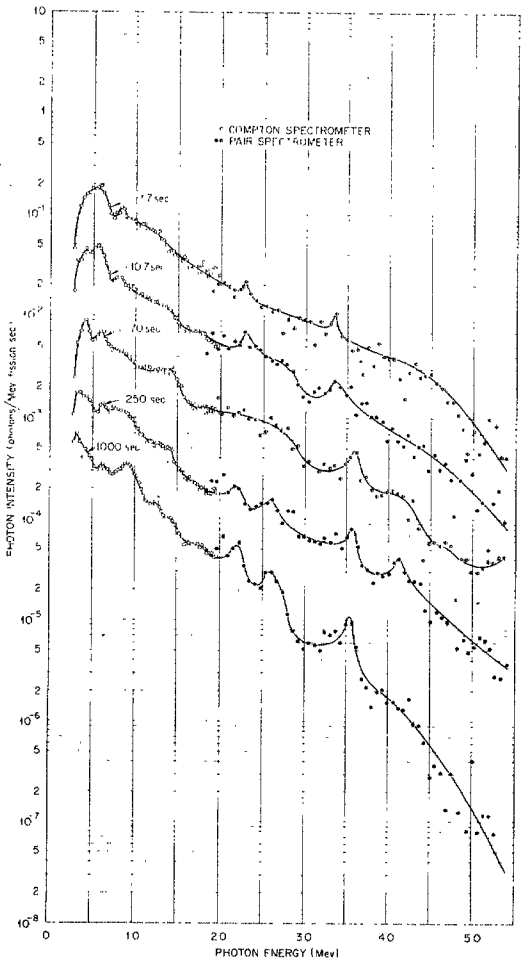

FIGURE 2.—*Fission product photon energy spectrum at 1.7, 10.7, 70, 250 and 1,000 sec after fission.*

448029 O—58——3

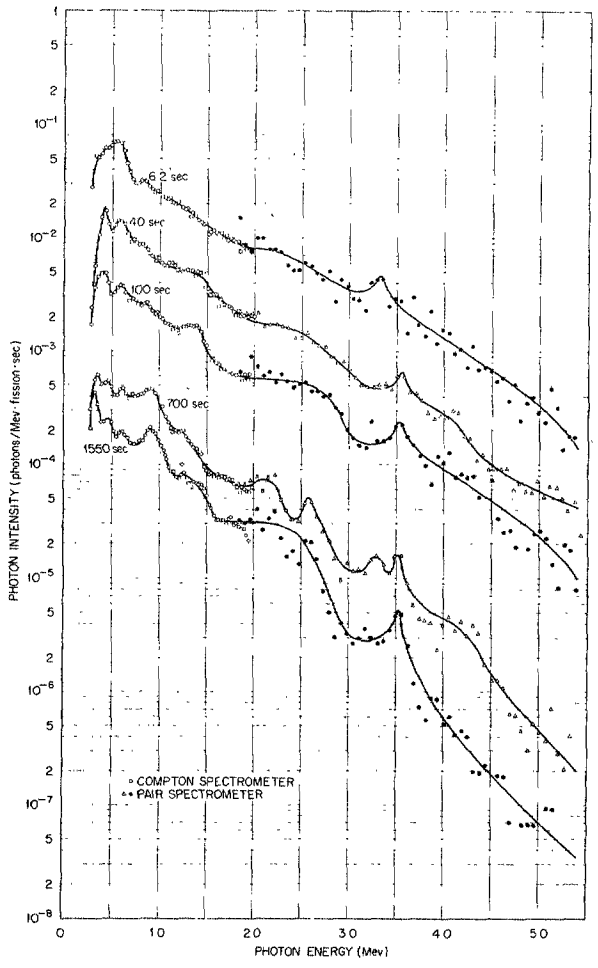

FIGURE 3.—*Fission product photon energy spectrum at 6.2, 40, 100, 700, and 1,550 sec after fission.*

curves were again integrated to obtain the photons/fission-sec and energy/fission-sec, and the results are shown in Table 2.

TABLE 2

Time after fission	Photons/fission-sec (0.28-5.0 Mev)	Energy (Mev.)/fission sec (0.28-5.0 Mev)	Average photon energy (Mev)
1.7	1.62×10^{-1}	1.89×10^{-1}	1.17
6.2	5.59×10^{-2}	6.58×10^{-2}	1.18
10.7	3.88×10^{-2}	4.48×10^{-2}	1.15
40	1.31×10^{-2}	1.53×10^{-2}	1.17
70	7.43×10^{-3}	8.97×10^{-3}	1.21
100	4.16×10^{-3}	4.90×10^{-3}	1.18
250	1.48×10^{-3}	1.68×10^{-3}	1.14
700	5.11×10^{-4}	5.25×10^{-4}	1.03
1000	4.60×10^{-4}	3.76×10^{-4}	.94
1550	2.34×10^{-4}	2.26×10^{-4}	.97

Crossplots of the data taken in one phase of the experiment on those of the other phase are shown in Figures 4 and 5. It is seen that the agreement is quite good.

An additional experiment was performed in cooperation with R. W. Peelle of this laboratory. In this case the equipment used integrated the spectrum over a longer time than was used in the first experiment. A representative spectrum, representing the integral between about 0.7 second and about 3 hours after fission is shown in Figure 6. While it is difficult to compare the results of the two experiments since they cover a different time range, a not unreasonable extrapolation of the curves from the first experiment leads to approximately the same number of photons/fission and energy/fission as was obtained in the second experiment.

The authors wish to express their appreciation to Mrs. G. Estabrook for her aid in the many calculations involved in the analysis of the data.

DISCUSSION

W. Zobel and T. A. Love

VOICE. I wonder if you could describe a little bit the type of radiation used to produce the fission products described in the first talk, the duration of this and the spectrum?

DR. ZOBEL. What did you have in mind? You want the experimental arrangement?

VOICE. Yes. I would like to find out how these fission products were produced.

DR. ZOBEL. Small samples of 235 were sent pneumatically into the graphite reactor and again pneumatically blown out. The time was set by an oscillator which was checked with, if you will, a frequency calculator, so that the time was reproduceable very well. The different bombarding times used, and sample sizes, the sample sizes varied from 2 milligrams to 32 milligrams the combination of bombarding time and sample size was chosen so that we could get the maximum number of counts in the spectrometer—this is coincidence counts—without overloading the central channel too horribly.

Are you familiar with the 2 or 3 crystal spectrometer?

VOICE. Yes.

DR. ZOBEL. We had in the central channel count rates as high as 150,000 counts a second, and we just refused to go above that. As you know, that is bad enough in itself. We ran a maximum of about 120 samples in any given run. This was all the samples we had. This is primarily at the short times, say on the 1.7 seconds, 6.2 seconds and 10.7 seconds runs.

We go to somewhat less samples on some runs, and the statistics got better. Unfortunately when we first started this, the machine ran off 1.7 seconds first, and this is one of the first cases. Does that answer your question?

VOICE. Yes.

DR. BORG (Brookhaven). I would like to ask one further question to follow up the last one. What were the actual bombardment times? How long did the fission occur for the samples that were analyzed 2 seconds later?

DR. ZOBEL. The bombarding times were again variable, varying between 1 second and 64 seconds. We tried to keep it so that the bombarding time and the counting time were less than or equal to the time elapsed in between.

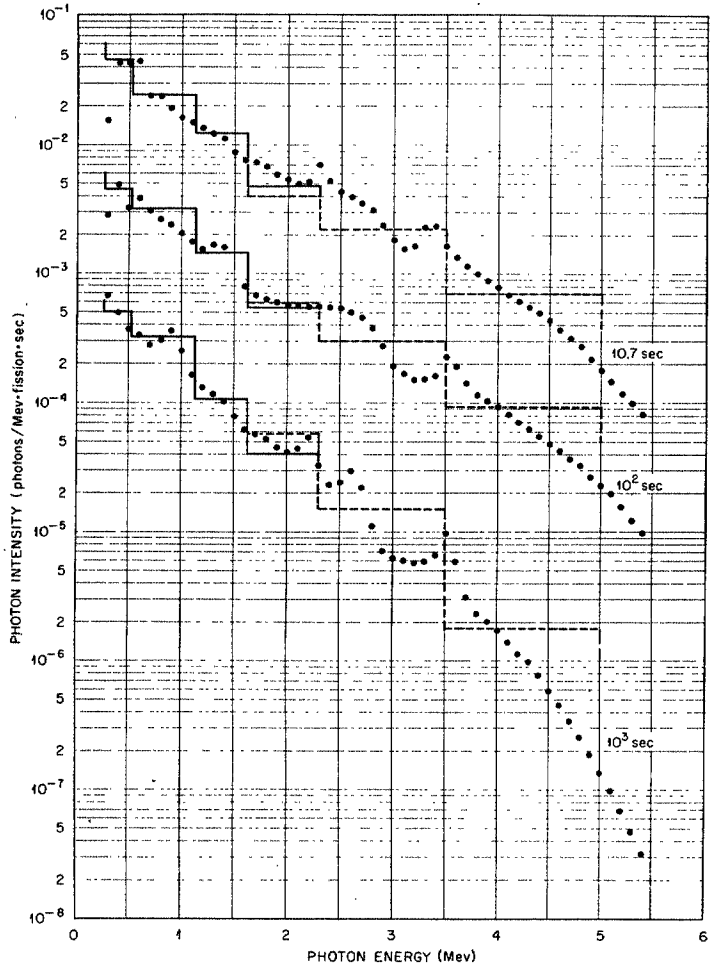

FIGURE 4.—*Fission product photon energy spectrum with superimposed spectrum from time decay study.*

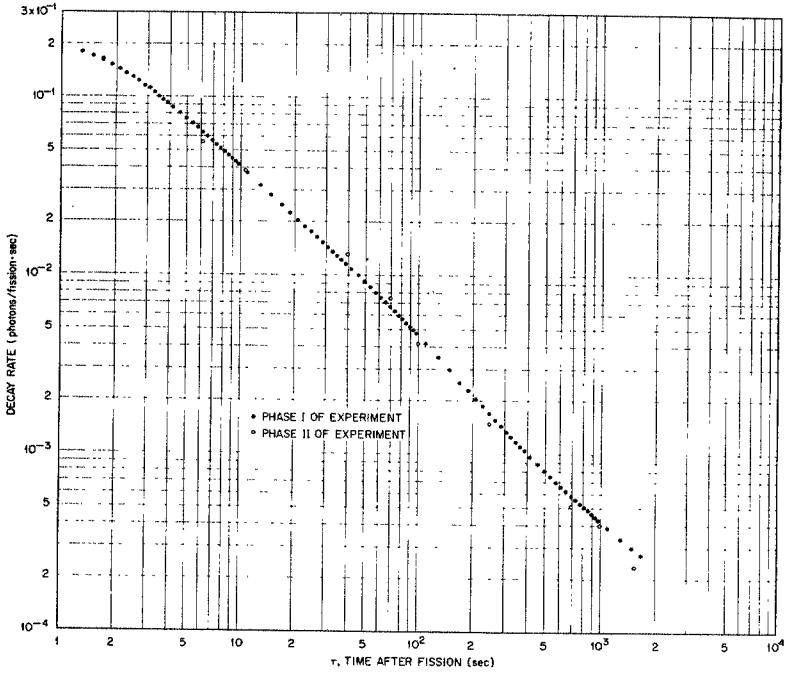

FIGURE 5.—*Decay rate of fission product gamma rays for 0.28 to 5.0 Mev energy range with superimposed points from energy spectra study.*

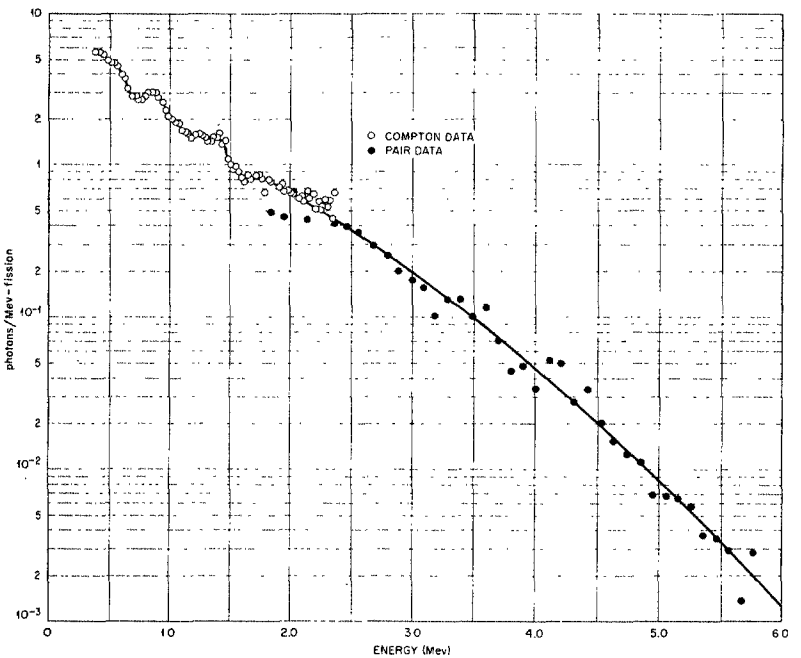

FIGURE 6.—*Typical spectrum of fission product gamma rays from a rotation fuel belt.*

In other words, for the 1.7 seconds run, the equipment was set to bombard 1 second, wait 1 second, count 1 second.

Dr. SIRI (University of California). Was there any reason to believe that the fission product distribution in your test might be different from that encountered in weapons? This would, or course, affect the gamma ray distribution, as well.

Dr. ZOBEL. I am afraid I don't know. If the fissioning process is the same, I see no reason why it should be different. That is for the U-235 fission product. If there is an appreciable amount of fast fission which might lead to different levels, I do not know.

Dr. BORG. Perhaps I can add a small bit to the last answer. The fission product distribution curves after fission in different materials are noticeably different. Fissioning in plutonium as against uranium shifts the curve. Fissioning with high energy neutrons as opposed to low energy neutrons will raise the value of the distribution curve for these who are familiar with such a curve. If some of the nuclides which are emitting important gamma spectra are on the portion of the curve shifted, there might be a significant difference in the gamma spectrum that results.

Dr. SIRI. Is there any possibility of venturing a guess as to the magnitude of this effect? Isn't it likely that the fission product distribution from weapons is more that representing high energy neutron fission?

Dr. BORG. Yes. I must admit I am not much of an expert along these lines, I think the answer is probably yes. I have asked this question myself of Dr. Spence at Los Alamos when he showed me different nuclide distribution curves. He threw up his hands and said it is hard to know. There are so many nuclides that are radiating that the chances are good that these spectra are similar for each case, and that the fast spectrum of plutonium or natural uranium or U-235 is not very different from the slow neutron fission spectrum.

Dr. CRONKITE. Are there any further questions?

Mr. KOCH (Bureau of Standards). How did you evaluate your absolute numbers of fissions and how accurate do you think your numbers are?

Dr. ZOBEL. That right now is the biggest source of error that we can see. The number of fissions is calculated and it is crudely calculated, admittedly. We have taken the weight of the sample, we have taken the cross section, and we have measured the flux. It is calculated on that basis. We expect to make a better analysis of the source and we expect that this will then bring the error down a fair amount. We hope that the final error will be of the order of maybe 15 percent. I don't think we can reduce it below that.

BRIEF SUMMARY OF GAMMA RADIATION SPECTRA FROM RESIDUAL RADIATION SOURCES FOLLOWING A NUCLEAR DETONATION

By R. L. Mather

U. S. Naval Radiological Defense Laboratory, San Francisco, California

Introductory Note.—The following brief summary is extracted from research carried out by members of the Naval Radiological Defense Laboratory, including Dr. C. S. Cook, Mr. F. M. Tomnovec, Mr. W. E. Thompson, Lt. R. F. Johnson, Mr. L. A. Webb, Mr. F. L. Bouquet and the author. The research has been supported by the Bureau of Ships, Navy Department, and in part by the Armed Forces Special Weapons Project.

In the progress of a nuclear detonation both fission product and induced activities are produced in ratios which may depend on the details of the weapon construction and of its environment. Following the detonation these activities are dispersed and fractionated by physical and chemical phenomena influenced by terrain and meteorological conditions. These activities come to rest and create a residual radiation field which can be controlled by shielding. The effectiveness of the shielding will depend on the nature of this radiation field.

This Laboratory has been gathering empirical data on the nature of the radiation fields following various weapon detonations of the past several years from which one can say what the usually observed effects are and can say something about their customary variability.

The distribution of residual activities is typically in two parts, one symmetrical about ground zero and due to activities induced in the soil by the bomb neutrons and to activities deposited there by the fireball, the second elongated and downwind due to fallout from the bomb cloud.

The total gamma radiation intensity from mixed fission products decays with time in a fashion which is the sum of the exponential decays of the various nuclides in the mixture. The decay is usually empirically fitted by a negative power function of the time after detonation. The power is usually observed to be one and a fraction with some variation from shot to shot, from sample to sample of the same shot, from time to time on the same sample, and on the definition of the measure of intensity.

A group of us has been applying gamma-ray scintillation spectroscopy to samples of residual activities from a dozen or so shots exploded in the last three years [1, 2, 3, 4, 5, 7]. A sample of some of our recent data is shown in Figure 1 which is a pulse height spectra of pulses from a 4-inch diameter by 4 inches long NaI(Tl) crystal detector but which, for purposes of this summary, may be called a gamma ray photon spectra. Beneath this spectrum are the spectra of 5 nuclides or nuclide chains which are often identifiable in these spectra. The first 3 are induced activities and the last 2 are fission products. There are, of course, many other isotopes present most of which seem to contribute unidentifiable lines in the region of 200 to 800 kev.

The first two induced activities are prominent in the soil around ground zero. The third can be formed from bomb materials which are in-

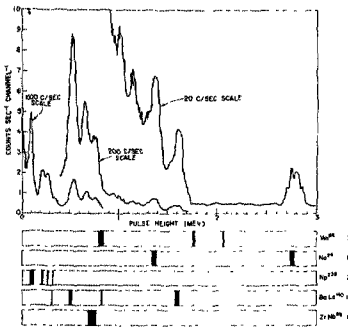

FIGURE 1.—*NaI scintillation detector pulse-height distribution (approximately the gamma-ray photon spectrum) from a typical fallout sample with the gamma-ray line spectra from 5 nuclide or nuclide chains often identifiable in such spectra.*

timately mixed with the fission products and deposited with the fallout from the bomb cloud.

These 5 isotopes tell most of the story in the time span from 2 hours to 3 months following the detonation. Each isotope becomes most prominent (to the extent of 20–50 percent of the gamma ray intensity) in the spectra about 1.5 half-lives after the time of detonation.

At 10–20 hours after the detonation, in those locations where Na^{24} is an important contribution, the very penetrating and biologically effective 2.8 Mev quanta may be found in abundance. Four days following the detonation the 105 kev quanta from Np^{239} generally constitutes a very large fraction of the quanta emitted but these quanta have relatively low penetration and biological effectiveness. Twenty days after, the quite penetrating and effective 1.6 Mev quanta from La^{140} are prominent. Two months after, the 750 kev radiation from $ZrNb^{95}$ dominates the spectra.

There appears to be real differences in the spectral composition of fallout radiation that are of the order of 2 to 1 for the contribution of individual gamma ray lines. These differences have been observed to be (*a*) characteristic of the weapon, (*b*) characteristic of the region of the fallout area, and (*c*) a characteristic of the individual fallout particles. There is insufficient information to make any consistent explanation of these variations.

Following the emission of the quanta by the radioactive nuclides the gamma ray spectrum is considerably altered by Compton scattering from materials which support and surround the residual radiation sources. The scattered radiation is continuous in its energy distribution but always less than the source energy. Usually the energy of the scattered quanta is less than 250 kev regardless of the energy of the source radiation.

Experimental measurements of radiation spectra have been made for the simple case of fallout on level land. The spectrum is a function of the direction of the radiation as shown in Figure 2. This data was taken 9 days following the detonation (when the 105 kev

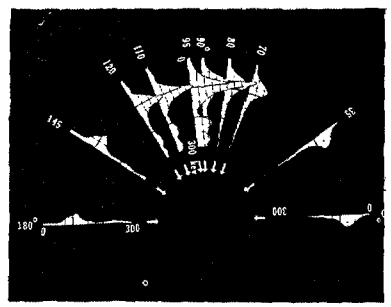

FIGURE 2.—*Experimental 20–300 kev gamma-ray photon spectra observed in various directions above a flat field covered with fallout activities 9 days after the detonation.*

Np^{239} line was very prominent) and shows the 20 to 300 kev region of the spect ..

The pronounced peak in the intensity of 105 kev radiation traveling in the horizontal direction (90°) is due to viewing this uniformly distributed source plane at grazing incidence where the effective radiation source strength per unit solid angle reaches a very large value. The most effective use of shielding in such a radiation field is to shield against radiation coming from slightly below the horizon.

The scattered radiation is more uniformly distributed in direction and for angles above the horizontal (<90°) the radiation is all from scattering. The 75 kev peak in the spectrum of radiation scattered down by the air is due to the degradation by multiple scattering of the 105 kev Np^{239} line.

The two extreme radiation spectra revealed by this information are (*a*) a field of 2.8 Mev quanta above induced soil activities near ground zero 10–20 hours after the detonation, and (*b*) the 40–100 kev air scattered radiation entering a freshly dug foxhole in a fallout area 2–10 days after the detonation.

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DISCUSSION

R. L. Mather

Mr. GRAVESON (New York Operations). Do you know whether the sodium 24 was primarily from your Teapot data, or have you encountered this elsewhere?

Mr. LARIVIERE. I don't know if it is Teapot. I am sure it was Nevada.

THEORETICAL CALCULATIONS OF THE GAMMA RADIATION SPECTRUM FROM INITIAL AND FALLOUT RADIATIONS OF NUCLEAR WEAPONS

By D. C. Borg

Brookhaven National Laboratory

INTRODUCTORY REMARKS

In another paper to be presented by Dr. Bond later at this conference emphasis will be given to the dependence of whole-body radiation effects upon depth dose factors. Since penetration of ionizing radiations into targets depends upon the energy of the incident photons as well as upon the geometry of exposure, development of spectral information concerning fallout gamma radiations becomes highly pertinent to the calculation of biological responses to be expected from fallout gamma fields.

However, questions may well be raised as to the pertinence of discussing *initial* gamma radiation spectra at a conference on fallout. In answer to this several considerations may be cited, to wit:

The same theoretical treatments apply to both initial and fallout geometries, so support for the former case by relatively good data increases the validity of conclusions and insights derived from the application of a companion approach to the latter geometry.

In actual fact the constancy and certainty of the input data for the initial gamma radiation case are far superior to those for the fallout gamma case. Furthermore, actual field measurements of gamma air dose correlated with weapon parameters are vastly more accurate and more numerous for initial bomb gamma radiations than for fallout gammas.

In short, the theoretical treatment that can give insight into aspects of fallout gamma radiations can best be checked experimentally for the initial bomb gamma radiation case.

Moreover, a parallel situation exists with respect to radiation damage criteria for man: namely, that the data correlated with initial bomb radiations and their laboratory counterparts are far more numerous and better documented than are those for fallout radiations. Thus, there is practical radiobiological significance in understanding the mechanisms of initial bomb radiations: so that the radiobiological dose-response criteria derived from them will be properly adjusted for application to fallout radiations or to other conditions.

This concept is generalized and developed more completely in Dr. Bond's companion paper.

THEORETICAL METHOD

The general nature of the theoretical method applied to bomb gamma radiations in this paper may be summarized briefly.

Gamma ray propagation in an infinite medium can be defined by a partial linear integro-differential equation—that is, a so-called "transport equation." [1, 2, 3]. This equation considers all the major interaction processes between gamma photons and the medium: namely, photoelectric absorption, Compton interactions with associated generation of secondary photons with their altered angular distributions, and positron-electron pair production. The equation that represents this can account for the

distribution of photons according to both energy and direction as a function of position in the transporting medium; and the equation can be set-up for several source geometries of interest [2].

Extensive recent calculations have been made with this equation using the method of moments as developed by Spencer and Fano [1], wherein the flux function of the transport equation is expanded into a series of Legendre polynomials. The first few of a series of linked integral equations related to these polynomials have been solved numerically on the NBS "SEAC" calculator for gamma sources of various initial energies in various media. From these solutions, in turn, differential spherical or so-called 4π energy spectra and integral energy or dose spectra at different distances from the source have been obtained. Then by superposition of solutions, spectra have been determined from sources composed of more than one energy.

Details of this method, its solution, and its application have been reviewed in unclassified AFSWP document 502–A [2].

APPLICATION

The application of this gamma ray transport equation and its solutions to bomb radiations has been dealt with most satisfactorily for the initial gamma radiation.

Here the problem resolves itself into the determination of the proper source input data for the transport equation when all that is known about the bomb *a priori* is its presumptive yield plus certain parameters relating to its nuclear fuel composition and internal geometry.

The important gamma radiation sources from bombs are the cloud of radioactive fission products and the radiative capture of bomb neutrons in external materials, particularly nitrogen of the surrounding air. These sources are often referred to as the fission product gammas and the nitrogen capture gammas, respectively.

The general theoretical treatment of gamma photon propagation from an effective point source in air takes the form shown in Figure 1. This figure shows the differential energy spectrum received from all directions 1,000 yards distant from a source of 2 million electron volts, that is: 2 Mev, gamma photons in air. Although the units along the ordinate may be taken as arbitrary units of energy, the *shape* of this spectrum shows that at 1,000 yards much of the gamma energy has already been degraded to less than the 2 Mev source energy.

These same conclusions can be expressed also by an integral energy spectrum; or after conversion to air dose by proper consideration of the true coefficient of absorption of air as a function of photon energy, they may also be expressed by an integral *dose* spectrum, as seen in Figure 2. In this case the ordinate represents the fraction of total energy or dose delivered by photons whose energy is less than a given value, as indicated by the abscissa. For example: 1,000 yards away from a 2 Mev gamma source, one-half the air dose is delivered by photons of energy less than 1 Mev.

Figure 3 presents the differential energy spectrum of the same 2 Mev source, now seen from 3,000 yards away. Compared with the spectrum at 1,000 yards (fig. 1), even further degradation has occurred—due mostly to Compton scattering events. Thus the *un*attenuated 2 Mev source photons are relatively even less prominent at 3,000 yards from the source.

By extending solutions of this type to a number of different source energies at several distances, interpolation curves can be drawn up, plotting fraction of dose delivered by photons of a given energy against source energy.

Figure 4 shows an example of interpolation curves at 1,000 yards from a point isotropic source. For example: For a gamma source component of 6 Mev, 35 percent of the dose at 1,000 yards is delivered by photons of 4 Mev or less, 56 percent by scattered photons of all energies, and the remainder by unscattered 6 Mev photons. Such interpolation curves enable the preparation of crude dose spectra for arbitrary source energies.

In Figure 5 are similar interpolation curves for 1,500 yards. One can see that for any given source component the fraction of dose delivered by scattered photons or by photons up to any given energy increases with increasing distance. This is also suggested by the differential dose spectra for a monoenergetic 2 Mev source seen in Figures 1 and 3.

In Figure 6, finally, are interpolation curves at 3,000 yards. At this distance even the very most energetic gamma sources deliver most of their dose through scattered photons. For example: even for a 10 Mev source component, 66 percent of the dose derives from scattered photons, compared with a comparable figure of 41 percent at 1,000 yards. In common technical jargon the dose build-up factor is defined as the total dose delivered by *all* photons derived from source photons of a given energy, divided by the dose delivered by un-

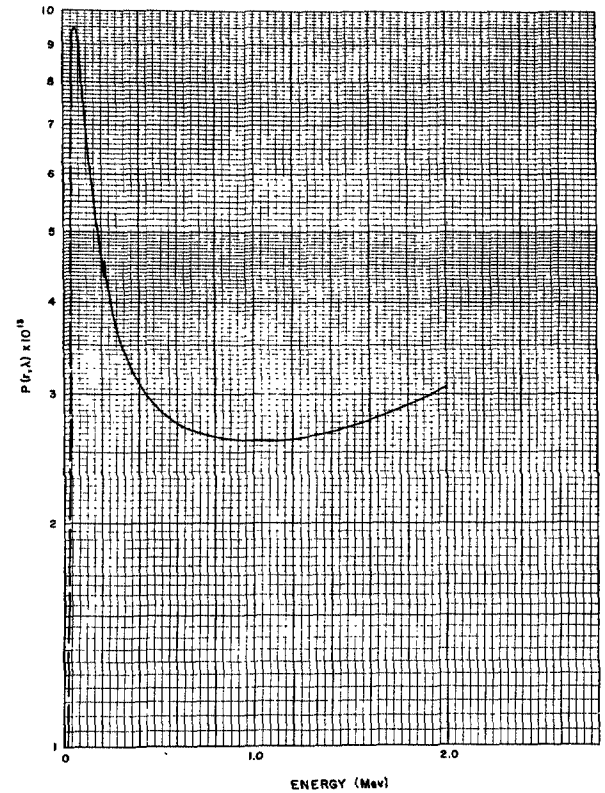

FIGURE 1.—*Point isotropic source, differential energy spectrum at 1,000 yards, E_0=2 Mev.*

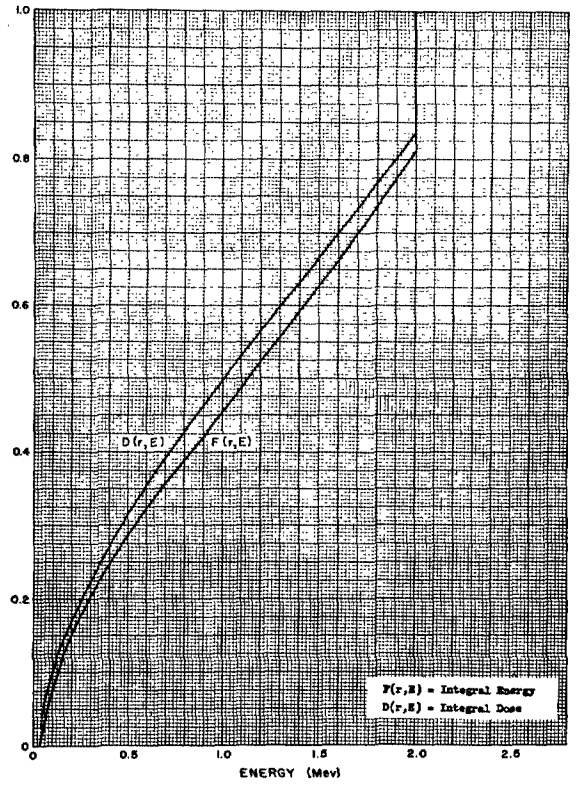

FIGURE 2.—*Point isotropic source, integral energy and dose spectra at 1,000 yards, E_0=2 Mev.*

attenuated photons only. In other words, then, the dose build-up factor can be determined theoretically by this method. Thus the build-up factor for a 10 Mev source 3,000 yards away in air would be one divided by one minus 0.66, or 2.27.

By using such interpolation curves and combining solutions for several energies, one can determine spectra from *poly*energetic gamma sources, thus beginning to approach the case of an actual bomb gamma source. As an example, Figure 7 presents an integral dose spectrum 1,000 yards away from an arbitrary source made up of 40 percent 1 Mev photons, 15 percent 2 Mev, 6.7 percent 3 Mev, and 2.5 percent 4 Mev photons.

INITIAL BOMB GAMMA RADIATIONS

In order to calculate a solution for an actual weapon source, however, the emission spectra of the previously mentioned fission product gammas and nitrogen capture gammas must be known, and the absolute abundance of each of these sources must be weighted according to the pertinent weapon parameters in order to determine air doses and spectra as a function of distance. These considerations are treated in detail in the classified literature and will not be developed here. In substance, however, appropriate fission product and nitrogen capture

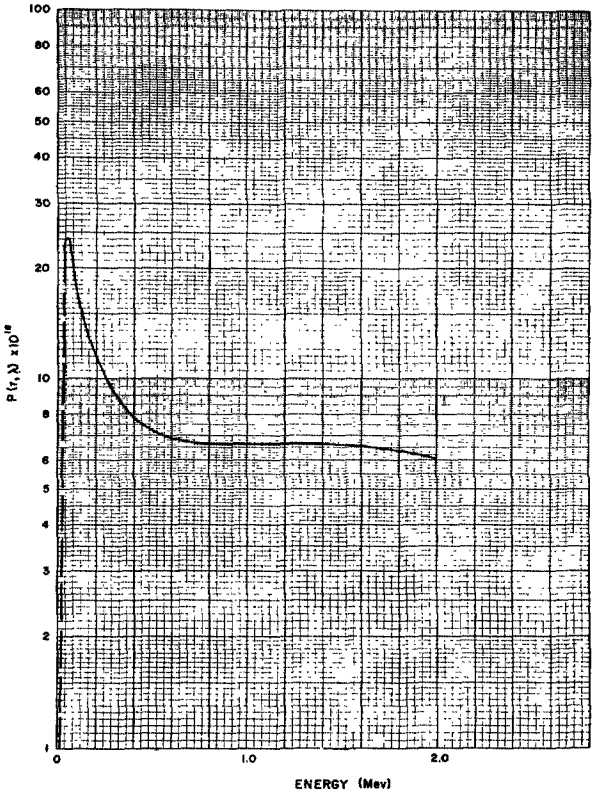

FIGURE 3.—*Point isotropic source, differential energy spectrum, at 3,000 yards, E_0=2 Mev.*

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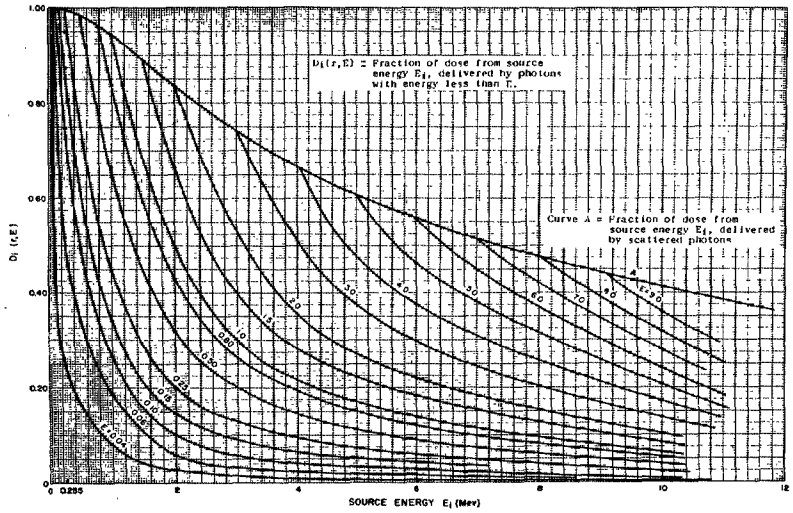

FIGURE 4.—*Point isotropic source, interpolation curves, at 1,000 yards.*

gamma spectra are normalized to known weapons parameters, and then treated in the manner previously developed.

In Figure 8 is a representative fission product source spectrum used for these calculations. At the times of gamma ray emission which are of interest from the point of view of initial bomb radiations, the fission product gamma source spectrum can be characterized by an exponential expression as is seen here. (See also reference 4 and Dr. Zobel's paper at this conference). It appears that the source spectrum corresponding to $e^{-1.16E_\gamma}$ in the figure is the best one to use: that is, the middle curve. For application to the transport equation solutions the continuous fission product spectrum presented here can be approximated by a discrete distribution, if desired. Note that on the logarithmic chart of the figure, the vast majority of fission product photons leave the source with energies of only a few Mev or less.

The decay scheme of excited N^{15} is shown by Figure 9. [5]. The column listing relative numbers of photons defines the source strength of the nitrogen capture gammas. In contrast to the continuous fission product spectrum, the nitrogen capture gamma source is seen to consist of relatively few discrete types of photons, many of which are exceedingly energetic, at around 10 Mev or more. It may be anticipated that the so-called "hard" or energetic nature of this nitrogen capture source will be reflected in the gamma dose spectrum at various distances from a nuclear device, and this will be further indicated later.

Using the appropriate normalization factors, initial gamma spectra can be calculated at various distances from actual nuclear weapons. As a representative example, a fairly typical small yield weapon might generate gamma dose spectra in air of the following nature:

Figure 10 shows the differential dose spectrum

at 1,000 yards, as represented by a histogram chart plotting fraction of total dose within the energy range Δ E against photon energy. Note that the spectrum is a "hard" one with prominent high energy contributions. Despite some degradation through 1,000 yards of air, many of the discrete source components of the nitrogen capture radiations are still prominent.

The spectrum of Figure 11 has been calculated for 1,500 yards, a range of some biological interest for weapons in this yield range. Two salient features are apparent in it:

1. This is a very "hard" or energetic spectrum indeed. Although these data are calculated for spherical or 4π geometry, the tendency of very energetic photons to scatter

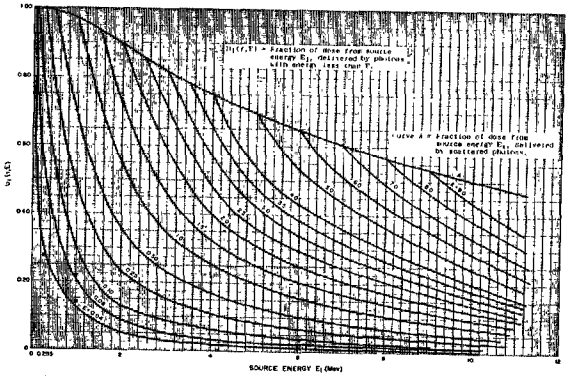

FIGURE 5.—*Point isotropic source, interpolation curves, at 1,500 yards.*

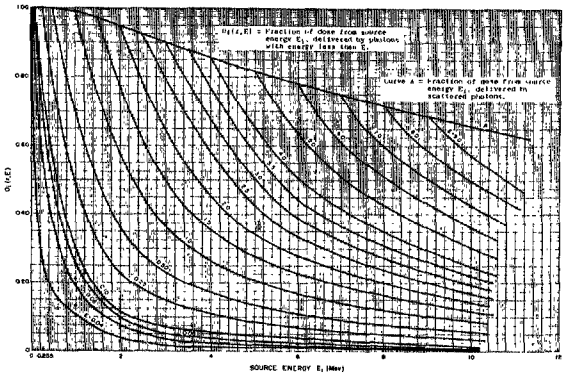

FIGURE 6.—*Point isotropic source, interpolation curves, at 3,000 yards.*

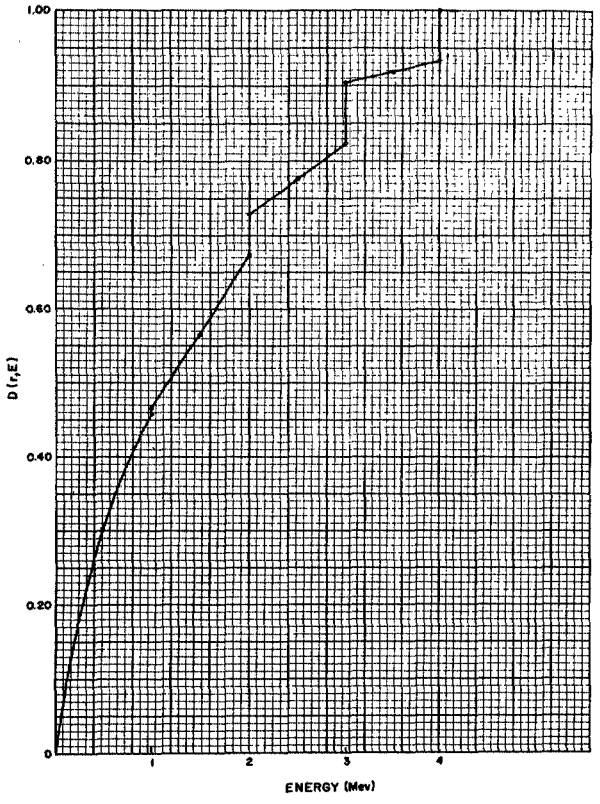

FIGURE 7.—*Sample polyenergetic point isotropic source, integral dose spectrum at 1,000 yards.*

preferentially in a forward direction would suggest that the spectrum incident on the presenting surface of any real target—such as a human torso—would be even "harder" than that shown here. This spectrum is vastly more energetic than conventional laboratory sources or than fallout radiation; and insofar as this greater hardness may affect depth dose curves or relative biological effectiveness, biological data derived from exposure to initial bomb gamma radiation should be suitably corrected before being applied to fallout or other conditions. (See Dr. Bond's paper, this conference.)

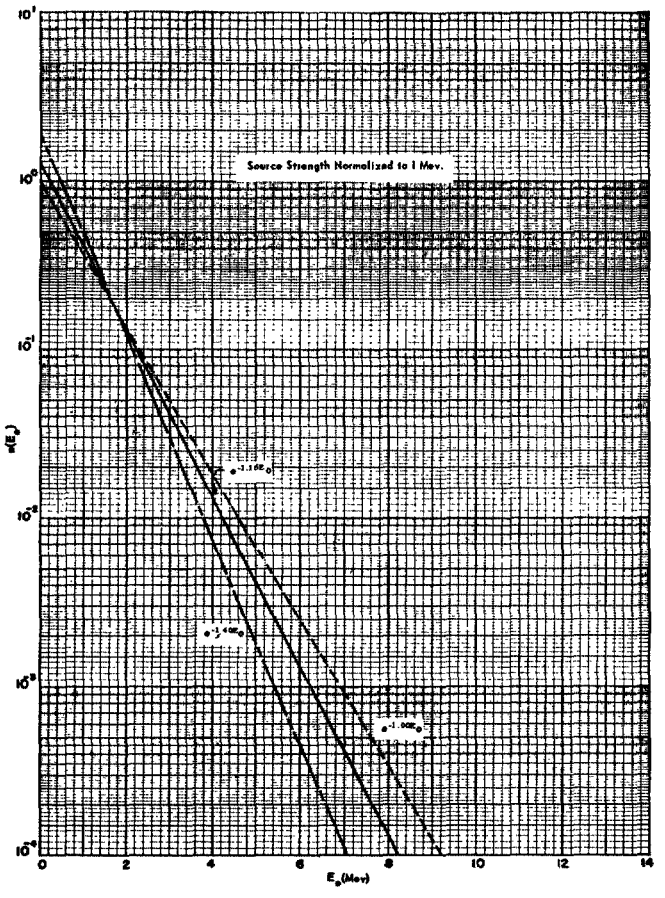

FIGURE 8.—*Fission product gamma source spectra.*

Decay Scheme of N^{15}

Photon	Energy E_i(Mev)	Relative Number n_i
(1)	10.816	1.0
(2)	9.156	0.09
(3)	8.278	0.19
(4)	7.356	0.56
(5)	7.164	0.19
(6)	6.318	0.90
(7)	5.554	1.5
(8)	5.287	2.3
(9)	4.485	0.8

$\sum_1^9 n_i E_i$ = 48.46 Mev $\sum_1^9 n_i$ = 7.53

$\Sigma' n_i$ = 5.23, excluding cascade photons (6) and (8).

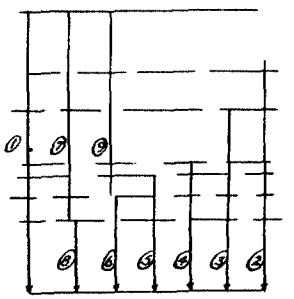

FIGURE 9.—*Decay scheme of excited* N^{15}.

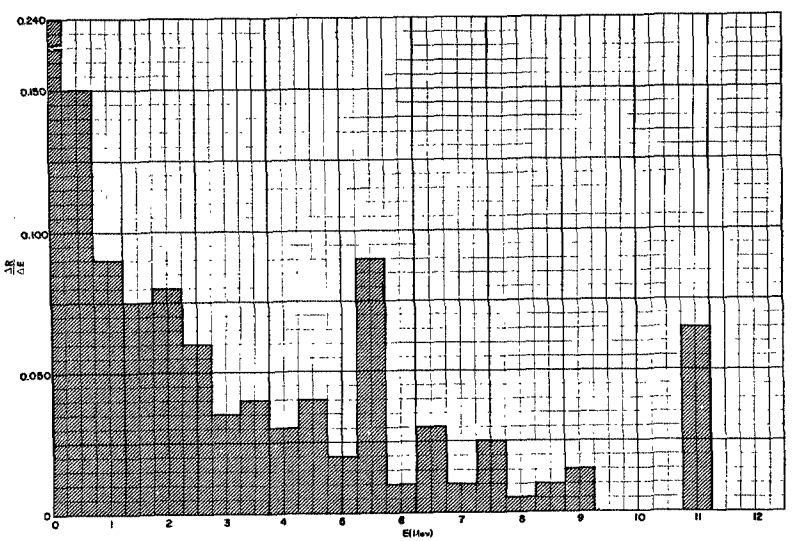

FIGURE 10.—*Initial gamma differential dose spectrum at 1,000 yards.*

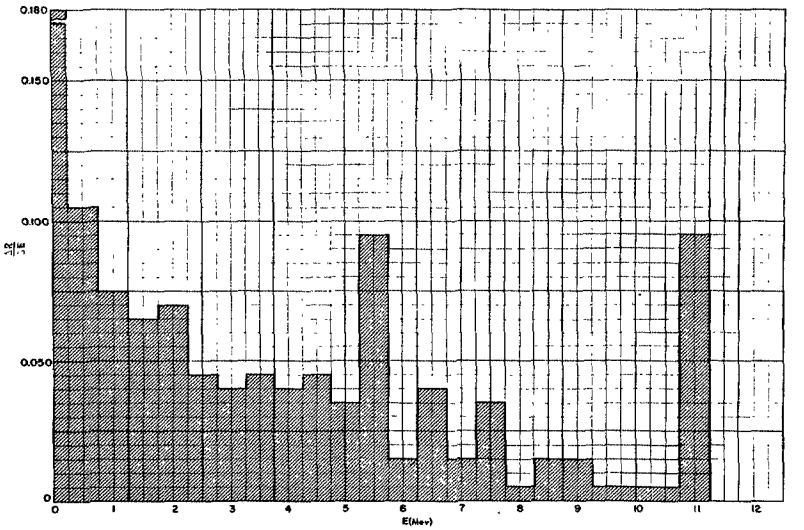

FIGURE 11.—*Initial gamma differential dose spectrum at 1,500 yards.*

2. The second point of interest is that the spectrum at 1,500 yards is harder than the spectrum at 1,000 yards: That is, its most energetic components are *relatively* more prominent. Previously it was shown that with *mono*energetic gamma point sources, spectra become "softer" or *less* energetic with increasing distance, due to the generation of secondary scattered photons of lower energy than the unattenuated source photons. Consequently, one must conclude that with the *poly*energetic bomb source spectrum, filtration of low energy components occurs more rapidly with increasing distance in this range than does degradation of the more energetic source photons. This would give rise to a net "hardening" of the spectrum with increasing distance.

At 3,000 yards the "hardening" effect of increasing distance is even more apparent, as is seen in Figure 12. In this spectrum the single most prominent dose contribution is made by the *very* most energetic constituent; namely, the 10.8 Mev gamma photons from the nitrogen radioactive capture sources.

The filtering effect of distance on the very energetic gamma spectra resulting from initial bomb gamma radiation is emphasized in Figure 13. Here there are presented simultaneously the integral dose spectra corresponding to the differential dose spectra just reviewed. It is seen that with increasing distance there is a decreasing fraction of total dose contributed by photons less than any one given energy. For example: at 1,000 yards about 73 percent of the total dose is delivered by photons of 5 Mev or less, but at 3,000 yards such photons contribute less than one-half of the total dose.

In summary, this theoretical calculation of extremely hard initial gamma radiations is of

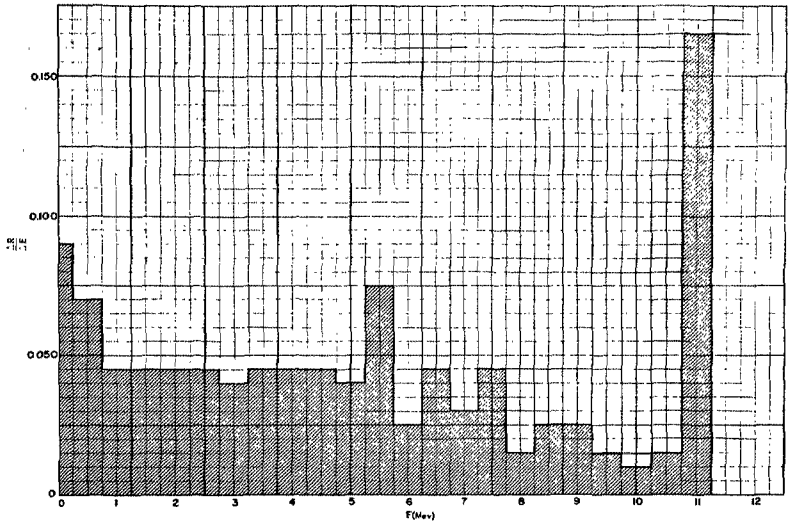

FIGURE 12.—*Initial gamma differential dose spectrum at 5,000 yards.*

great interest, but ". . . the proof of the pudding is in the eating." It would be desirable to have field measurements to support these predictions. Unfortunately, very little experimental work has been conducted to determine the spectrum of initial gamma radiation, except for some general and non-definitive conclusions to be drawn from absorption and depth-dose measurements. Although some field data based on photon-activated reactions of high energy threshold do attest to the presence of at least *some* very energetic gamma rays from nuclear detonations, it is difficult to check definitively the conclusions derived from the transport theory approach. However, one can compare theoretical predictions of total air dose with the well documented film badge gamma dose-versus-distance data from weapons field tests in order to determine in a general way if the calculated spectra yield dose information that is consistent with the field data. Then, by inference, the spectral information leading to the total dose calculations would also be validated.

COMPARISON OF INITIAL GAMMA RADIATION CALCULATIONS WITH FIELD TEST DATA

Figure 14 presents the air dose-versus-distance curve for the representative bomb configuration discussed before. The components of total dose due to fission product gammas and to nitrogen capture gammas are indicated separately. The composite dose is then determined by adding these. For actual test nuclear devices, with known bomb parameters, *specific* dose-versus-distance predictions can be made by the methods that have been discussed. These can then be corrected by conventional techniques to the appropriate atmospheric densities and compared with measured field data.

Figure 15 represents a typical small-yield bomb. The theoretical data represented by the "X" 's conform amazingly well to the measured doses, symbolized by the circles.

In the case represented by Figure 16 a very low yield device was fired, and the bomb parameters were such that the relative contribution of nitrogen capture gammas was small. Still, the calculated points are acceptably close to the measured data.

For contrast, Figure 17 pertains to a case wherein nuclear parameters predicted an unusually significant nitrogen capture contribution and a total gamma dose per KT of yield

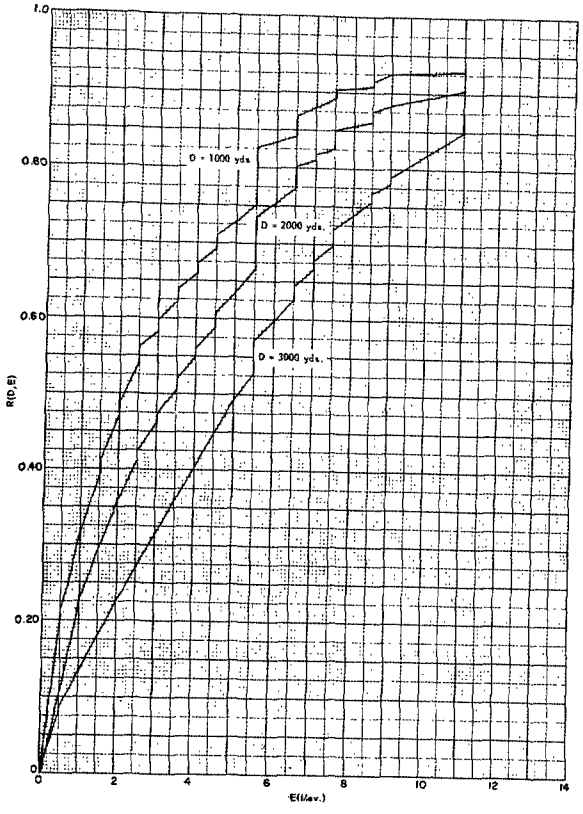

FIGURE 13.—*Initial gamma integral dose spectra.*

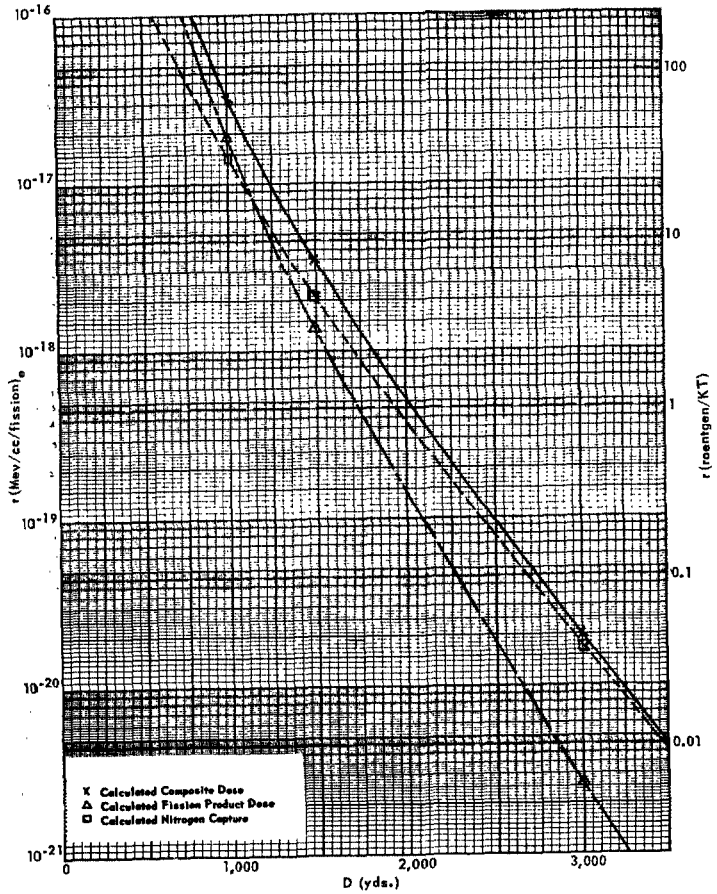

FIGURE 14.—*Attenuation of initial gamma dose with distances.*

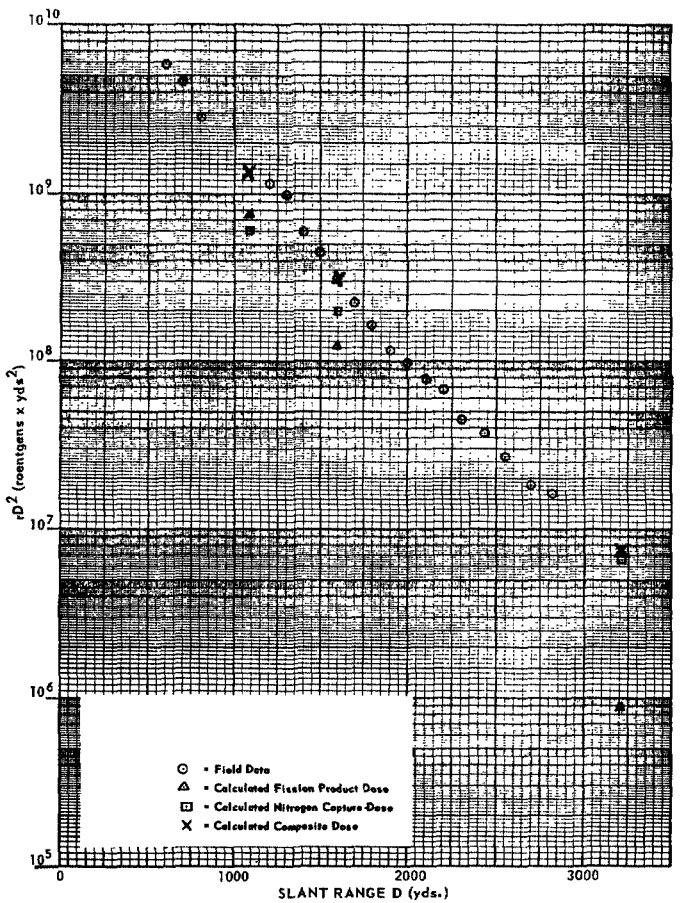

FIGURE 15.—rD^2 vs D *plot, comparison with field data.*

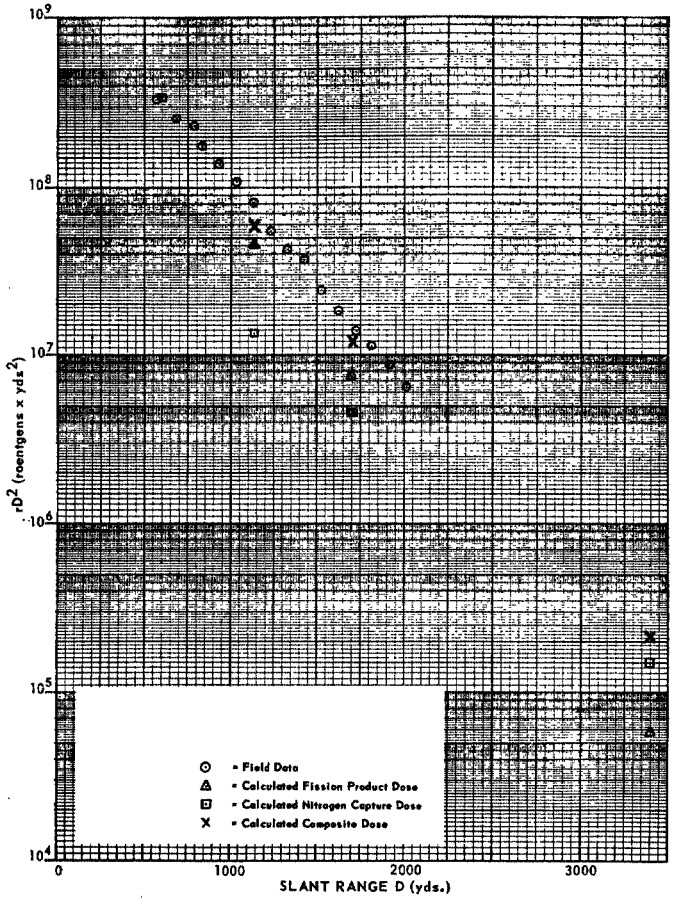

FIGURE 16.—rD^2 vs *D plot, comparison with field data.*

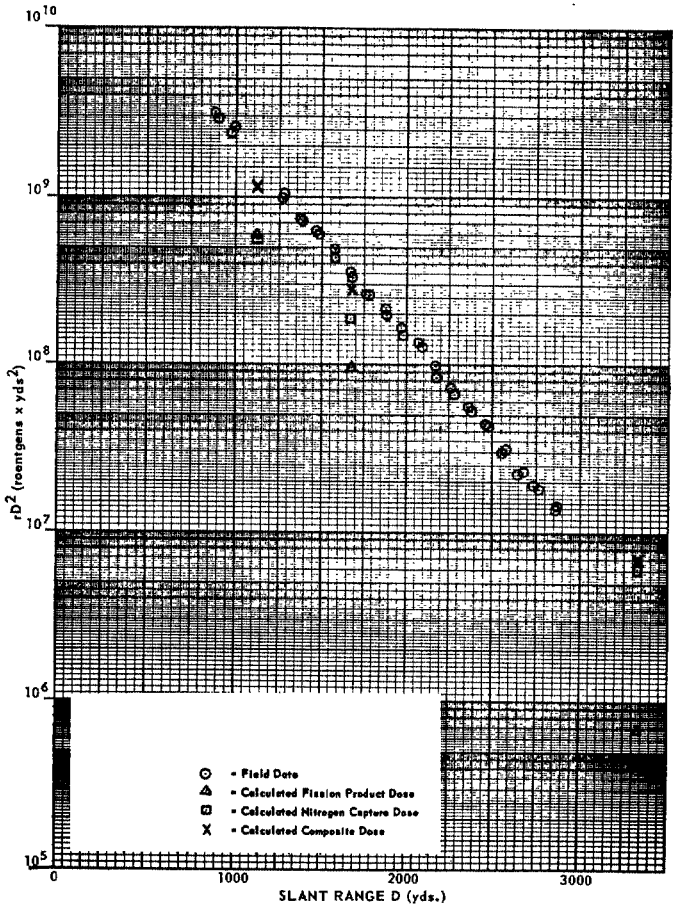

FIGURE 17.—rD^2 vs *D plot, comparison with field data.*

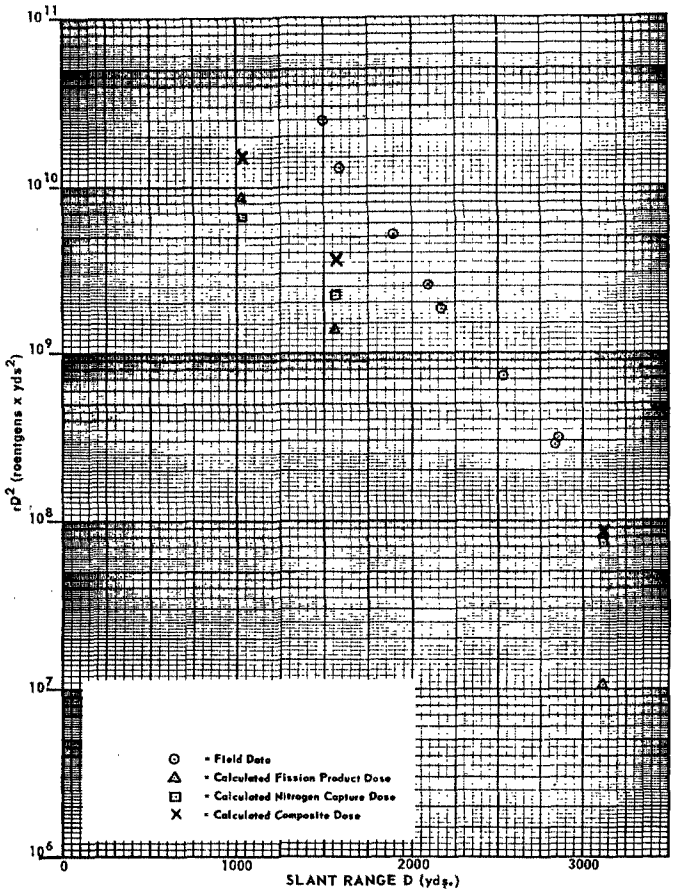

FIGURE 18.—rD^2 vs D *plot, comparison with field data.*

more than one and one half times that of the previous device. Again, however, the theoretical prediction appears sound.

Figure 18 presents an instance wherein unmodified theory and measurement do *not* agree—especially at closer distances. However, this shot represents a weapon of relatively large yield, and it is presented as a reminder that with high yields—such as more than about 100 KT—a phenomenon comes significantly into play that is relatively unimportant at lower yields but which cannot presently be dealt with analytically for inclusion in the unmodified transport theory. The phenomenon referred to is the radiation enhancement that is due to the modification of the previously homogeneous atmosphere by the weapon's blast wave. This enhancement amplifies total dose above that predicted by theory for the unmodified atmosphere; and since it affects fission product gammas far more significantly than it affects nitrogen capture gammas, it also alters the spectral shape from that predicted by the methods previously discussed.

In other words, for very large yield weapons initial gamma doses are much greater than predicted by the unmodified transport theory; and furthermore, the dose spectrum is much "softer" or less energetic due to the relatively decreased contribution of the very "hard" nitrogen capture gammas at this yield range. Interestingly enough, however, an empirically estimated enhancement factor to allow for this hydrodynamic enhancement effect corrects the calculated points shown in Figure 18 so that then they *do* fit the field data.

The analytical method is further supported by calculations made for the Hiroshima and Nagasaki bombs. As can be seen in Table I, the calculated points lie within a few percent

TABLE I.—COMPARISON OF INITIAL GAMMA RADIATIONS CALCULATIONS FOR THE ATOMIC BOMB IN JAPAN

Distance from explosion (yd.)	"Calculated Dose" Reported in Table 3.8 of Oughterson & Warren [7] for both cities (r)	Doses for both cities read from Fig. 7.42 of "Effects of Atomic Weapons" [6] (r)	Gamma doses predicted by the methods of this paper (r)	
			Hiroshima	Nagasaki
0	(1)			
1,267	700	750	630	630
1,733	100	105	105	98
2,267	15	15	18	17
2,800	3	4	3.5	3.2
3,333	0	1	0.80	0.73

[1] "Maximum."

of the doses predicted by the effects handbook "Effects of Atomic Weapons" [6] on the basis of compiled empirical measurements; and they agree to within less than 10 to 20 percent error with the essentially identical values quoted by Oughterson and Warren in their book, "Medical Effects of the Atomic Bomb in Japan." [7]

FALLOUT BOMB GAMMA RADIATIONS

Since the field data on initial gamma radiation seem generally to confirm the validity of the transport theory approach, it is particularly appropriate at this fallout conference to pursue the application of the transport theory method to fallout gamma dose and spectrum. The geometry of fallout as represented by an effectively infinite plane source of radiation is amenable to theoretical treatment. Data can be presented in a fashion analogous to that previously utilized for the effective point source geometry.

For example, Figure 19 shows a differential dose spectrum calculated for a height of 3

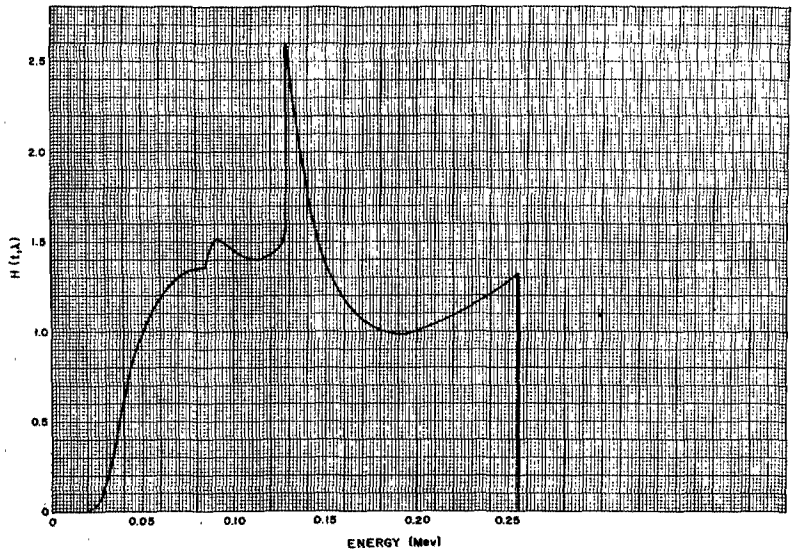

FIGURE 19.—*Plane isotropic source, differential energy spectrum, 3 feet above the plane, E_0=0.255 Mev.*

feet above a plane contaminated with a source emitting monoenergetic photons of 0.255 Mev. Despite the proximity to the "ground," much of the radiation reaching the detector position originates at considerable distances and is significantly degraded by a long path through air before reaching the detector. The abrupt peak and discontinuity seen on this chart represent the maximum energy loss achievable in a *single* Compton interaction: namely, the case where the secondary photon is emitted at 180° to the path of the primary photon.

There are in Figure 20 the integral dose and energy spectra corresponding to the differential dose spectrum of Figure 19. Similar spectra are calculable for other source energies, of course, and from these solutions interpolation curves can be drawn up.

In order to calculate crudely dose spectra from fallout once the source energies are known, an interpolation curve such as that of Figure 21 can be used. Its use and interpretation are the same as for the case of interpolation curves for point isotropic gamma sources, as discussed earlier.

With a sample source spectrum similar to the one applied before to the point source case, but now modified to fit the plane source or fallout case, the integral dose spectrum of Figure 22 was generated. For the energetic sample source used, there is relatively little degraded radiation received by the gamma detector.

The next step would logically appear to be analogous to the procedure applied to the initial gamma case: that is, normalization of fallout source spectra to actual weapon or

target parameters. Unfortunately, however, fallout gamma sources are not constant nor even easily predictable.

For different weapons types the very nature of the radioactive materials available for fallout may vary. For example, some devices may produce significantly large yields of induced activities in addition to fission products. So-called "clean" weapons will produce relatively few fission products and may produce *comparatively* significant induced activities from soil—and so on.

However, even for a *given* weapon type, soil and meterological conditions will vastly alter

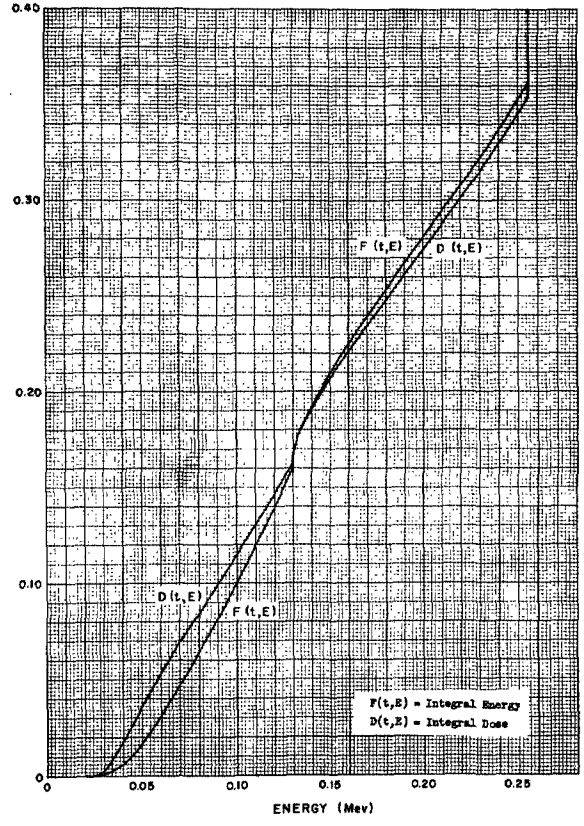

FIGURE 20.—*Plane isotropic source, integral energy and dose spectra, 3 feet above the plane, E_0=0.255 Mev.*

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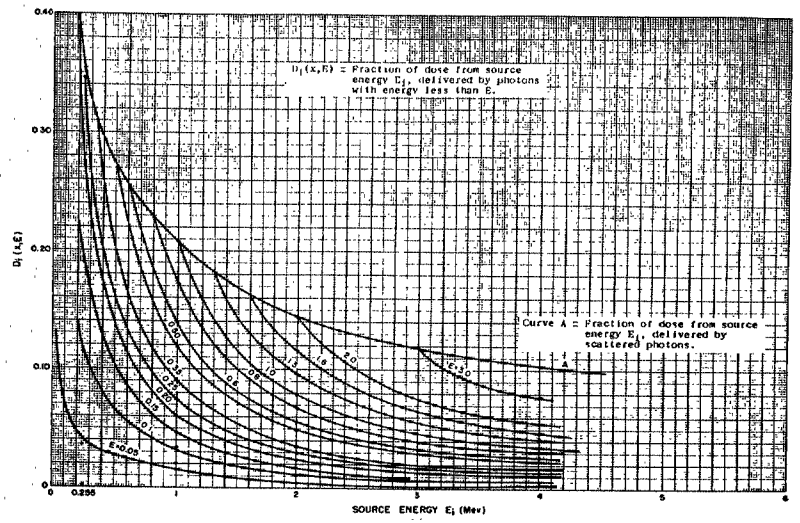

FIGURE 21.—*Plane isotropic source, interpolation curves for elevation of 3 feet above the plane.*

not only the intensity of fallout at a given location, but also the fractionation and partition of fission product source activities within the fallout.

Moreover, even for a given set of weapon, surface, and meterological conditions—in fact, even after all fallout actually has been deposited in a given instance, the nature of the fission product gamma source will change with time. This will result from both the natural decay scheme of fission products and from leaching and weathering processes.

Despite these uncertainties in the prediction of fallout gamma sources, some representative picture of fallout gamma spectra may be drawn by applying interpolation curves for plane source geometries to fallout sample sources analyzed in actual field experiences. This has been done by Sondhaus [8] for a fallout sample obtained following the "Bravo" event of Operation CASTLE, as is seen in Figure 23.

In this figure the dashed bars represent the original source spectrum analyzed from a fallout sample on the fourth day after the detonation. The histogram spectrum was then constructed following the methods discussed here. This dose spectrum is relatively "hard" or energetic compared with most laboratory sources, but it is not nearly so energetic as the dose spectra previously presented for the initial gamma radiations.

Although this spectrum is generally compatible with dose spectra actually surveyed in comparable fallout fields, even further support might be given to the analytical method if attempts were made to correlate computer-calculated predicted total gamma dose above analyzed fallout fields with actual measured data. This would be similar to the test employed in the initial gamma case, but to date apparently it has not been attempted.

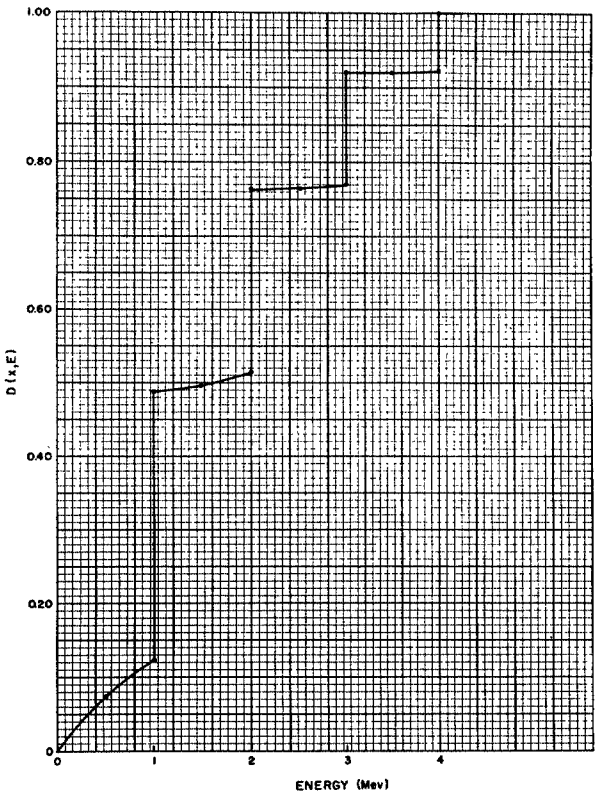

FIGURE 22.—*Sample plane isotropic source, integral dose spectrum, 3 feet above the plane.*

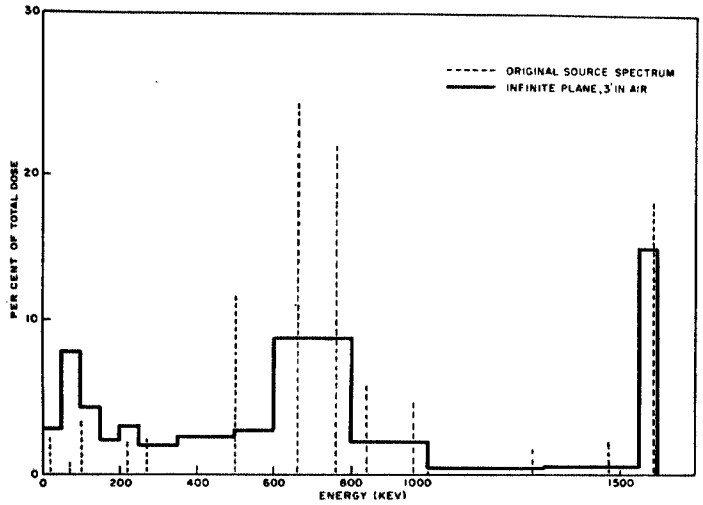

FIGURE 23.—*Distribution of inherent energies of gamma radiation from an actual mixed fission product sample and histogram of degraded energies calculated for 3 feet in air above a uniformly distributed fission products field.*

COMPARISON OF INITIAL AND FALLOUT GAMMA SPECTRA

In conclusion it may be said that the theoretical calculations of initial and fallout gamma spectra and dose appear reasonably well supported by known data. In this regard it then becomes of interest to review and compare representative spectra calculated for initial and fallout gamma radiations.

Figure 24 shows again the fallout spectrum of Figure 23, this time in conjunction with an initial gamma spectrum at 1,500 yards from a typical relatively low yield weapon, which was presented earlier in Figure 11. Noting the different expansions of the energy scales along the abscissas for the two cases presented here, it is apparent that although the fallout spectrum might be said to be a "hard" one, the initial gamma spectrum is still vastly *more* energetic.

CONCLUSIONS AND SUMMARY

1. A theoretical method for describing the propagation of gamma rays in various media has been developed, and numerical solutions have been carried out to the point where total dose and 4π-volume spectral calculations can be made. Further computer calculations *could* make available spectral information as a function of angular distribution, and this might be desired for shielding studies.

a. The method has been normalized to weapons parameters for the case of initial bomb gamma radiation, and resultant dose-versus-distance predictions generally fit in well with observed field data.

b. Although application to the fallout case is more empirical, the compatibility of the calculations with what data *are* available and the general validation of the underlying theory and application in the initial gamma

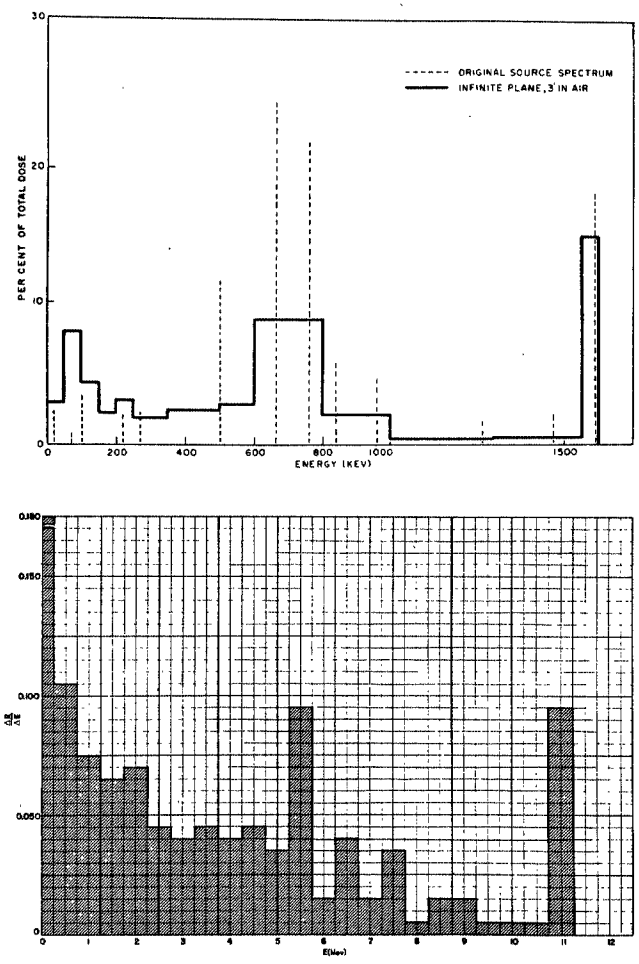

FIGURE 24.—*Comparison of differential gamma dose spectra for initial and fallout radiation (Figs. 11 and 23).*

case suggest that the method is valid for consideration of fallout gamma radiations also.

2. Application of transport theory to the initial gamma radiations shows that the majority of the air dose delivered at distances of a thousand-or-so yards and further is deposited by very energetic photons, ranging up to the 10.8 Mev gamma rays emitted by the nitrogen capture component of the bomb gamma source.

a. It further appears that for these composite energetic radiations the air acts more as a filter than as a scattering medium, so that the initial bomb gamma spectra "harden" with increasing distance.

b. In the case of very large yield detonations blast wave radiation enhancement factors may vitiate the theoretical predictions and produce larger total doses with softer energy spectra.

c. Nonetheless, the exceedingly hard spectra present in most cases of initial bomb gamma radiation from which biological radiation damage criteria have been derived must be taken into account before applying these criteria directly to fallout or other situations.

3. Calculation of fallout gamma spectra has been less extensive. Generally fallout dose spectra must be far less energetic than are initial gamma spectra.

a. Theoretical calculations of *both* dose and 4π-spectrum from fallout, based on either measured or predicted gamma source data as a function of time, and of weapon and of environmental parameters should prove feasible but apparently have not been attempted.

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GEOMETRICAL AND ENERGY FACTORS INFLUENCING THE EFFECT OF PENETRATING RADIATIONS ON MAN [1]

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INTRODUCTION

In considering the degree of effect to be expected in man exposed to penetrating radiations from the atomic bomb, it is necessary to examine the extent to which the geometry of the various possible exposure situations and the energy or spectrum of the beam may influence the result. These factors are known from laboratory experience to be of considerable importance, and must be taken into account when efforts are made to compare quantitatively the results under different conditions of exposure.

In this paper, the patterns of dose deposition through a man-sized phantom to be expected theoretically are developed for a variety of exposure conditions, and these are compared with the experimentally determined depth dose patterns. The degree to which biological effect is influenced by the various patterns of dose deposition are then considered. It is shown that such considerations can result in a difference of a factor of 5 or more in the degree of effect to be expected under various conditions of exposure, for the same monitored air dose.

The laboratory situation will be considered first for two reasons. The simpler situations in the laboratory allow a basis for developing the situations to be expected under the more complex field conditions. In addition, the hazard to man in the field of necessity must be evaluated in terms of laboratory experience with large animals and man. In general, laboratory biological data are far more reliable than those obtained under trying field conditions. In the field situation, the immediate and fallout gamma radiation from the atomic bomb will be dealt with mainly. Fast neutrons will be considered briefly. Some of the present material is presented in more detail elsewhere [1].

A rather obvious fact must be introduced initially. Monitoring instruments measure the free-in-air dose. However, there is no real interest in the dose received by the ambient air—the degree of biological effect is determined by the radiation dose received by the tissue. It is this dose, and its distribution in the body that governs the degree of biological response. This basic fact has, of course, been long recognized by radiologists, and the recommendations for many years in the reports of National and International Committees on Radiation Units in that dose be reported in terms of tissue dose [2] rather than the free-in-air dose [2, 3]. Thus some of what I say has long been known by radiologists; however, much of it has not been brought to the attention of radiobiologists and others concerned with hazard evaluation in man.

The use of tissue dose has gone far in resolving apparent quantitative differences in biological response in radiology, and in radiobiology concerned with small animals. Both, in general, are concerned with radiation effects in a relatively small, circumscribed volume of

[1] Research supported by U. S. Atomic Energy Commission

[2] See refs. 2, 4, and 5. Tissue dose refers only to the ionization measured by the detector embedded in the material being irradiated and usually does not indicate accurately the absorbed dose, i. e., the energy per unit mass deposited in the irradiated material, here tissue or unit density material. Over much of the range of radiation energies usually of interest in large animal work, from 250 KVP to 1.5 Mev or higher, the tissue dose will be equal to the absorbed dose in soft tissue, expressed as rads (100 ergs/gram), to within 10 percent or better. Much larger discrepancies occur in bone.

tissue. With "total body" irradiation of large animals and man, however, the problem cannot be taken care of this simply and new complications enter. Frequently, in practical situations involving hazard evaluation (in the field, and around reactors or other nuclear machines), *only the monitored air dose will be known at the time.* In addition, with large animals and man, the dose throughout the body frequently is markedly inhomogeneous. With some types of "total body" exposure, portions of the tissues receive but a very small percent of that received by other tissues. Thus two separate problems emerge, (*a*) for a given monitored air dose, what is the tissue dose and its distribution pattern through large animals and man for different conditions of exposure in the laboratory and in the field, and (*b*) with large animals, to what degree does the extent of biological effect vary with different patterns of dose distribution in tissue?

Terms used in this report conform to the recommendations of national and international committees [2, 3]. Dose and exposure are used interchangeably. "Free air dose" or "air dose" indicates the dose measured free in air in the absence of animal, phantom or exposure equipment. Unless otherwise specified, this refers to the dose as it has been conventionally measured at a point in space corresponding to the proximal skin surface (the side nearest the radiation source) of the animal or phantom when it is later put in place for irradiation. This is termed more explicitly the "entrance air dose" and is expressed in roentgens. Air doses at other points in space are easily approximated under most circumstance by use of the inverse square law. Dose measured with the dosimeter embedded at any position within the animal or phantom in place for irradiation is termed "tissue dose," also expressed in roentgens. Thus, "entrance tissue dose," "midline tissue dose," "exit tissue dose." Tissue doses are not converted to absorbed dose [2], expressed in "rads," because of the uncertainty of the conversion factor from tissue dose under some conditions discussed, and because of the considerable variation of the conversion factor with different tissues [4, 5].

A word should be said initially regarding the possible application of the large amount of dosimetry data that has been published in connection with clinical radiation therapy to the problem. Most clinical radiotherapy exposures differ fundamentally from the "total body" exposures considered here in that the object of the one is to obtain localized, circumscribed partial body irradiation of a diseased area, while the object of the other usually is to obtain an equal degree of exposures to *all* tissues of the body. The one usually attempts to narrow the beam by collimation or by the use of ports; the other requires a beam sufficiently broad to expose the entire irradiated object. Thus, the numerous depth-dose figures published for radiotherapists usually cannot be carried directly to the "total body" exposure situation, although the curves obtained with very large area ports apply approximately in some situations. Since the depth-dose pattern with "total body" irradiation is highly dependent on the precise conditions of exposure, it is not practical to compile complete tables of depth-dose values for references. The patterns to be presented here obviously apply strictly only to the specific conditions employed.

EXPERIMENTAL

The exposure geometries considered, all described more fully below, include unilateral, bilateral, multiport, rotational, ring, and "4 Pi" exposures in the laboratory, and exposure to immediate and fallout gamma radiations in the field. In what follows, each situation is investigated in terms of geometrical considerations and the principles of interaction of electromagnetic radiations with matter. The expected curves are compared with experimentally obtained depth-dose patterns. In the experimental work, a cylindrical Masonite (density of 1.1) Masonite phantom 26 cm. long and 26 cm. in diameter, corresponding to a 32-inch waist, was exposed under each of the

laboratory conditions listed, and depth-dose measurements were made. This phantom obviously does not represent exactly the essentially oval configuration of man in cross section in the region of the trunk, but was felt to be a sufficiently close approximation. A diagram of the exposure conditions for a "point" source is shown in Figure 1 for reference purposes. A target to "skin" distance (TSD) of 100 cm. was used for all exposures unless otherwise indicated.

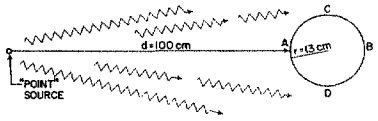

FIGURE 1.—*Schematic diagram showing method of exposure of a Masonite phantom to a "point" source of X- or gamma radiation.*

Studies showed that lengthening the cylindrical phantom beyond the 26 cm. did not alter the depth-dose curves detectably. The laboratory radiation used for most of the exposures was cobalt-60 gamma rays. As will be seen, high voltage (250 to 2,000 KVP) would have served as well for most exposures; however, the use of Co^{60} allowed more direct comparison of the geometry effect with some exposures not attainable with X-rays (ring, 4 Pi and field exposures). A description of the cobalt generator used for bilateral cross fire, ring and 4 Pi exposures is given in references 1 and 6.

For essentially all laboratory dosimetry, the same 100 r capacity Victoreen thimble chamber and charger-reader were employed. For a few low dose rate exposures with the bilateral and ring exposures, a 10 r capacity Victoreen thimble chamber, intercalibrated with the 100 r chamber, was used. The chambers were embedded in a thin, close-fitting plastic shell which was, in turn, inserted into closely machined holes drilled in the Masonite phantom. Thus, the phantom was essentially solid during exposure. The same observer took all laboratory measurements. The phantom measurements in the field were made with thin-walled Sievert-type ionization chambers embedded throughout the thickness of the phantom. For measurement of gamma radiation in the fallout field, the chambers were enclosed in sufficient copper to exclude beta radiation. The thimble chamber measurements did not allow accurate characterization of the depth-dose pattern at the surface and just beneath the surface of the phantom. Since only relative measurements were used in the phantom measurements, absolute calibration of the chambers used was not necessary. Curves were not corrected for inverse square fall off, since it was desired to present depth-dose patterns as actually observed.

RESULTS

Unilateral exposure.—The basic exposure technique, unilateral irradiation, is shown diagramatically in Figure 1. Radiation from the Co^{60} "point" source traverses air and impinges on the unit-density cylindrical phantom. In Figure 2 are shown curves describing the rate of fall off in dose through the phantom along a diameter parallel to the central axis of the beam. It is useful to attempt to derive the expected curve, since very few experimental depth dose curves for various energy gamma rays are available. The exponential curve "a" indicates the rate of fall off under narrow-beam or "good" geometry conditions, in which no scattered radiation reaches the detectors placed in the phantom. This indicates the rate of fall off of the primary beam uncorrected for inverse square effect. Curve "b" indicates approximately the rate of fall off in the phantom due to inverse square along with a TSD of 100 cm. The measured curve c, can be regarded as curve a, corrected for scattered radiation and for inverse square (only the primary beam closely follows inverse square from target). The amount of scattered radiation, or the "build up factor," has been calculated using the theories of Spencer and Fano [7, 8] for the infinite medium [9] and for the barrier problem [10], but not for the geometry considered here. The infinite medium build up factors underestimate the rate of fall off

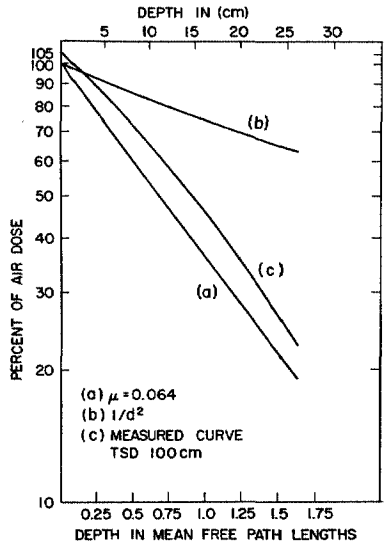

FIGURE 2.—*Depth-dose curve for cobalt-60 gamma radiation, unilateral exposure, TSD of 100 cm.*

(calculated curve higher than measured curve by factor of 1.3 at the midline and 1.6 at the exit). The build up factors for a water barrier describe the measured curve within 10 percent, however this appears to be fortuitous and little theoretical justification exists for applying barrier build up factor to the present geometrical situation. The true absorption coefficient (σ_a, approximately 0.03) predicts the midline dose within 5 percent but overestimates the exit dose by a factor of 1.3. Thus the depth-dose curves to be expected with gamma rays cannot be predicted precisely from presently available theoretical data; however a basis does exist for approximating the curve to be expected from a monochromatic beam (a beam composed of several monoenergic components can be handled by treating each component separately and adding the results). The build up factor varies markedly with energy and depth of penetration. Build up is rapid over the first mean free path, which results in a low energy beam appearing to be more penetrating than it is over the first few cm. of unit density material. X-ray beams, with their broad and continuous spectra, cannot be handled in this fashion. The considerations developed above are of particular importance later in considering bomb radiations.

In Figure 3, the measured curves for Co^{60} gamma and other radiations are shown for comparison. In all cases the total dose is delivered in a single exposure from one side of the phantom.[3] It is apparent from the figure that marked nonuniformity of dose deposition results even with highly energetic radiations,

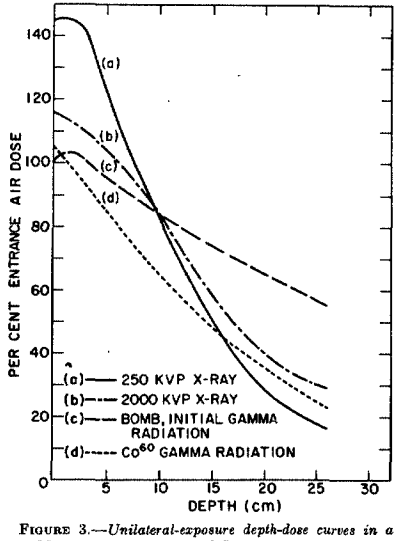

FIGURE 3.—*Unilateral-exposure depth-dose curves in a Masonite phantom for different energy radiations; depth-dose expressed as percent of entrance air dose.*

[3] The term "unilateral" is applied for convenience to the exposure to the initial gamma radiation from the atomic bomb, even though an appreciable component of the total dose undoubtedly is received from the lateral and distal aspects of the phantom.

and that with this type of "total body" exposure, the distal surface may receive only a very small percentage of the "dose" that the phantom or animal, by convention, is said to have received. The marked fall off in dose results from both absorption in the phantom and from the inverse square effect.

Bilateral exposure.—In an effort to overcome the marked lack of uniformity of depth dose obtained with unilateral exposure, a number of investigators have employed the "bilateral exposure" technique (see the excellent work of Tullis, ref. 11). This procedure is identical to the unilateral exposure, except that one-half of the "total dose" is administered from one side. Thus, if a total of "300 r" is to be given, 150 r as measured free in air at the proximal skin surface is given from side A (fig. 1). The remaining 150 r is then administered from side B. The depth-dose pattern for each separate exposure to cobalt-60 gamma rays and the total obtained by combining the values obtained with each separate exposure are shown in Figure 4–A.

It can be seen from the curve that the tissue dose throughout the phantom is remarkably uniform when contrasted with that obtained with unilateral exposure, and that a maximum variation of only 10 percent is obtained in traversing the phantom. *Of equal importance, however, is the fact that the tissue at no point in the phantom exceeds 62 percent of the entrance air dose, the dose that the phantom, by convention, is said to have received.* The reason for this discrepancy lies mainly in the fact that during each half-exposure, the distal side of the phantom is receiving only a very small percentage of the dose received by the proximal side, and on adding the half-exposures, the total falls far short of the dose said to have been given (see under "crossfire" exposure below for additional reasons).

If the midline air dose, instead of the entrance air dose, is taken as the total exposure, the resulting curve retains the shape noted above, but becomes 70 percent (instead of 55 percent) at the midline. Thus it is seen that use of the midline rather than the entrance air dose tends to equalize the tissue dose and the total air dose, but does not accomplish this fully.

Multilateral and Rotational exposure.—In these techniques, instead of giving one-half the dose from each of 2 sides, the dose is administered one-fourth from each of 4 "sides," one-eighth from each of 8 "sides," etc. The limiting situation involves rotating the source about the phantom at TSD of 100 cm., or equivalent, rotating the phantom placed 100 cm. in front of the stationary sources. It is easily shown [1] that these procedures do not differ materially in effect from bilateral exposure, and the depth dose patterns obtained (curve d, fig. 4–A) superimpose essentially on the bilateral curve.

Crossfire technique.—With the crossfire technique, only a *single* exposure using two opposing "point" sources energized simultaneously is used, as opposed to the bilateral technique in which two exposures, first one side and then the other, are made with a single source. The resulting dose pattern is shown as curve a, Figure 4–B. It is apparent that the shape of the curve is negligibly different from that obtained with bilateral, multilateral or rotational techniques, and that the tissue dose is still considerably below the air exposure dose that the phantom is said to have received.

The reason for the low tissue dose relative to air dose may not be immediately apparent, since with crossfire technique the air exposure dose throughout the exposure volume is essentially constant. It is easily seen, however, if one considers that as soon as the animal or phantom is introduced, the entrance tissue dose at either side (and throughout the phantom) immediately drops considerably because of absorption in the tissue or phantom. Thus, the entire curve is well below the entrance air dose.

The crossfire curve is higher than the bilateral curve because of what might be regarded as an artifact of dosimetry resulting from the manner in which *air dose* is measured with the two techniques. This can be seen as follows: with the bilateral technique, the total air "dose" given is the sum of two *entrance* air doses from the two half-exposures. With the crossfire

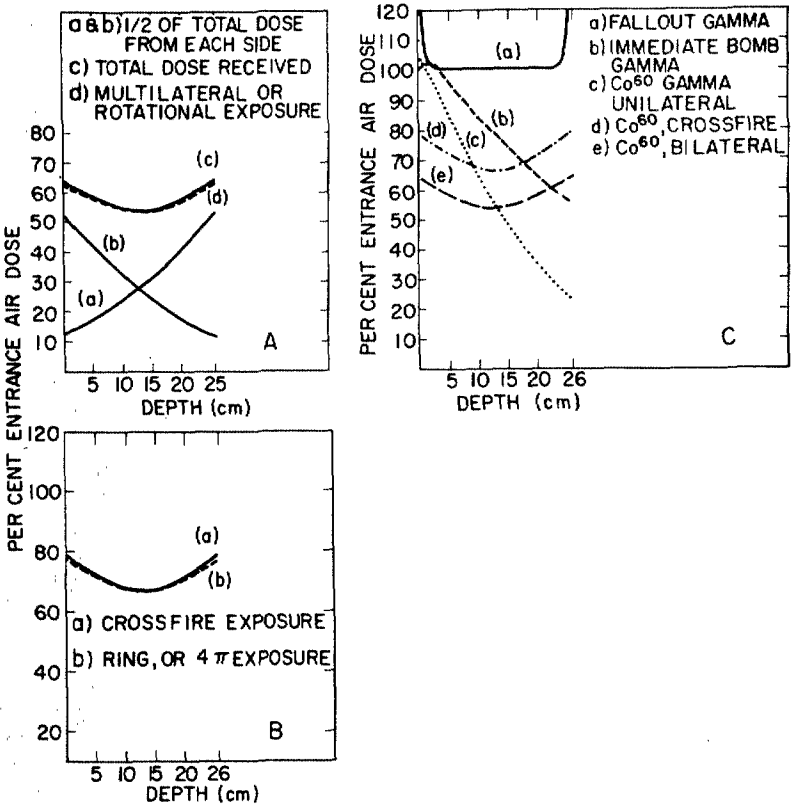

FIGURE 4.—*Depth-dose curves for cobalt-60 gamma radiation in Masonite phantom material for several exposure geometries, depth-dose expressed as percent of entrance air dose.*

technique, the total air "dose" given is the sum of the *entrance* air dose from one machine and the *exit* air dose from the opposite machine (less by inverse square). Thus the air "dose" with crossfire is *less* with bilateral and the tissue dose, in terms of percent of air "dose," is correspondingly *greater*. It should be noted that exposure with crossfire for one half the *total time* for both half-exposures with bilateral (two tubes on simultaneously with crossfire) yields a tissue dose curve that superimposes on the bilateral curve. However, since as noted, the air dose for the same total time is less with crossfire, the exposure time with crossfire for the same total air "dose" is *longer* than one-half the total time for bilateral, and the depth dose curve is thus above that for bilateral.

Thus, the difference noted is seen to result from the inverse square effect. However, it is important to note that while the crossfire technique has taken into account to a degree the inverse square effect, it has not, of course, in any sense eliminated the effect. It has averaged the entrance and exit exposure doses, and thus has raised the depth-dose curve, somewhat as might result if inverse square were negligible. An identical superimposed curve is obtained if, with bilateral, the average of the entrance and exit doses is used as the "air dose," instead of the entrance air dose with each half-exposure. If the midline air dose is used with bilateral exposure, the curve is essentially identical in shape to the crossfire curve, but is placed a short distance above it. Of importance later in considering the curve for fallout radiation, if the half-exposure curves for bilateral radiation are corrected for inverse square fall off before addition, the resulting curve, while placed at approximately the level of the crossfire curve, is considerably flatter than the crossfire curve (70.5 percent at the edges, 69.0 percent at the midline).

Ring and "4 Pi" exposures.—With ring geometry, the phantom is at the center of a concentric ring of fixed sources [1]. With "4 Pi" geometry, the phantom is placed in the geometric center of a group of sources arranged in essentially a spherical configuration [1]. The depth-dose pattern for both exposures is shown as curve b, Figure 4–B. They are essentially identical and are negligibly different from those obtained with the crossfire technique. These types of exposure can be considered to bear a similar relationship to crossfire exposure, as does multilateral or rotational exposure to the bilateral technique. Inverse square is taken into account to a degree, but is not corrected or eliminated.

Bomb, fallout gamma radiation.—The geometrical and other considerations noted above are of importance in considering the curve to be expected with fallout gamma radiation. The fallout field in the simplest case can be considered as a semi-infinite plane uniformly contaminated with gamma emitters. The spectrum of course varies with time and place; however, that given by Sondhaus [12] can be taken as sufficiently representative for the present purposes. It is seen to consist of a group of monoenergic sources, that can be considered to be composed of energies grouped at approximately 100 to 200 kev (11 percent), 0.75 kev (67 percent) and 1.5 Mev (22 percent). Scatter of radiation from partially-buried isotopes in the overlying ground, and secondary scatter from the ground will be neglected since considering only the undegraded beam will result in the largest possible dose to the phantom. The radiation at any given point in air above the plane will of course be coming from all directions; however the primary source can be considered as an infinite number of concentric ring sources and can be treated as such. As noted above, the crossfire or ring depth-dose curve[4] can be constructed from the unilateral curve, adding together two half-exposures from each side. No corrections for inverse square should be made in the unilateral curve since, as shown above, the resulting pattern on adding the half-curves is thus placed in correct relation to the air dose. Also, two separate calculations by Drs. Robertson and Brennan have indicated that the bulk of the radiation comes from several meters or more which tends to flatten the curve but not alter its relation to the air dose. The unilateral curves for the components of the fallout gamma spectrum were approximated in several ways as follows: since the bulk of the fallout radiation is approximately 0.75 kev (67 percent) and 1.5 Mev (22 percent), a curve closely approximating the unilateral curve for Co^{60} gamma would be expected. Uncorrected for inverse square, the curve

[4] The phantom in the fallout field is above the plane of the ring sources, as opposed to in the same plane in the laboratory situation. It can be easily shown, however, that this does not appreciably affect the mean path length of radiation in the phantom in reaching a given point, and thus absorption in the phantom is not significantly altered.

would be approximately 70 percent at the midline and 35 percent at the exit. This particularly, since the curves for Co^{60} gamma (1.3 Mev) and a cesium-137 source (0.7 Mev) agree within 3 percent at distances corresponding to the midline of the phantom [13]. Infinite media build up factors corrected for the discrepancy noted between theoretical and measured curves for Co^{60} gamma yielded midline and exit doses of 71 and 40 r, respectively. The build up factors for a water barrier [10], applied empirically, yielded corresponding percentages of 68 and 40 percent. Use of σ_a only results in values of 70 and 50 percent. It is reasonable to assume, then, that the unilateral curve for the fallout spectrum is approximately 70 percent at the midline, and 40 percent at the exit. Construction of a curve from this for the fallout field yields an expected depth dose pattern in the field that is essentially flat, with values of approximately 73 percent at the surfaces and 70 percent at the midline.

A depth dose curve experimentally obtained in a fallout field is shown as curve a, Figure 4–C. Doses were measured with Sievert-type ionization chambers. The high surface doses include beta radiation measured by the thin-walled ionization chambers. The air dose was determined by covering the ionization chambers with sufficient copper (approximately 800 mg/cm^2) to exclude beta radiation. As expected, the gamma tissue dose throughout the phantom was essentially constant. The tissue gamma dose was approximately equal to the air dose, however, as opposed to the approximately 70 percent predicted from theory. The reason for this discrepancy probably lies in the manner in which the air dose was measured. The thickness of copper, equivalent to the wall thickness of some "gamma" monitoring instruments, undoubtedly excluded some gamma as well as beta radiation.

Bomb, initial gamma radiation.—The curve to be expected with the immediate bomb gamma radiation was approximated in two ways. The linear absorption coefficient for bomb immediate gamma radiation observed at distance of biological interest (quoted on page 97, ref. 14) can be converted to the mass absorption coefficient by correcting for the small difference in electron density and for inverse square (no detectable fall off through the 26 cm phantom). Application of the absorption coefficient thus derived yields a decrease in tissue dose at the exit side to approximately 50 percent of the entrance tissue dose. A very similar result is obtained if the mass absorption coefficient for several Mev gamma rays (about 0.03) is used with the appropriate build up factor. The factors for infinite media apply closely here, since the large air mass constitutes an adequate scatter medium.

A measured depth-dose curve in phantom material exposed to the immediate gamma radiation from the bomb is shown as curve c, Figure 3. The phantom employed was a cylinder measuring 25 cm. in diameter, and measurements were taken approximately 3 feet above the ground. The agreement with prediction is good. It is apparent that while the rate of fall off of dose in tissue is still appreciable in a thickness of tissue approximating man, the exit tissue dose of approximately 55 percent is well above the value of approximately 20 percent for cobalt-60 gamma radiation in the laboratory. It is pointed out that with both initial and fallout gamma ray exposures, the dose is essentially uniform as one goes from one end of the phantom to the other. This is in contrast to all of the laboratory geometries described, and is approached only with "4 Pi" exposure.

Bomb, fast neutron irradiation.—Since fast neutrons are attenuated rapidly in traversing hydrogenous material, the considerations set forth for gamma radiations apply to fast neutrons from the atomic bomb as well. No measured neutron depth dose curves for the field situation are available; however, it is possible to estimate how the curve might look. It can be assumed that the source spectrum for relatively small weapons is not unlike the fission spectrum measured in the laboratory. In traversing approximately 1,000 meters to air to arrive at distances of biological interest, it is doubtful that the spectrum would change appreciably. Elastic multiple scattering in air would result in some departure from a monodirectional beam; however, it is probable that the beam would be far from isotropic. Therefore, the curves calculated by Snyder [15] for a plane monodirectional source would apply approximately. It is seen that the rate of fall off is quite rapid in hydrogenous material such as water. For a fission spectrum with average energy of about 0.8 Mev, and the very large majority of neutrons below 3 Mev, the dose could be expected to fall to the order of 10 to 15 percent of the surface dose at the midline, and considerably less than this at the exit surface. It is emphasized that this is only a rough approximation, and more refined calculations or measured curves should be obtained.

From X-ray data, however, it can be said that such shallow curves are relatively quite ineffective in producing *acute* illness or death in large animals (consider the very large monitored doses of beta rays required to produce acute effects). The relative biological effectiveness for fast neutrons, determined with essentially uniform tissue dose distribution in mice, appears to be of the order of 2 [16], i. e., neutrons are twice as effective as X-rays for the same tissue dose in small animals in which essentially all tissues receive the same dose. Because of the shallow depth dose pattern in large animals, however, the neutrons may be *less* effective for acute endpoints than penetrating X- or gamma radiation by a factor several times greater than the RBE determined in mice. It also becomes apparent that it is not possible to add the effects of the relatively nonpenetrating bomb neutrons and the very penetrating bomb immediate gamma radiation in a one-to-one ratio.

Body shielding, "local" geometry.—Allied to the depth-dose problems are those of partial body shielding, and localized concentrations of fallout material. Some degree of partial shielding probably will be common in the fallout field. Shielding of a relatively small region of the body, particularly if bone marrow is contained in the shielded portion, will markedly reduce the effect of a given radiation dose. "Hot spots" probably will be common in a fallout field because of drifting, buildings and local terrain configurations. The depth dose pattern may thus be essentially unilateral rather than flat as observed in the semi-infinite plane. As will be seen, the biological effects are reduced with unilateral exposure. It is highly probable that movement of the individual will result in a highly complex and unpredictable depth-dose pattern.

DISCUSSION

Comparison of depth-dose patterns.—In the preceding results, the marked difference in tissue dose, obtained with different exposure geometries for the same air dose as conventionally expressed, have been stressed. The large discrepancies possible must be kept in mind when only the air dose is quoted or is available. It is seen that no laboratory radiations as they have been employed quantitatively simulate the initial or fallout gamma radiations from the atomic bomb. Perhaps more striking than the differences, however, is the marked *similarity* of the depth-dose patterns for most of the exposure situations, and their essential *identity* if the artifact of expressing dose in terms of that received by the air rather than the tissues could be abandoned. The geometries fall into two basic categories—unilateral exposure, and a second to include all of the other types considered. With the exception of unilateral exposure, all those considered yield reasonably flat or uniform depth-dose patterns [11, 17].

The relationship of the midline tissue dose to the entrance air dose, for any exposure geometry, will vary considerably with beam energy, target-to-skin distance and animal thickness. The shape of the depth-dose curves (essentially flat) for all geometries except unilateral exposure is remarkably insensitive to these factors for radiations and exposure conditions commonly used for large animals irradiation (200 to 2,000 KVP X-rays, cobalt-60 gamma rays). As the beam energy becomes low (practically at about 100 KVP, 30 kev effective), or with animals of very large diameter (as with burros), the midline tissue dose

becomes very small compared to the entrance air or entrance tissue doses, and the depth-dose curve is far from flat. This type of "energy dependence" and the resultant biological effect has been studied [18, 19], and is discussed below. It should be noted that while fallout gamma radiation has been termed "soft," only a very small percentage of the primary beam is below 100 to 200 kev under most practical circumstances [1]. This is equivalent in penetrating power in tissue to a highly filtered X-ray machine of 250 or higher *peak* voltage, or KVP. Thus the fallout gamma radiation must be considered quite penetrating in terms of biological effectiveness.

Correlation of depth-dose patterns with biological effect.—From the depth-dose considerations outlined above, wide variations in the dose required for a given biological effect, expressed as *air dose*, would be expected with different exposure conditions. A glance at Tables I and II, in which large animal mortality data from the literature are collected, shows this to be true. The LD_{50} values for dogs and swine are given in the tables in terms of entrance air dose, as well as in terms of the entrance, midline and exit tissue doses.

A better correlation between dose and effect would be expected if tissue dose is used unless (*a*) an energy dependence of biological effect is present, (*b*) marked differences in the *shape* of the depth-dose pattern exist, or (*c*) strain differences in the degree of biological effect exist.

"Energy dependence" of biological effect as commonly used has included usually two separate phenomena to varying degrees, i. e., (*a*) a "true" or intrinsic energy dependence in which dose deposition through the irradiated objects compared is well known and *uniform*, and quantitative differences in effect for the same dose reflect different properties of the radiations, related to linear ion transfer (LET), or specific ionization; and (*b*) an "apparent"

TABLE I.—LD_{50} DOSES FOR DOGS EXPOSED UNDER DIFFERENT GEOMETRY CONDITIONS

Method of exposure	Radiation used	Radiation factors					LD_{50} dose				Reference
		Filter (mm.)	HVL (mm.)	TSD (cm.)	Dose rate (r/min.)	ma	Entrance air	Entrance tissue	Midline tissue	Exit tissue	
Unilateral (from above).	250 KVP X-ray (Picker).	14.2 Al Parabolic 0.5 Cu.	2.15 Cu	102	9	15	450	562	332	160	Michaelson,[a] Rochester.
Unilateral (from above).	1,000 KVP X-ray (G. E., transmitted beam).	12.7 Pb	5.6 Pb	274	10	3	450	495	360	202	Michaelson,[a] Rochester.
Unilateral	Bomb gamma	None		1,000 yds	High variable.		271	271	255	210	(14).
Bilateral	200 KVP X-ray (G. E.)	0.5 Cu	0.98 Cu	100	6	15	[b] 360		[b] 260		Prosser et al. Argonne (20).
Bilateral	250 KVP X-ray (G. E., radial beam).	0.5 Cu 1.0 Al.		100	15	15	281	252	252	252	Bond, USNRDL (17).
Bilateral	1,000 KVP X-ray (G. E., radial beam).	(Inherent)	2.0 pb	110	27	3	304	290	255	250	Bond, USNRDL (17).
Bilateral	2,000 KVP X-ray (G. E., radial beam).	6.3 Fe (Inherent).	4.3 Pb	200	15	1.5	312	265	265	265	Cronkite, NMRI (21).
Bilateral	2,000 KVP	6.3 Fe.	4.3 Pb	200	15	1.5	316	262	262	262	Gleiser, NMRI (22).
Bilateral	1,000 KVP	Not given in report					335		[b] 258		Boche + Bishop Rochester (23).
Bilateral	Co^{60} gamma	None	10.5 Pb	115	7				334		Shiveley [a] et al.

[a] S. Michaelson, J. N. Shiveley and J. Howland, personal communication.
[b] Calculated or estimated; value not given in reference cited.

TABLE II.—LD_{50} DOSES FOR SWINE EXPOSED UNDER DIFFERENT GEOMETRY CONDITIONS

Method of exposure	Radiation used	Radiation factors					LD_{50} dose				Reference
		Filter (mm.)	HVL (mm.)	TSD (cm.)	Dose rate (r/min.)	ma	Entrance air	Entrance tissue	Midline tissue	Exit tissue	
Unilateral	2,000 KVP X-ray (G. E., radial beam).	6.3 Fe (Inherent).	4.3 Pb	200	15	1.5	500	580	311	130	Tullis, NMRI (11).
Unilateral	Bomb gamma	None		1,000 yds	High variable.		225	225	191	145	Tullis, NMRI (24).
Bilateral	1,000 KVP X-ray (G. E., radial beams).	(Inherent only).		100	30	3	510	357	252	357	Tullis, NMRI (11).
Bilateral	1,000 KVP X-ray (G. E., radial beam)	(Inherent only).	2.0 Pb	110	27	3	425	302	255	302	Bond, NRDL (25).
Bilateral	2,000 KVP X-ray (G. E., radial beam).	6.3 Fe (Inherent).	4.3 Pb	200	15	1.5	398	279	242	279	Tullis, NMRI (11).
Multi-Source Field.	Co^{60} gamma	None	46.2 Al	Variable	About 0.85		618		300		Rust, et al., Oak Ridge (26).

energy dependence secondary to differences in penetration. These effects are considered below.

Low energy radiation can be considered first, and beta radiation provides the absurd case because it penetrates only a few mm. in tissue. Thus "total body" beta radiation in reality results in a type of partial body radiation of one organ, the skin. Energy is not deposited at depths sufficient to produce the "total body" irradiation syndrome of penetrating gamma radiation. Very low energy X or gamma radiation, e. g., 50 KVP X-rays, result in virtually the same picture as beta radiation when applied to the entire body surface, and the acute LD_{50} here is of the order of several thousand r or rep to the skin, as opposed to a few hundred r for penetrating rays. This would be expected with any type of partial body radiation.

As the beam energy increases, the effects of penetrating whole body radiation do appear, and the energy level where this occurs varies with body size and the geometry of exposure. In mice, with essentially bilateral (uniform) irradiation [18], the transition occurs at somewhere between 80 and 135 KVP; at about 80 KVP the LD_{50}, expressed as tissue dose *or* air dose, begins to rise rapidly. In the rabbit, the change occurs at a higher KVP, probably near 150 KVP. With dogs, the LD_{50} for 100 KVP X-rays (midline tissue dose) is 1.4 times that for 250 KVP, thus the transition occurs somewhere between these energies. From Table II, it is seen that above 250 KVP, the LD_{50} for dogs (bilateral X-irradiation, midline tissue dose) is independent of energy. No such data are available on larger animals the size of man; however, it appears likely from depth-dose curves that the transition would occur at 250 KVP or somewhat higher.

The above "energy dependence" thus is seen to be in reality a pseudo energy dependence—if the radiation dose cannot be delivered to the vital tissues, "energy dependence" of effect cannot exist. This effect has nothing to do with relative biological effectiveness (RBE) in the strict use of the term, although RBE frequently is used loosely to include it. As stated above, many of the radiations of concern in hazard evaluation are sufficiently energetic such that this factor is not large. The chief exceptions are bomb neutrons and beta radiation. With these radiations, however, the effect exceeds by far in magnitude the effect resulting from intrinsic RBE.

A possible "true" energy dependence of biological effect on energy over the ranges of in-

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terest has been discussed [1], and can be summarized briefly. The available data are conflicting; however, it appears that such an energy dependence may exist in mice over the range of 250 to 2,000 KVP, and that 1,000 and 2,000 KVP X-rays may be *less* effective by a factor of 0.8 or 0.9 (in terms of tissue dose). There are several pieces of evidence that Co^{60} gamma may be even *less* effective—perhaps 0.7. Part of these differences may be dosimetric in origin; however, they appear to be real as doses are measured at present. With large animals, dogs and swine, there appears to be no such dependence of effect over the range of 250 to 2,000 KVP. Undegraded gamma radiation (Co^{60}) appears to be less effective in the dog (Table I), as with mice. It would appear that intrinsic energy dependence over the range of energies of interest is at most of the order of 10 or 15 percent, a factor much smaller than other sources of uncertainty.

In considering the effect of *distribution* of dose as it affects degree of response, the concern is mainly in comparing one type of unilateral exposure to another, and unilateral to bilateral exposures. It is obvious by now that with identical depth-dose patterns, the same degree of effect, within a few percent, will result from the same dose. In comparing one type of unilateral irradiation to another, it is of course known that the shallower the curve, the *less* the effect for a given entrance or midline tissue dose. This can be easily seen from the data of Potter [27] and Ellinger [28], and that of Tullis in swine (Table II). Little difference is noted for dogs irradiated unilaterally with 250 and 2,000 KVP X-rays (Table I); however, the beams were filtered such that the depth-dose patterns were not greatly different [29]. It is thus clear that differences do exist; however, the data are not sufficiently good to allow quantitative treatment.

As for a means of predicting effects with a given unilateral pattern, some data obtained with small animals indicate that the *exit* tissue dose may be a normalizing quantity [27, 28]. The data in large animals are insufficient to evaluate this point. Integral dose or gram roentgens has been proposed as a normalizing quantity. Grahn and Sacher [18] have shown that with different types of "total body" irradiation, integral dose is of no value in this regard and the concept does not apply in predicting mortality with partial-body irradiation [30]. Even if integral dose were the normalizing factor, the computations involved are so complex and lengthly that this parameter would have no practical usefulness in hazard evaluation.

Some additional points will be mentioned in regard to the large animal data in Tables I and II. Looking first at the *bilateral* data for dogs and swine, it is seen that the *air dose* LD_{50}'s vary considerably among investigators, but that the LD_{50}'s in terms of midline tissue dose are remarkably constant for *X-rays* with a variety of energies and experimental conditions. The discrepancy between air dose and midline dose is much larger for swine than for dogs, which would be expected from the larger swine. This indicates that such data, to be quantitative for man, *must* be obtained on man-sized animals. Data from dogs or monkeys do not apply directly. It is apparent that the usually quoted LD_{50} values for large animals, in terms of air dose, are much too high, and that there is no true energy dependence of effect over the range of 250 to 2,000 KVP. The LD_{50} for dogs and swine are approximately equal and considerably below the LD_{50} for mice or rats. No biological data are available for large animals exposed to fallout gamma radiations; however, the LD_{50} in terms of midline tissue dose would be expected to equal those in the tables to a few percent.

With regard to the Co^{60} gamma data in Tables I and II, the higher LD_{50} values may reflect in part the apparent intrinsic energy dependence that has been noted for mice. With the swine exposed to Co^{60} in the multisource field at Oak Ridge, however, additional factors enter. It can be easily shown that approximately 65 percent of the radiation received at any point in air at the "center" of any unit of 3 of the total of 19 sources comes from a distance of approximately 1.5 meters.

Thus inverse square fall off is appreciable, unlike the fallout field. Also with large animals placed in a standing position among the sources, a large percentage of the radiation traverses the *long axis* of the animal, rather than a transverse (shorter) diameter as with animals exposed to bilateral X-irradiation or with man upright in the fallout field. Thus the midline dose would be expected to be relatively quite low compared to the air dose. With the cooperation of Col. Trum, additional depth-dose curves were obtained in the Co^{60} field, which indicate that the midline dose in a swine phantom is less than half of the entrance dose. The LD_{50} value (Table II) is correspondingly low in terms of midline tissue dose.

From Tables I and II, it can be seen that in the laboratory, more radiation dose (entrance air or tissue dose) is required to produce a given effect with unilateral than with bilateral exposure. With "unilateral" exposure to the immediate bomb gamma radiation in the field, however, the LD_{50} values are lower than for unilateral irradiation in the laboratory, and approximately equal to bilateral irradiation in the laboratory. This could indicate uncertainties in the field data—the LD_{50} values were obtained in a single determination with 10 animals per point, and the swine used were smaller than those used in the laboratory. It could also mean that the relatively flat curve for bomb immediate gamma resembles in effect bilateral, more than unilateral irradiation.

The considerations outlined must be taken into account in hazard evaluation. The problem is analogous to the RBE problem, which gave rise to the dose unit "rem" to more closely estimate hazard than is possible with the roentgen or rad. The dose in rem is equal to the dose in r multiplied by an experimentally determined RBE factor. It would appear that another factor should be introduced, a geometry or g factor, which must be experimentally determined for each situation as is the RBE factor. It is seen from the present paper, that under many circumstances the g factor may greatly exceed in magnitude the RBE factor.

The problem of accurate hazard evaluation in large animals and man is seen to be particularly complex. It is not possible to use a single quantity such as "r" or "rem" alone to predict hazard under a variety of circumstances—additional factors to describe the situation considered must be introduced. No one would ask for the "hazard" from a given dose of any common toxic agent such as arsenic without describing the situation further—how the drug is to be given, the chemical form, part of the body receiving it, time over which it was administered, size of individual, etc. Yet it is frequently expected that a "dose" of radiation in "r" or "rem" will describe the hazard under all situations. And the difficulties cannot be circumvented by changing a name—introducing, as has been suggested, some arbitrary type of "hazard" unit that supposeldy will indicate what effect can be expected in man. No one unit can ever describe the hazard; other quantities are necessary. Substitution of a "hazard" unit represents a regression to the "skin erythema dose" days, that nullifies the very great advance made with the introduction of the roentgen unit. The roentgen (or rep or rad) is as good as any presently available single quantity to allow a very general estimate of hazard. If greater accuracy of prediction is desired, then the situation must be recognized and treated as a complex one. This is done in other disciplines, and personnel are trained to handle the problem. Quantities in addition to the instrument reading in r or rads (where dose is measured, type of exposure, type of radiation (RBE), type of biological response of interest, dose rate, body region exposed, etc.) *must* be taken into account. These factors could be incorporated into one, or a series of nomograms; however they cannot be incorporated into a single "hazard" unit or into a single instrument reading. Perhaps most dangerous in attempts to devise a hazard unit is that it will involve combinations of several factors in unknown proportions. Thus one trained and conversant will not be able to sort out the important quantities that would allow accurate evaluation of the hazard.

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DISCUSSION ON TOPIC II

Gamma Energy Spectra and Geometry Factor

Dr. Cronkite. Thank you, Dr. Bond. Before throwing this open for general discussion and comment, it was called to my attention earlier by one of the members here that Dr. LaRiviere in his presentation of Dr. Mather's paper stated that 105 gamma radiation from neptunium was not important. I don't think you meant that, because work done in your own laboratory showed it was quite important.

Dr. LaRiviere. I am afraid he did say that in his paper.

Dr. Cronkite. Possibly you would take back to him that there is a little difference of opinion, predominantly from work done in the Division of Biology and Medicine at NRDL.

The following comments were later supplied by Dr. R. L. Mather:

> Unfortunately I could not be present at the meeting, and during the discussion exception was taken to my statement that the 105 kev quanta from Np^{239} have relatively low penetration and biological effectiveness. The statement is true to the extent that the usual gamma radiation from radioactive sources is of higher energy than 105 kev and will penetrate farther into a given material, particularly those materials with a high atomic number which are usually employed for shielding purposes. The biological effectiveness per quantum of radiation is proportional to the average amount of ionization which it produces in a small volume of air (roentgens) which when computed turns out to be closely proportional to the energy of the quantum for energies above 100 kev. In relation to the human body, however, a 105 kev quanta has a 10 percent chance of passing through the body, front to back, without experiencing any interaction (rather good penetration).
>
> Because of the very large proportion of 105 kev quanta in the typical fallout radiation 4 days post detonation this radiation may account for 20 to 50 percent of the gamma ray intensity (either energy flux or roentgen or biological effectiveness) as stated. Neither the hazard of this 105 kev radiation nor the fact that it can be controlled by relatively thin layers of dense materials should be ignored.

Dr. Cronkite. Dr. Borg, in your presentation you were obviously discussing things that were exclusively in a free air situation, without buildings and so on around. I believe the intent of this symposium was to eventually get down to some practical situations of what might happen to man. I would like not to get into a dissertation on this, but for you to make some comment on the general situation that existed in Japan where there were large concrete buildings next to people. How does this influence the dose that might be expected from prompt radiation?

Dr. Borg. The answer is that I don't know exactly, but the problem has been brought up before and looked into in this regard. The calculations which I discussed were made assuming the detector to be well up in the air, without even a ground interface nearby to interfere. Most of the measurements with which they were checked, however, were made close to the ground surface. There have been attempts made to reason through the effect the ground might have on a measurement made

nearby it in air: whether the ground acts more like a sink or a reflector for radiations.

To follow your suggestion, I won't make a dissertation of it. The answer is that the surface appears to act as either one under different circumstances, and apparently when the incidence is close to grazing, as it is a large distance from a not-too-high burst of a nuclear weapon, the model holds pretty well. Buildings, on the other hand, if they were close by, would probably decrease the dose over what had been calculated for free air. As we saw on the interpolation curves, even from the hardest components of the radiation a great deal of the dose that is delivered at a large distance from the bomb comes from scattered radiations, and they in turn to some extent, especially the lower energy ones, do not come from straight ahead but from the side; and occasionally the lowest energy photons even are back scattered toward the bomb again. So some large dense volume, such as a concrete building, that occupied a large part of the volume of this scattering source would probably decrease the dose to some extent over that predicted for free air.

Dr. CRONKITE. Are there any questions from the floor?

Dr. TERESI (NRDL). I would like to make a comment concerning this energy dependence. The comment I want to make is concerning the biological effectiveness of energies below the 250 KVP that was presented here. I am under the impression that for very low energies you do have a difference in biological effect, which is much less than these higher energies. If this is the case, what I would like to know is what would be the effects of shelters; for instance, individuals will be in shelters. Air doses will be measured inside. Therefore, the LD-50 may be very much different because of degradation of energy in going through shelters. I was just wondering whether or not you want to make some more comments on that.

Dr. BOND. Before I could answer that, I would have to ask you to give me the energy spectrum of the material after it went through the shelter.

Dr. TERESI. I don't know. I think this is something that people have neglected.

Dr. BOND. I think that is a very good point. I know of no experimental data on it at all. If the energy is sufficiently low, the radiation will be less effective for the same air dose.

Dr. BORG. There is one point in Dr. Bond's presentation which strikes me as being very noteworthy, indeed, and this is his comment about neutrons and their presumptive depth dose curve, and the resultant biological effect. Thus near the lethal dose range, bomb neutrons can almost be thrown away. Such a conclusion would be a surprise to some people. On the other hand, there are some bomb effects, perhaps at very high doses of neutrons and gamma rays where there is primary damage to the cortex of the central nervous system fairly close to the body surface. In these instances neutrons as well as gamma rays might be effective. But for cases where transmission of neutron effects through the whole body is required, it looks like the self-shielding factor that is implied by this depth dose curve must markedly reduce the whole-body radiation effects due to the neutrons. There are few bombs where the neutron rep divided by this factor will become very important.

Dr. BOND. I hasten to add, however, that again the curves I showed were calculated curves, and I have every reason to believe and physicists have assured me that these would be the worst case in the field. Again, we have no measured neutron depth dose curves in the field.

Dr. CRONKITE. There is another problem of practical importance not directly commented on, Dr. Bond, and that is what proportion, in a fallout field, of a dose is coming from close in and what proportion from far out? How big an area does one have to clear if you are at the center to effectively reduce the dose by a factor of 10 or 2 or whatever you wish?

Dr. BOND. I have seen several estimates of this, and Dr. Robertson in our laboratory carried out a calculation along these lines. The answer one gets depends to a large extent upon the assumptions made in the calculation. It appears definitely that at least half of the radiation comes from 10 meters away or more. I have seen other estimates that most of the radiation comes from the order of 100 meters or more.

Dr. BORG. The method I talked about could answer this question. The machine calculations, if carried further would be susceptible to analysis in this regard. You could tell not only what the 4π spectrum was at a given point, but from what solid angle the radiation was coming. If there is a general interest in this, some of the people who generated the original material might be able to carry the problem further. A comparable solution can be made for the initial gamma case: that is, a spectrum can be generated as a function of angular distribution, and for penetration through shielding this information might also be valuable. I don't think it has ever been done, but it certainly could be done.

Dr. BOND. This information is of great practical value in regard to the question that was asked, how much of an area must be cleared. I will say again that in terms of the depth dose pattern obtained in the individual as we saw under these conditions we apply the curves corrected for inverse square. So as far as the depth dose patterns are concerned, it does not matter from what distances the radiation effectively originates.

Dr. CRONKITE. Are there any further questions or comments on any of the papers of this afternoon? I notice that everybody so far has rather artfully dodged what I still think is a rather essential part of this symposium, to somehow or other come along with an estimate or guestimate of really how effective is radiation in man. I would choose not to answer this myself, but I see Col. Maxwell, who has had a lot to do with fallout. After all, how can one assess the hazard if you are not willing to comment somewhat on the effectiveness in man? I think it is self-evident that any reanalysis of the Japanese data has to take in a lot of practical considerations about where the individuals were, how far they were away from the bomb, how close they were to large concrete buildings, and so on. It may be a completely impossible question to answer, but I am sure that someone here is not so shy, other than Dr. Borg, that they are not willing to comment on the subject.

Dr. BORG. Utilizing data concerning weapon type, yield, burst height, and atmospheric density, I calculated gamma-distance curves for the Nagasaki bomb. Casualties have been reported in some detail for the Fuchi school in Nagasaki (Oughtsozon, A. W., and Warren, S., "Medical Effects of the Atomic Bomb in Japan," New York, McGraw-Hill, 1956, p. 68). There were some wooden sheds in that building where apparently, as Dr. Bond and I looked it over the other day, approximately 50 percent of the inhabitants of the wooden buildings—about 30 in number—died of radiation disease, and were presumably exposed fully to bomb nuclear radiations only. At this distance the free air calculation that I made was 600 roentgens. This is a weapon, whether we choose in general to discount neutrons or not, which did not have a large neutron contribution. If the remaining concrete structure of the school nearby served to decrease that dose even further, and if the tiles and roofing, even if they didn't account for a great deal of shielding, had any effect, I would say about 100 or 150 roentgens less than 600 roentgens would be the LD–50 for man for initial gamma radiation. The mortality figures are not a good statistical series, I will admit.

Dr. CRONKITE. I cannot refrain from commenting somewhat further that I see people here sitting who are responsible for writing handbooks and who put these numbers in them. I would not want to go so far as to call them by name, but possibly they would like to comment.

Mr. LINDWARM (Chemical Warfare Laboratories). Obviously the sort of question that Dr. Cronkite is pushing for is one, is there such a thing that can be drawn up at the pres-

ent time, a so-called table of effects other than LD-50's. In other words, gamma dose versus biological effects for considerations other than LD-50's. This kind of information commanders in the field would like to know from casualty assessment points of view.

Topic III

Biological Repair Factor

REPAIR ASSOCIATED WITH THE EROSIVE EFFECTS OF FALLOUT DAMAGE IN INDIVIDUALS AND POPULATION GROUPS

By Paul S. Henshaw

U. S. Atomic Energy Commission, Washington, D. C.

This symposium is devoted to damage caused in living systems by radiation from fallout. Yesterday, attention was given to the physical aspects of problems involved. Today, we are *turning to the biological aspects—particularly repair*. Because fallout is pervasive and effective through time, its action is more subtle. This action is clarified, however, by considering certain features of both acute and protracted irradiation exposures.

The ideas I have to present deal with the problems of recovery associated with three types of *erosive effects* caused by radiation: (1) Those that occur following irradiation sickness; (2) those connected in a way with life shortening; and (3) those involved with reduced *fitness in population groups*. The comments I shall make will be more in terms of interpretation than in mathematical treatments such as have been used frequently during the past several months.

Figure 1 gives orientation with respect to phases of the injury response in human beings following acute irradiation exposure—phases

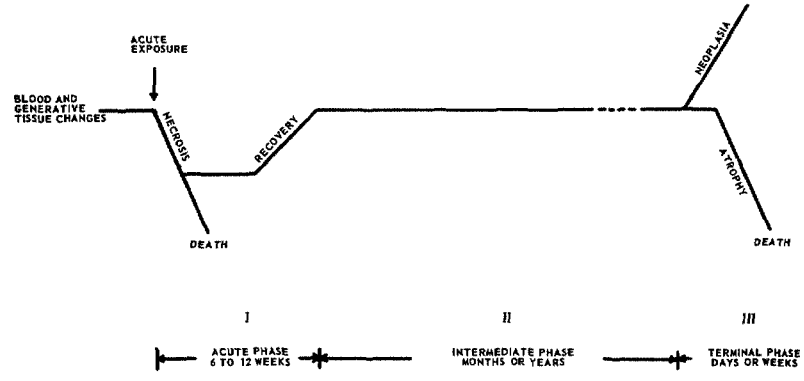

FIGURE 1.—*Pattern of irradiation injury.*

which probably would be typical for most or all mammalian forms. The figure gives orientation also with respect to the integrated effects of key reactions, such as the blood dyscrasias, epithelial sloughing in the gastrointestinal tract, hemorrhage, blood clotting failure, epilation, sterility, cataract, etc. It is of practical, and also some scientific, significance to deal with the net effects of radiation in individuals and in population groups, as a means of gaining impressions of what individuals and groupings of people can do following irradiation.

Implications of Figure 1 are: (1) That the necrosis in growing tissues (bone marrow, lymph nodes, spleen, gastrointestinal epithelium, germinal epithelium of the testis, and skin) is precipitous following near lethal irradiation exposures; (2) that if the necrosis is excessive, death will result; (3) that if it is not too extreme, repair by means of mitosis and tissue replenishment will take place, reaching the normal range in a matter of weeks or months; and (4) that the acute reaction of degeneration and repair (Phase I) is followed by a long period of apparent normality (Phase II) and, in turn, by a terminal period (Phase III)—which, aside from infections and accidents, involves degenerative diseases and neoplasia mainly. Inherent, but not made evident by the figure, is the fact that the Intermediate Phase is foreshortened by exposure to radiation, and that the Terminal Phase involves the same kinds of features as are present irrespective of radiation.

Dealing with short duration exposures (minutes, seconds, or less), Figure 2 pictures performance ability during the Acute Phase—the phase of tissue necrosis and replenishment (especially in the gastrointestinal tract and hemopoietic organs). The scale for performance ability—*work capacity* as it is labeled—is somewhat arbitrary, but, as will be seen, is nevertheless useful. It was developed in the following manner. Descriptive terms or expressions

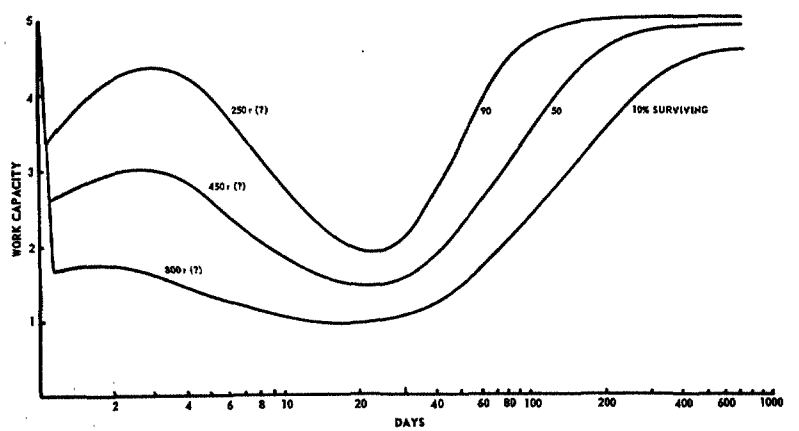

FIGURE 2.—*Work capacity—Acute exposure.*

were chosen to represent different levels of work capacity or illness as follows:

a. Reserve energy—ability to do a 10-mile march.
b. Normal work—ability to perform a regular day's work.
c. Lassitude and easy fatigue.
d. Illness (sickness, discomfort, anxiety) but capable of self care.
e. Illness, but with need of nursing care.
f. Death.

These terms or expressions were then arranged on a scale in order from 5 downward, respectively, as shown in the figure, and, on the basis of clinical, hematological and histopathological information, choices were made as to the level at which the majority of people exposed would be expected to exist at different times after different acute exposures. This gave the curves as shown. Since some interpolation was necessary to obtain smooth curves and since the descriptive terms did not have precisely uniform quantitative significance, the values on the scale cannot be said to represent the descriptive terms concretely or vice versa. The development as a whole, however, gives a consistent picture, and one that has meaning.

In terms of integrated effects of near lethal dose of radiation of short duration on the body as a whole, the following can be said: (1) That there is an immediate condition of sickness or shock; (2) that the degree of illness varies directly with dose; (3) that the illness may be less during the second to fourth or fifth days; (4) that during the second and third weeks there is a precipitous fall in fitness which coincides with the cascade of tissue necrosis; and (5) that during the fourth week, recovery sets in (in survivors), which then for certain organs (gastrointestinal and hemopoietic which in particular are of vital importance) reaches the normal range in 2 to 4 months.

Turning to protracted or intermittent exposures, Figure 3 shows performance ability at different times in connection with daily treatments of different amounts, using the same plan as employed in connection with Figure 2. Here a shock response is totally absent due to any dramatic effects at the beginning, but work capacity falls with accumulation of the integrated effects. Implications of the curves are: (1) That for doses of 20 r per day, work capacity becomes noticeably reduced in 2 to 3 weeks with death occurring as an end result at about 2 months; (2) that for doses of 5 r per day, reduction in work capacity is barely noticeable in 3 months but that it does fall gradually with death occurring in 3 to 4 years; and (3) that for doses of 1 r per day there is no noticeable reduction in work capacity in 3 years time.

Of importance in connection with protracted radiation is the fact that damage and repair—more particularly, cell destruction and replacement—go along together in the growing tissues, and also the fact that defects in the organism begin to show only when the rate of destruction exceeds the rate of repair. From Figure 3, there is indication that the threshold of injury to the organism, so far as work capacity is concerned, is about 1 r per day, and from this it follows that the resiliency of the growing tissues in general—that is, their maximum capacity to regenerate—must be offset or counteracted effectively by radiation doses in the neighborhood of 1 r per day.

How great the resiliency of tissues may be and how much reserve capacity exists in at least some of the organs, are indicated by the following facts (developed from animal experiments mainly): Three-fourths of a liver can be removed by surgery and a whole liver will regenerate; one and a half kidneys can be extirpated without reducing normal excretory efficiency; and a full body content of blood can be drawn off every two and a half weeks (i. e., blood being removed at intervals during such periods) without distortion or reduction of the peripheral blood picture. On the basis of such information, it would seem that the subthreshold or *subliminal* effects of protracted irradiation may be quite large in terms of cell destruction and replacement—that is, beyond that which takes place during the normal course of

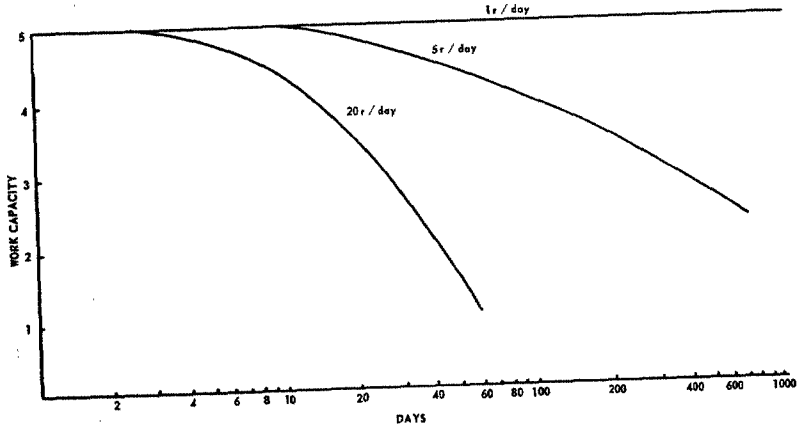

FIGURE 3.—*Work capacity—Protracted exposure.*

life. This we identify as the first type of erosive effect we have in mind. The consequence is a *racing of the regenerative motor,* so to speak—actually, a consumption of a portion of the regenerative reserve which is drawn upon in the case of body emergencies.

The second erosive effect to be considered is very different in character. That it exists as a reality there is no longer any doubt, but as yet the experimental proof for it is somewhat sketchy. It can be identified most easily by reference to the Intermediate Phase as set off in Figure 1. If damage from acute exposure has not been too great, the post irradiation sickness phase is one of apparent normality—yet the length of this phase varies inversely with the size of dose administered even before the sickness developed. Bearing in mind that tissue replacement appears to approach normal, attention is drawn by elimination to *residual quality* of the growing tissue as a basis for understanding the life shortening process.

It is known that cells are killed or modified as a result of irradiation by induction of biochemical changes and/or mutations (a very specific kind of biochemical change). It is to be presumed that life shortening must be produced by the same means inasmuch as the induction of biochemical and mutational changes appear to be the main means by which radiobiologic changes are induced. Choosing between these two possibilities, it appears unlikely that biochemical changes, which are produced so very precisely in accordance with dose at the time of exposure, should persist with the same preciseness through the comparatively quite long Acute and Intermediate Phases to cut off life prematurely in the Terminal Phase. This leaves mutational change as the most likely radiobiologic change that provides a link between irradiation and premature death which occurs months or years later. As will be seen, it also provides a plausible basis for explaining radiation life shortening, and, at the same time, what is identified as a third type of erosive action.

Since it is known that radiation produces mutations in proliferating cells, that certain of the induced types are sublethal and therefore able to continue with proliferation, and that

most of the mutations that persist are of the deleterious type involving reduced cellular efficiency and leading to reduced organ and organism efficiency, there exists a means not only for the persistence of irradiation effects throughout life, but also for degenerative changes that lead to earlier times of death. As a consequence of irradiation, defective mutant cells lie scattered at random throughout the growing tissue elements. These are in competition with nonmutated cells in the same localities and it is necessary to assume that at least part of these are successful in starting strains of cells that develop into scattered islands of cells, some widely separated and others overlapping and even diffusing into each other. The net effect over time is a gradual tissue transformation, involving at the same time reduced organ efficiency. Death is the natural consequence when tissue transformation reaches a point of organ failure in a vital part.

The erosive effect in this case is secondary to the initial irradiation effect and involves strictly biologic action—the multiplication and spread of less efficient mutant cells. With continued power to proliferate, but with reduced power to perform specialized functions as required by the host organism, the organism is jeopardized increasingly so far as its ability to cope with the rigors of life is concerned. For this type of erosive effect—to the extent that is exists—recovery consists of competition between the normal and mutated cells or tissues, and insofar as growth of normal tissues dominates growth of the abnormal, it can be said that repair takes place. But, of significance is the fact that there is a systematic correlation between size of dose (acute) and amount of life-shortening. This means that repair, if any takes place, is also systematic and bears a close relationship to the amount of effect produced.

The third type of erosive effect to be considered is still different in character. It involves effects on populations rather than effects on individuals alone. Of significance here, radiation appears to act on population groups in much the same way that it acts on individuals but with certain important differences.

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Mutations are produced in germ-line cells of the reproductive system the same as in other proliferating tissues. The mutations induced in both cases consist of three types: The *lethal,* which culminate in early cell death and thus drop out of the picture very quickly; the *deleterious,* which are responsible for reduced efficiency in cells so far as well-being of the organism is concerned, but not for preventing proliferation; and the comparatively very rare *beneficial* type. Deleterious mutations, as a consequence of mitosis of affected cells and of breeding, become spread in the aggregate germ plasm of the population—sometimes called the *gene pool*—in much the same way that they are spread in individuals by proliferation alone. Deleterious mutant cells, both in the germ line and in the soma, multiply and tend to exert increasingly depressive effects on vigor and stamina—vigor and stamina of the population group in one case, and of individuals in the other. An important difference, however, is that in populations there is opportunity for selection of the type that, in connection with mating, favors the more able and discriminates against the less—a type of process for which there is no counterpart in individuals. Favorable selection is benefited additionally by retention of any beneficial mutations that occur. The fact that selection can occur at the population level and that such does not occur in connection with mutations in individuals, furnishes some explanation of why species have the opportunity of living on indefinitely whereas organisms must inevitably die.

The erosive effects involved here are similar to those of the first type described, in that continuous irradiation tends to use up or consume a reserve, and that recovery consists of counteracting this influence in such a way as to maintain a suitable margin of safety. In obtaining the benefits of variation that stem from the induction of mutations at random, by irradiation or otherwise, and from the selection which goes along automatically, it is obvious that a certain *load* of deleterious mutations is carried more or less continuously. It is obvious also that population groups can carry a given load

of deleterious mutations and at the same time survive with reasonable vigor. The level of the load carried—*genetic quality*—varies naturally with the rate at which deleterious mutations are added to and removed from the gene pool. If, then, it can be said that a stock has been weakened by an excess of deleterious mutations, the obvious steps for achieving recovery, or reduction of the load, would be decreasing the rate of mutogenesis and increasing the rate of removal. This means lowering exposure to mutogens like radiation on the one hand, and lessening the factors conducive to maintenance of the less fit on the other.

In summary, the attempt in this brief paper has been to consider some of the effects induced in living systems by radiation from pervasive sources such as fallout and the kinds of repair that accompany them. Three types of erosive effect have been identified: (1) That resulting from necrosis of growing tissues in individuals and leading to various forms of cytopenia, eventual organ failure and death; (2) that resulting from generalized degenerative change in growing tissues and culminating in earlier time of death; and (3) that resulting from mutational changes in the germ plasm of population groups and leading to loss of group vigor and stamina. It was pointed out that recovery in connection with the first consists of tissue replenishment—a biological factor; that recovery in connection with the second had no meaning with respect to mutational changes but did have in terms of competition between normal and mutated tissue materials; and that recovery in connection with the third consisted of lowering the rate of inducing mutations and also increasing the rate at which mutations are removed from the gene pool.

DISCUSSION

Paul S. Henshaw

Dr. Bond (Brookhaven). I would like to make a comment on the steepness of the slope that Dr. Henshaw presented that presumably applies to human beings. I cannot certainly argue with this slope, because I know of no definitive data on human beings that would allow us to define this. However, I would like to say that the slope for most mammals that have been studied is considerably steeper than the slope indicated by Dr. Henshaw. The factor that would apply in his case in his curve in going from LD-zero to LD-50 would be about two. In most mammals this factor is about 1.2.

Dr. Berlin. Thank you, Dr. Bond. Is there any other discussion? Dr. Henshaw?

Dr. Henshaw. My main experience has been with laboratory animals also. When I began to consider this question, I, too, had in mind that the time intervals involved, and indeed the slope of the curves presented, would be somewhat different from this picture as presented. But in asking these questions in relation to human beings and taking the fragments of information as we are able to get them from those who have had experience in connection with the Japanese damage, the few radiation accidents, and some other kinds of considerations, the indications are that the time intervals involved are longer in the case of human beings than in the usual laboratory animals, like rats and mice.

So I concur completely with the implications of the question that was raised.

RATE OF REPAIR OF RADIATION DAMAGE IN MICE

By John B. Storer

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In the following study the repair rates of tissues exposed to ionizing radiation were not measured directly. Rather, the rate of repair of damage contributing to the death of mice by two different mechanisms was determined. The end-points used were median lethal dose for death in the first 30 days following exposure (LD_{50}-30 days) and the median lethal dose for death in the first 100 hours after exposure (LD_{50}-100 hours). In the case of X-radiation exposure, deaths occurring in the first 100 hours are generally due to radiation damage to the gastrointestinal tract (in the dose range below 10,000 r). The later deaths associated with the LD_{50}-30 days are believed due primarily to hematopoietic damage. Thus, it was possible to measure indirectly the rate of repair of two radiosensitive organ systems.

The split-dose technique as described by a number of authors was used. Large groups of mice were exposed to an initial sublethal dose of X-rays and at various time intervals thereafter they were divided into subgroups and given graded doses of X-rays in order to determine the LD_{50}-30 days or LD_{50}-100 hours. The extent to which the LD_{50} was lower than the value for the control group then gave a measure of the amount of the damage remaining from the first exposure. This residual was necessarily measured in roentgens but since the damage is proportional to dose, this system of measure is probably sound. The residual was then converted to percent of initial damage by dividing "residual roentgens" by "initial roentgens" and multiplying by 100.

Thus:

$$R_t=\frac{LD_{50_c}-LD_{50_t}}{D_t}\times 100$$

where R_t=percent of initial damage (or dose) remaining at time t

D_t=initial dose in r
LD_{50_t}=LD_{50} dose at time t
LD_{50_c}=LD_{50} dose for controls

Female CF—1 mice, 2–3 months of age, were used throughout these studies. X-rays were delivered from a G. E. Maxitron operated at 250 KVP and 30 Ma. A Thoraeus II filter was added. HVL of the filtered beam was 2.6 mm Cu. Mice were exposed 15 at a time in a shallow Lucite cage curved on a radius of 50 cm. The TSD was 50 cm.

In the study utilizing 30-day lethality as the biological end-point, groups of mice were exposed to an initial dose of 100, 200 or 400 r. At intervals of 4, 8, 18, 32, 72, 144, 264, 504, 1,920, or 3,000 hours, the LD_{50}-30 days was determined in groups of these mice and (at similar intervals) in control mice from the same initial population. The results are shown in Table I. A plot of these data showed that the best empirical fit to a regression line was obtained when percent residual was plotted as a function of log time. The least squares calculation gave an equation

$$Y=106.03-26.79\log X$$

where Y is percent residual and X is time in hours. This line and the experimentally determined points are shown in Figure 1.

It is apparent from this line that, over the range of doses tested, the percent residual injury was independent of dose. Since this type of exponential is difficult to integrate into a biological model for the repair process, the data

TABLE I.—RESIDUAL INJURY AT VARIOUS TIMES AFTER RADIATION EXPOSURE AS MEASURED BY DEPRESSION OF THE LD_{50}-30 DAYS

Initial dose (r)	Time to second dose (hrs)	LD_{50}-30 days [1] (r)	Residual from first dose [2] (percent)
100	8	520	79
100	32	562	37
100	144 (6 days)	516	83
100	504 (21 days)	548	53
100	1,920 (80 days)	597	−6
200	8	445	77
200	144	477	61
200	504	491	55
200	1,920	524	34
400	4	236	91
400	8	261	85
400	18	315	71
400	32	318	70
400	72	366	58
400	264 (11 days)	562	9
400	1,920 (80 days)	565	7
400	3,100 (125 days)	509	3
None	4–264 hr control	599	
None	504 hr control	601	
None	1,920 hr control	591	
None	3,100 hr control	519	

[1] LD_{50}-30 days for the second dose.
[2] Percent of initial dose remaining at time of delivery of second dose.

were replotted as log percent residual *vs.* time. This resulted in a curved regression line that could be adequately described by the sum of two exponential lines. These lines were tentatively identified as representing a slow component and a fast component of the repair process. The experimentally determined values at each time interval were averaged and those for time intervals of 32 hours or more were plotted as a function of time. These values represent the slow component in repair. They are plotted with their calculated regression line in Figure 2. It can be seen from this figure that the half-time for repair for the slow component was about 33 days. By subtracting the contribution of the slow component from the total residual, it was possible to determine the half-time for repair of the fast component. These points and the regression line are shown in Figure 3. The half-time for repair of this component was about 9–10 hours.

In summary, when 30-day lethality was used as a biological end-point, there appeared to be two exponential components to the repair process, a slow component repairing with a half-time of about 30 days and a fast component repairing with a half-time of 9–10 hours. The size of the initial dose did not appear to influence the repair process. There was no evidence of a nonrepairing residual injury.

TABLE II.—RESIDUAL INJURY AT VARIOUS TIMES AFTER RADIATION EXPOSURE AS MEASURED BY DEPRESSION OF THE LD_{50}-100 HOURS

Initial dose (r)	Time to second dose (hrs)	LD_{50}-100 hrs [1] (r)	Control LD_{50}-100 hrs (r)	Residual from first dose [2] (percent)
400	2	770	1025	64
	4	872	1025	38
	4	894	1038	36
	8	784	1025	60
	24	884	1025	35
	72	931	1025	24
	240	944	1021	19
	336	979	1052	18
600	0.5	508	1038	88
	1	640	1038	66
	2	684	1038	69
	4	672	1038	61
	4	660	1065	68
	8	648	1038	65
	8	730	1065	56
	24	780	1038	43
	24	765	1065	50
	48	780	1038	43
	48	900	1065	28
	72	810	1038	38
	72	830	1065	39
800	2	336	1025	86
	4	394	1025	79
	4	463	1038	72
	24	534	1038	63

[1] LD_{50}-100 hours for the second dose.
[2] Percent of initial dose remaining at time of delivery of second dose.

In the second series of studies, mice were exposed to 400, 600 or 800 r and at time intervals of 2, 4, 8, 24, 72, 240, or 336 hours a second dose was delivered to determine the LD_{50}-100 hours. In this case, we were dealing primarily with injury to the gastrointestinal tract, since it is injury to this system that results in survival times of this magnitude. Residual damage was calculated as before. Table II summarizes the results of these studies. As in the case of the LD_{50}-30 day studies, the best empirical regression line relating percent residual to time was of the type

$$R = a + b \log t$$

where R=percent residual and

t =time between doses.

These data are plotted in Figure 4. The percent of residual injury at all times appeared to be related to the size of the initial dose. The higher the dose, the higher the percent residual injury measured at any time. This finding contrasts with the LD_{50}-30 day results which indicated no differences in percent residual with initial dose. The data were replotted as before as log percent residual *vs.* time. A curved regression line resulted which could be described as the sum of two exponentials. These were again identified as a fast and a slow component to the repair process. The points obtained at 24 or more hours after the initial dose were plotted and regression lines of the type

$$\log R = a + bt$$

were calculated. The slope constants for various sized initial doses did not differ significantly. They were averaged by weighting

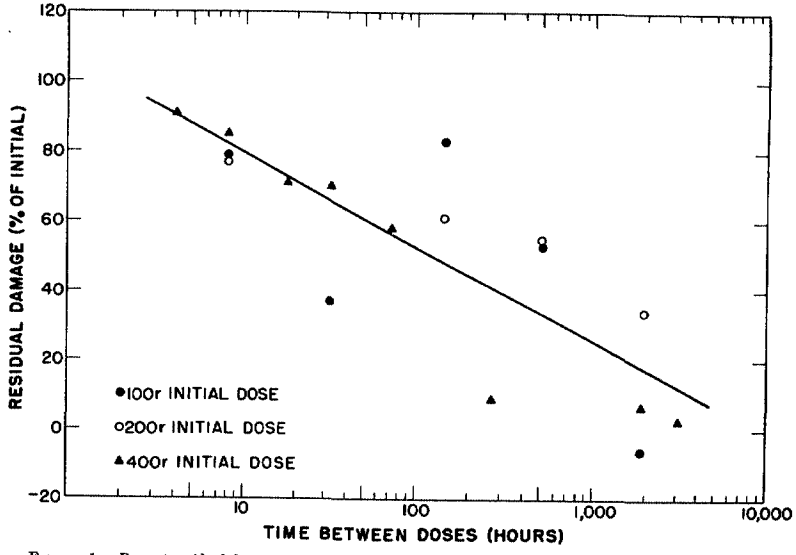

FIGURE 1.—*Percent residual damage as measured by depression of the LD_{50}-30 days as a function of log time.*

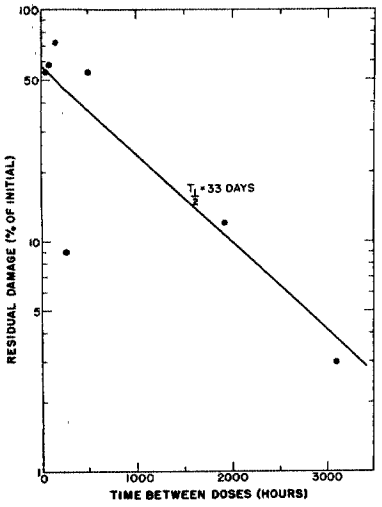

FIGURE 2.—*Slow component in repair of damage contributing to 30-day lethality.*

by the inverse of the variance and the common slope obtained. These data are plotted in Figure 5. It is thought that this plot represents the slow component of repair. The half-time was approximately 15 days as opposed to the half-time of 33 days obtained when the LD_{50}-30 days was used as the end-point. By subtracting the contribution of the slow component from the total effect, it was possible to obtain values for the fast component. These data are shown in Figure 6. The regression lines shown were arbitrarily forced through the zero time point since this point is based on 100 percent total residual at time 0 and probably has more validity than the other points. The 8-hour values fell badly out of line and accordingly were not used in this figure. The half-times for repair of the fast component were 1.2, 2.0, and 2.4 hours, respectively, for initial doses of 400, 600, and 800 r.

On the basis of the preceding results, the following tentative conclusions were reached:

1. The damage leading to death in the first 100 hours repairs at a faster rate than the damage responsible for 30-day lethality.
2. Repair in both cases appears to consist of two components, one component having a short half-time and the other a long half time.
3. Neither the extent of percent residual damage nor the repair half-time is affected by the size of the initial dose in

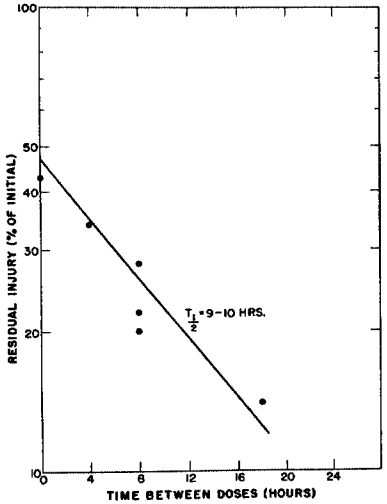

FIGURE 3.—*Fast component in repair of damage contributing to 30-day lethality.*

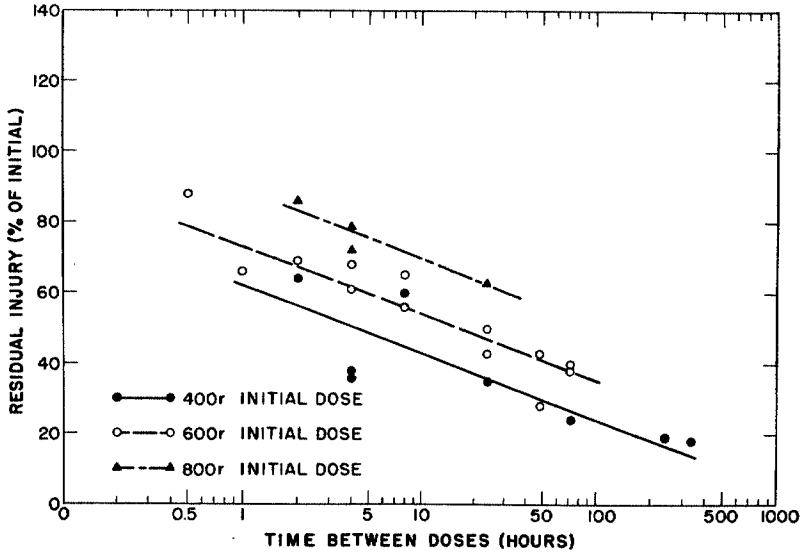

FIGURE 4.—*Percent residual damage as measured by depression of the LD_{50}-100 hours as a function of log time.*

the case of 30-day lethality. Both the extent of percent residual and the repair rate of the fast component are proportional to dose in the case of 100-hour death.

4. No evidence of a permanent level of residual damage was obtained in either study.
5. Since repair of damage leading to death by two different mechanisms shows different characteristics, it is likely that death from other mechanisms (such as premature aging) will also differ from the two mechanisms studied.
6. The residual injury leading to life shortening is probably not related to the residual injury measured in the present studies, since it is reasonably certain that a permanent residual injury causes life shortening. No permanent residual was detected in these studies.
7. Both the LD_{50}-30 days and the LD_{50}-100 hours should be dose rate dependent with the LD_{50}-100 hours being much more rate dependent because of the very short half-time for repair of the fast component. Preliminary studies have supported this view.

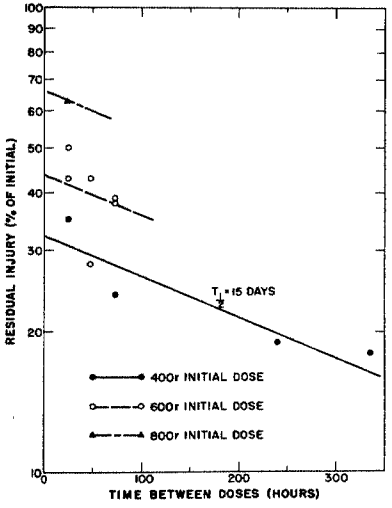

FIGURE 5.—*Slow component in repair of damage contributing to 100-hour lethality.*

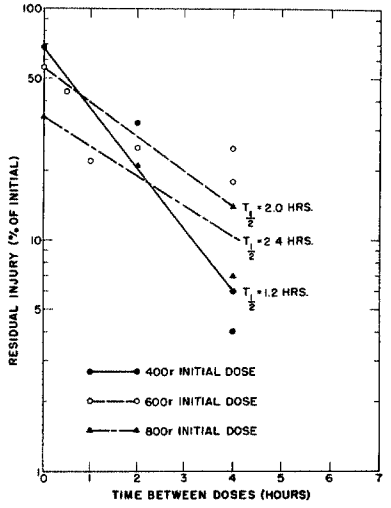

FIGURE 6.—*Fast component in repair of damage contributing to 100-hour lethality.*

APPENDUM

After the presentation of this paper, Drs. E. P. Cronkite and D. Borg, in a private conversation with the author, suggested that all the individual mice might repair their damage by a process showing a single characteristic half-time but that the half-times for the population might vary greatly and show a Gaussian distribution. This distribution might then explain the empirical fit of a line of the type

$$R = a + b \log t.$$

This suggestion seems plausible. Further calculations are in progress to determine whether the required ranges in half-times are reasonable.

DISCUSSION

J. B. Storer

Dr. CRONKITE. I just want to make the comment that following the study of the Marshallese where the changes in the blood were somewhat different than we had anticipated we wondered whether there might be a dose rate phenomenon, and simulated the exponentially decaying field with the 4 pi cobalt radiator at the Naval Medical Research Institute by successively withdrawing slugs. We did not do an LD-50 study, but just studying the changes in the peripheral blood of the dog exposed exponentially compared to dogs exposed at 15 r per minute there is practically no detectable difference in the pattern in the peripheral blood. All of our previous experience in dogs would indicate that we could judge the effective dose biologically very well by the changes in the peripheral blood.

Dr. SACHER. John Storer presented some very interesting data. We have a little bit of data done by a different method. I am not going to report on it, so I thought I might mention it now. The method is to use as a second test condition not a single dose LD-50, but the accumulated dose to death, giving daily dosages of about 100 r a day, such that the animals will survive approximately 30 days, accumulating 2 or 3 odd thousand roentgens.

Under these conditions, going out to about 4 months we find a persistent residue of damage on the order of about 10 percent. In other words, the groups that received the conditioning dose, usually sublethal or sometimes correctionally lethal, always could tolerate only 90 percent as much as the controls for this kind of run which was about 4 months. I think that this represents no inconsistency, but a response to a different test situation which stresses the organisms in a different way.

Dr. STORER. I would like to ask Dr. Cronkite over what period of time was this radiation dose delivered?

Dr. CRONKITE. It was given over identically the same period of time that the Marshallese were exposed, and starting at the same dose rate as the individuals were receiving as measured by the monitoring instruments. Actually a 48-hour period.

Dr. STORER. This would be fairly early. They were exposed to the fallout field fairly early so that the dose rates initially were quite high.

Dr. CRONKITE. The initial dose rate as I recall was approximately 3.5 r per hour.

APPROACHES TO THE QUANTITATIVE ESTIMATION OF RADIATION INJURY AND LETHALITY*

By GEORGE A. SACHER

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INTRODUCTION

There are serious difficulties in the way of a satisfactory quantitative theory of the lethal action of ionizing radiations. Since only the occurrence of an all-or-none end-point is observed, the yield of information from experiments is small. The nature of the end-point is ill-defined, because several kinds of injury contribute and the relationships among them that determine the boundary between viable and inviable states are not yet known. Moreover, several kinds of physiologic injury that have been studied are known to have non-linear dependence on dose and on time, especially when the injury approaches lethal levels. There is also the limitation on the predictability of response imposed by the differences between individuals and by the fluctuation of individual performance from time to time.

These questions must be answered in order to provide the foundation on which to build an adequate general theory in which lethality becomes an understandable consequence of the failure of adjustment of organisms to their environment. The most significant research contributions in the present period are those which throw light on one or another specific aspect of the total problem. The recovery process is being intensively studied, especially by the paired-dose technique [1–4]. The sensitivity of specific organ-systems or body regions is under active investigation [5, 6]. Theoretical and experimental approaches to the dynamics of turning-over cell populations have begun [7, 8]. The age-dependence of radiosensitivity is under investigation [9–11]. Strain and species differences in lethal responses are being explored [12–14], but differences with respect to specific physiologic responses are not yet under systematic study. The nature of the statistical relation of mortality to injury is being examined [15].

The above are a few examples of research under way on some topics that are of immediate relevance to the overall problem of radiation lethality. Many others have not been mentioned. Some problems have not yet been put under investigation. Foremost among these is the question of the way in which injury in several independent systems interacts to influence lethality. The outline of an integrated theory embracing all these aspects can be conceived, but a realization in any meaningful and useful sense is not yet within reach.

The mathematical treatment of radiation lethality presented below is to be regarded as an approach which is specifically devised to establish some properties of the lethal response to radiations. The characteristics of radiation lethality revealed by this type of analysis are, like the other physiologic characteristics enumerated above, part of the total response to be accounted for by an adequate theory. In short, the application of a mode of mathematical analysis to lethality does not constitute *ipso facto* a theory of that subject.

*Work performed under the auspices of the U. S. Atomic Energy Commission.

A GENERAL LINEAR MODEL OF RADIATION LETHALITY

When several increments of exposure are given sufficiently close together, the physiologic response is some function of all the dose increments and of the time intervals between them. We know that a single dose produces an injury response with a characteristic amplitude and time-course. The simplest hypothesis about the combined effect of a sequence of dosages is the additive hypothesis which states that the effect produced at a given time is the sum at that time of the effects of the dosages given separately in their proper positions in the sequence. This implies that every increment of dose produces an effect that is independent of the effects of the other increments.

An example of an experimental test of the additivity hypothesis is given in Figures 1 and 2. Figure 1 shows the weight effect in groups of male Sprague-Dawley rats given 4, 8, or 16 r 6 days per week beginning at 53 days of age [16]. Weight effect, $E(t)$, is defined as $E(t) = \log C(t) - \log R(t) - \log C_0 + \log R_0$ where logarithms are to base 10, $C(t)$ is control weight at time t, $R(t)$ is weight of irradiated animals at time t, and C_0 and R_0 are mean weights over the pretreatment period. In Figure 2B, the same measure of effect is applied to the weight response of male rats given a single dose of 200 r at 53 days of age. Figure 2A gives the result of a graphical differentiation of the $E(t)$ curve in Figure 1 for rats given 16 r daily. The daily dose weight curve can be accounted for on the basis that each daily dose produces by itself a weight response as in Figure 2A. If the curves in Figure 2A and 2B agree, the additivity hypothesis is upheld. There is agreement in overall amplitude of the first peak, and in the presence of two peaks of effect separated by a minimum at about 14 days. There is disagreement with respect to the depth of the minimum and also with respect to the stable value reached after 60 days postirradiation. This comparison is not quantitative, because of the difficulty in obtaining reproducibility of weight responses after single doses of 200 r or less. However, the comparison of hematologic responses to single and daily doses yields results of a similar nature. It is concluded [17] that the responses to small doses may be additive, but the departure from additivity increases with the size of the dose.

The properties of the linear model may be expressed in the following set of postulates [14, 17, 18].[1]

1. Radiation in general produces injury in several physiologic systems, and the degree of lethal injury is a sum of these.
2. Injury from other causes, and in particular accumulated injury due to ageing processes, also contributes additively to the lethal injury

[1] These are *postulated* properties of the *model* system. They are *hypotheses* about the properties of *real* biological systems. Their value as hypotheses is limited, as is discussed below.

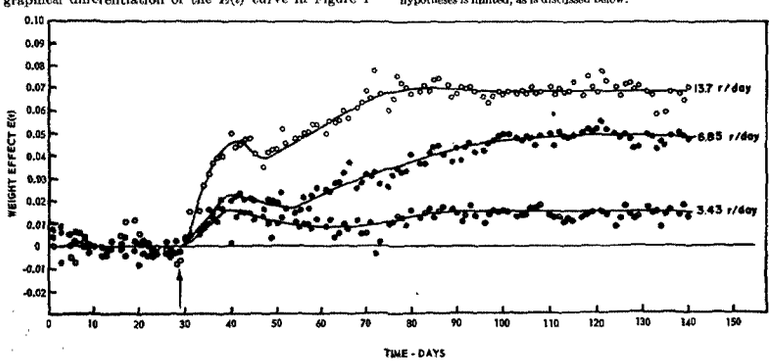

FIGURE 1.—*Effect of daily X-ray exposure on weight of growing rats, expressed as the difference of logarithms of experimental and control weights. Upward displacement signifies weight loss in treated groups.*

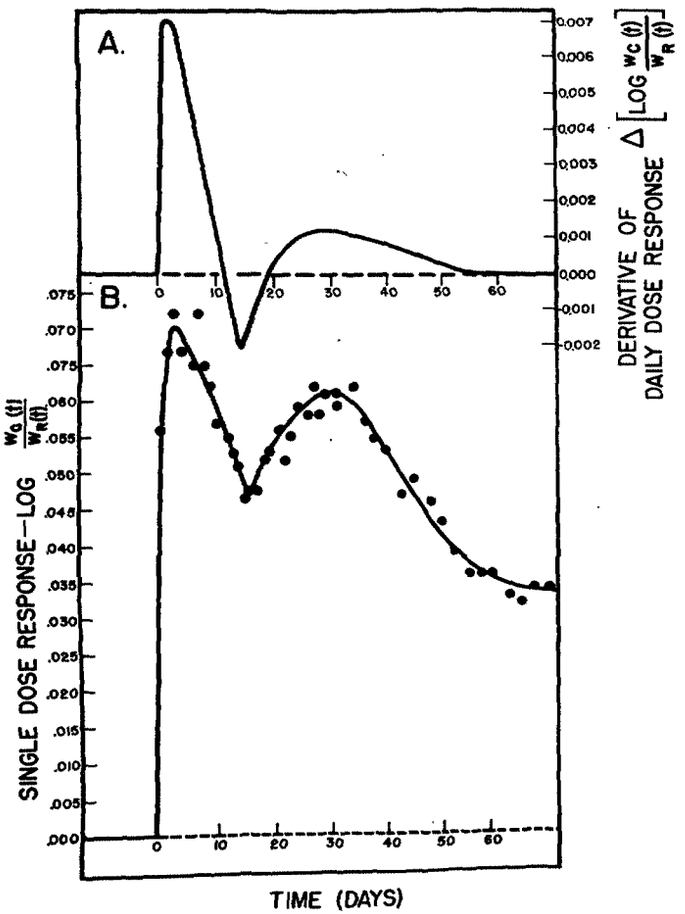

FIGURE 2.—*Observed and calculated response of growth curve following single exposure to X-rays. Lower curve is the observed effect of a single dose of 200 r. Upper curve is a graphical differentiation of the curve for daily exposure at 13.7 r/day in Figure 1.*

3. Death ensues when the lethal injury exceeds a critical level, the lethal bound.

These postulates are formally equivalent to the set used by Blair [19,20], but in the subsequent development, the writer and Blair follow different paths. Blair introduces quantitative assumptions for the injury and recovery processes and for the ageing process, to derive an explicit equation for the dependence of survival on exposure The alternative course followed here is to solve for an *empirical lethality function* using survival data for a given species. This lethality function would be a description of the course of lethal injury in the given species *if that species conformed to the postulates above.*

In previous presentations [14, 18], the integral equation of injury was obtained in the form

$$X(t)=\int_0^t I(t-\tau)\phi(\tau)d\tau+\beta t \qquad (1)$$

where $I(t-\tau)$ is the intensity of exposure at time $t-\tau$, $\phi(\tau)d\tau$ is the increment of injury appearing at time τ after instantaneous exposure to unit dose, βt is the accumulation of injury due to the natural ageing process.

Equation 1 introduced the assumptions that the accumulation of injury due to ageing is a linear function of age, and that the effectiveness of each increment of dose $Id\tau$ is proportional to I. We have since obtained evidence that these two assumptions may be incorrect [10, 15]. The integral equation of injury will therefore be written in the more general form

$$X(t)=\int^t E(I,t-\tau)\phi(\tau)d\tau+A(t) \qquad (2)$$

where $A(t)$ is the ageing function and $E(I,t-\tau)$ is the *effectiveness* of the dose increment $I(t-\tau)d\tau$.

The effectiveness function, in a form that takes account of effects that depend on the second power of the dose, is

$$E(I,t-\tau)=I(t-\tau)+ m\int_0^{t-\tau} I(t-\tau)I(t-\tau-\zeta)e^{-\mu(t-\tau-\zeta)}d\zeta \qquad (3)$$

When $I(t-\tau)$=constant$=I$, Equation 3 becomes

$$E(I,t-\tau)=I+mI^2\int_0^{t-\tau} e^{-\mu(t-\tau-\zeta)}d\zeta$$
$$=I+\frac{mI^2}{\mu}\left(1-e^{-\mu(t-\tau)}\right) \qquad (4)$$

The time constant $1/\mu$ is the mean time that damage can persist and be potentially able to combine with later damage to produce second-power injury. The value of $1/\mu$ is probably in the range from hours to a few days. In this case the term $(1-e^{-\mu(t-\tau)})$ in Equation 4 is negligibly different from unity over the time period of interest here. The effectiveness function then becomes, to a sufficient approximation

$$E(I)=I+\frac{mI^2}{\mu} \qquad (5)$$

This same approximation may be used when I is time-dependent, if the change of I with time is small over a time period on the order of $1/\mu$. When $E(I)$ is approximated by Equation 5, Equation 2 reduces to

$$X(t)=E(I)\int_0^t \phi(t-\tau)d\tau+A(t) \qquad (6)$$

Death occurs when $X(t)$ reaches a critical value, the lethal bound, which can be set equal to unity. Equation 6 becomes,

$$1=E(I)\int_0^{t^*} \phi(t-\tau)d\tau+A(t^*) \qquad (7)$$

where t^* is now a definite mean survival time corresponding to an exposure at constant daily dose I. The lethality function for constant exposure, called the cumulant lethality function, C_L, is immediately found to be

$$C_L=\int_0^{t^*}\phi(\tau)d\tau=\frac{1}{E(I)}\left[1-A(t^*)\right] \qquad (8)$$

The ageing function is very imperfectly known, but can provisionally be specified, in view of available data (10), as

$$A(t)=\frac{t^*+b+q(t^*+b)^2}{t_0+b+q(t_0+b)^2} \qquad (9)$$

where b is the age at the beginning of exposure, and t_0 is the control survival. The ageing function becomes equal to the lethal bound, and therefore to unity, when $t^*=t_0$.

PROPERTIES OF THE LETHALITY FUNCTIONS AND SOME IMPLICATIONS FOR PREDICTION

Let us first summarize the previous developments. If samples from a homogeneous population are exposed to different constant intensities I, we can deduce from data on daily duration-of-life exposures a lethality function of the form

$$C_L=\frac{1}{E(I)}\left[1-A(t^*)\right] \qquad (10)$$

When explicit expressions are assigned for $E(I)$ and $A(t)$, then, with a set of known values of I and t^* we obtain a numerical estimate of a *cumulant lethality function* that describes the course injury would follow *in a model system conforming to the postulates stated above.*

The most extensive lethality data over a wide range of daily dosages are those for ABC male mice given X-ray dosages ranging from 20 to 1,000 r/day [18]. The daily dosages and survival times are given in Table 1. The corresponding values of the cumulant lethality are also given in Table 1. The cumulant is computed on the assumptions that

$$A(t^*)=t^*/t_0 \qquad (11)$$

where t_0 is the mean survival time of controls, and

$$E(I)=I \qquad (12)$$

These expressions for $E(I)$ and $A(t^*)$ are not realistic, as noted above, but the discussion below will center on some properties of the lethality function that are not qualitatively affected by any bias introduced by these approximations.

The cumulant values for the ABC mice are plotted in Figure 3. The function obtained is not a simple curve. There is a sharp flexion at about 15 days and another near 40 days. The flattening of the curve as drawn between 80 and 220 days is not arbitrary, but is based on certain properties of the survival of ABC mice in this time period [18], and on the behavior of other strains and species, as will be shown below (Figure 5). This plateau period implies the existence of a "silent period" between the acute and chronic phases of injury. Evidence for such a silent period is also found in the recovery studies of Storer [1] and others.

TABLE 1.—TABULATION OF THE SURVIVAL OF ABC MALE MICE GIVEN DAILY X-RAY EXPOSURE FOR THE DURATION OF LIFE, AND OF THE CUMULANT LETHALITY VALUES DETERMINED

Mean daily dose [a] (r)	Mean after-survival (days)	Cumulant lethality [b] (r/day)$^{-1}$
0	538	
16.5	213	0.0366
33.3	78.8	.0256
41.6	36.8	.0224
50.2	39.2	.0185
66.7	29.9	.0142
88.3	20.6	.0109
103	15.2	.0094
174	11.5	.0056
275	8.0	.0036
343	7.0	.0029
500	5.2	.0020
1000	3.8	.0010

[a] Exposures given 6 days per week.

[b] Using Equation 10 with $E(I)=I$ and $A(t^*)=\frac{t^*}{t_0}$.

The *impulse function* obtained from these data by numerical differentiation is plotted in Figure 4. This is an estimate of the course of injury after a single exposure. The existence of two major peaks of injury, at 15 and 40 days, is indicated. The minimum at about 120 days again represents the "silent period" noted above.

Cumulant values were computed for all available data on experimental animals given uniform duration-of-life exposure [14] and are presented graphically in Figure 5. The cumulant values are here plotted on a log-log scale.

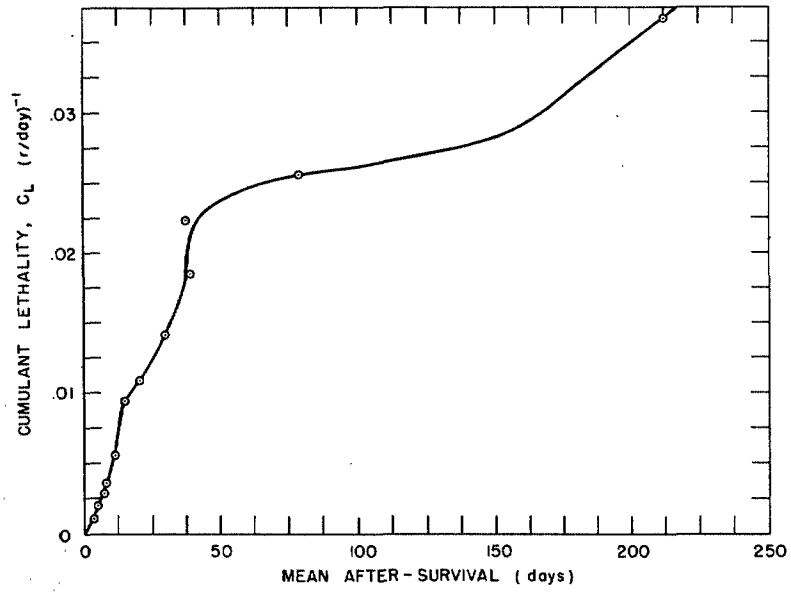

FIGURE 3.—*Cumulant lethality function, for ABC male mice exposed to daily dosages ranging from 20 to 1000 r/day.*

It is evident that the lethality cumulant is species-characteristic, for each species has a consistent pattern of behavior, and the cumulants for different species differ in form.

What are the implications of these observations for the mathematical theory? First, the lethality functions cannot be adequately represented by simple mathematical expressions. Thus, the impulse lethality function (fig. 4) for the ABC mouse does not agree well with the formula of the type offered by Blair [19, 20] to describe this function

$$X=C_1e^{-kt}+C_2 \qquad (13)$$

where C_1, C_2 and k are constants. This expression would put the peak of injury at time zero. Blair acknowledges the existence of a delay in the appearance of recoverable injury [19] but does not take account of it in his mathematical developments. A simple way of introducing the delayed appearance of injury in an explicit formula is to assume that injury becomes manifest as an exponentially decreasing function of the time after exposure [21],[2]

$$V(t)=\frac{1}{a}e^{-at} \qquad (14)$$

where $V(t)$ is the amount of injury that appears at time t after exposure to unit dose. If this is combined with the assumptions that (*a*) recovery is linear, and (*b*) there is a non-recovering component, we obtain an expression

[2] A modification of Blair's theory based on this consideration has been developed by Dr. D. Mewissen (personal communication).

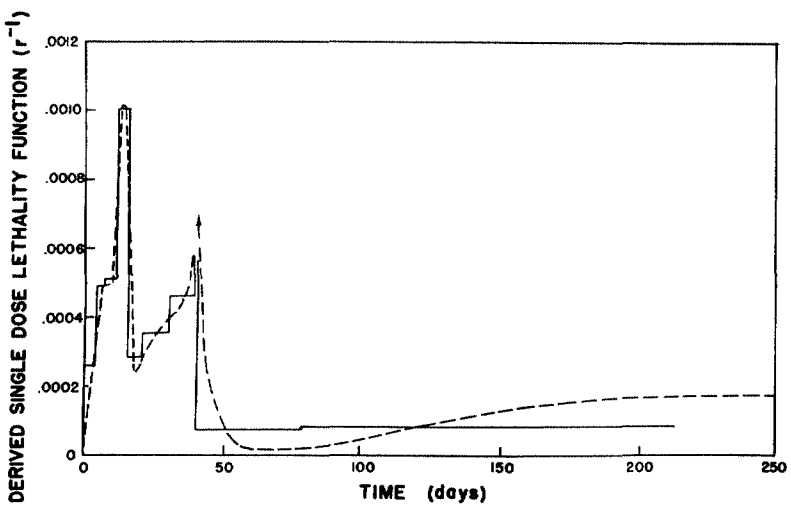

FIGURE 4.—*Impulse lethality function, obtained by graphical differentiation of the curve in Figure 3.*

for the impulse function which is of the catenary form

$$X=C_1(e^{-\alpha t}-e^{-\beta t})+C_2 \qquad (15)$$

where β is the recovery rate.

This expression would perhaps give a fair description of an individual injury process, but an adequate description of the empirically determined impulse function during the first 100 days (fig. 4) would require at least two catenary terms. Even this more elaborate expression would fail to describe events accurately between 50 and 200 days, in view of evidence that the non-recovering lethal effect has a mean latent time of about 200 days for the mouse and rat, and a greater magnitude for the dog and guinea pig [14]. This accounts for the extended plateau region in the cumulant functions of the various species shown in Figure 5. This latency property of the non-recovering injury is not present in the Blair formulation. Since the nonrecovering injury is manifested in neoplasia and degenerative disease, the delay in its appearance, as seen in Figures 3, 4, and 5, is the expected behavior.

Our actual problem is to estimate the lethality functions for man. This does not mean that we need to trace a complicated curve. In fact the important parameters needed can be reduced to a set such as the following.

1. The sensitivity of the recoverable injury, as measured by the plateau level of the cumulant function.
2. The sensitivity of the nonrecoverable injury, as measured by the constants of of the final rising branch of the cumulant function.
3. The mean latent time of the recoverable injury.
4. The mean latent time of the nonrecoverable injury.

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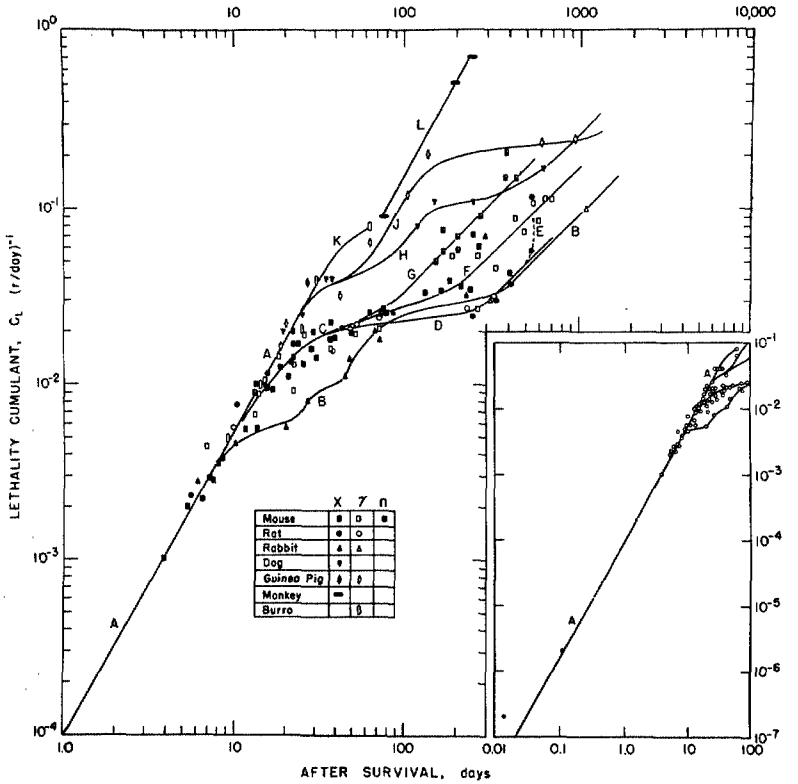

FIGURE 5.—*Cumulant lethality functions for several experimental species, based on all available data. The curves are plotted on a log-log grid. Note (a) lack of species differences in first 7 days, (b) existence of a plateau region indicating partial equilibration of the recoverable injury, (c) large species differences in the plateau level of injury, (d) appearance of non-recoverable injury, as indicated by final rising branch, after a latent period of several hundred days.*

The contribution of the empirical analysis presented thus far is to suggest that these parameters are independent and must be determined separately. According to present evidence, the LD_{50} is a poor predicter of the later phase of the recovering injury, and there is as yet no evidence that it has predictive value for the true chronic injury, which is expressed in neoplasia and degenerative disease.

There would appear to be only one methodologically sound approach to the problem of predicting lethality, that of pursuing the consequences of the fundamental postulate that radiation lethality is a consequence of physiologic injury. Therefore a correct description of lethality can only follow from correct conceptions of the nature of physiologic injury and recovery. Despite the complexities that have been pointed out (and others that have not been considered) the prediction of lethal effects in man is possible if we can identify the physiologic correlates of the various components of the lethality function.

DETERMINATION OF THE CUMULANT LETHALITY FUNCTION FROM DATA ON TIME-DEPENDENT EXPOSURES

In the previous section, lethality functions for several species were obtained from data on duration-of-life exposure at a constant rate. It was possible to determine thereby some general properties of the injury process. However, the validity of the basic postulates of the linear model was not tested thereby. The postulate of linearity of mechanism can be tested by using data from a number of different exposure patterns.

In this section it is shown that the cumulant lethality function may be deduced from data on time-dependent exposures. The comparison of the derived cumulant function with one determined directly permits a test of the consistency of the model.

To simplify the derivation, the integral equation for injury will be solved for time-dependent exposure on the assumption of linear effectiveness (Equation 12).

The exposure intensity function, $f(t)$, will be written as a sum of exponentials [3]

$$f(t)=\sum_{i=1}^{n} A_i e^{-\alpha_i t} \tag{16}$$

with

$$\sum_{i=1}^{n} A_i=1 \tag{17}$$

[3] This representation is convenient for the application to exposures from retained isotopes. Other classes of exposure patterns (linear rise or fall, square wave, power function, etc.) can also be solved.

We define the new variable

$$Z=\frac{1}{\bar{I}_e}\left[1-A(t^*)\right] \tag{18}$$

Equation 2 then becomes, with $X(t)=1$,

$$Z=\int_0^{t^*}\phi(\tau)\textstyle\sum_i A_i \exp\left[-\alpha_i(t^*-\tau)\right]d\tau \tag{19}$$

$$=\sum_i A_i e^{-\alpha_i t^*}\int_0^{t^*}\phi(\tau)e^{\alpha_i\tau}d\tau \tag{20}$$

There is no need for the general solution because the limitations of the empirical data preclude the use of more than two exponential terms in $f(t)$. The case that $f(t)$ has two exponentials is now considered.

Let us define the new variable

$$P_i=A_i e^{-\alpha_i t^*}\int_0^{t^*}\phi(\tau)e^{\alpha_i\tau}d\tau \tag{21}$$

The derivative of P_i with respect to t^* may be written

$$DP_i=\alpha_i A_i e^{-\alpha_i t^*}\int_0^{t^*}\phi(\tau)e^{\alpha_i\tau}d\tau+A_i\phi(t^*) \tag{22}$$

$$=-\alpha_i P_i+A_i\phi \tag{23}$$

where

$$D=\frac{d}{dt^*} \tag{24}$$

$$\phi=\phi(t^*) \tag{25}$$

With $n=2$, Equation 20 becomes

$$Z=P_1+P_2 \tag{26}$$

We also obtain readily

$$DZ=-\alpha_1P_1-\alpha_2P_2+\phi \tag{27}$$

$$D^2Z=\alpha_1^2P_1+\alpha_2^2P_2-(\alpha_1A_1+\alpha_2A_2)\phi+D\phi \tag{28}$$

We can eliminate P_1 and P_2 between these to obtain

$$(D^2+(\alpha_1+\alpha_2)D+\alpha_1\alpha_2)Z=(D+\alpha_2A_1+\alpha_1A_2)\phi \tag{29}$$

In this differential equation, the α_i and A_i are known constants of the exposure function. In application to experimental data, Z is a known numerical function of the dosages, survival times and the ageing function. We therefore have a first-order linear differential equation in the unknown impulse lethality function, $\phi(t)$. Now let

$$\int_0^t Z(t)dt=Y \tag{30}$$

and integrate term by term, remembering that

$$\int_0^t \phi(t)dt=C_L(t) \tag{8}$$

We find

$$DZ+(\alpha_1+\alpha_2)Z+\alpha_1\alpha_2 Y=(D+\alpha_1A_2+\alpha_2A_1)C_L \tag{31}$$

This is solved for C_L as

$$C_L=e^{-Bt}\int_0^t e^{Bt}[DZ+(\alpha_1+\alpha_2)Z+\alpha_1\alpha_2 Y]dt \tag{32}$$

where B equals $\alpha_1A_2+\alpha_2A_1$.

The integral may be evaluated numerically, using numerical data to specify $X(t)$, or it may be evaluated analytically by first fitting $Z(t)$ with a graduation formula.

In the event that the model is validated for application in a given range of conditions, and given also that an acceptable estimate of C_L exists, then Equation 32 becomes a formula for estimating the expected relation of dose and survival time for a given time-pattern of exposure.

We [4] have evaluated C_L from some data obtained by the late Mr. Howard Walton [22] on the toxicity of Ru^{106} for CF–1 mice. Equation 32 was evaluated numerically, using the data given in Table 2. Figure 6 represents the numerical estimates of C_L based on the ruthenium data, and also an estimate of C_L obtained from data on CF–1 mice given constant daily dosages. In both cases $A(t)$ and $E(I)$ were assumed to be given by Equations 11 and 12 respectively. The scaling factor for best adjustment of the

[4] The assistance of Mr. Robert Schweisthal is gratefully acknowledged.

TABLE 2.—CUMULANT LETHALITY VALUES FOR CARWORTH FEMALE MICE (A) EXPOSED TO CONSTANT DAILY DOSE OF X-RAYS FOR THE DURATION OF LIFE AND (B) INJECTED WITH Ru^{106} *VIA* TAIL VEIN

A. Daily X-ray			B. Ru^{106} injections		
Mean daily dose [a] (r)	Mean after-survival (days)	Cumulant lethality [b] $(r/day)^{-1}$	Mean injected dose (μc/g)	Median after-survival (days)	Cumulant lethality [b] $(\mu c)^{-1}$
0	425		0	500	
33.3	63. 1	0. 0256	1. 42	140	2. 047
50.2	36. 4	. 0182	3. 21	37	. 704
66.7	21. 9	. 0142	4. 96	18	. 418
103	16. 4	. 0093	8. 94	12	. 151
174	13. 4	. 0056			

[a] Exposures given 6 days per week.

[b] Using Equation 10 with $E(I)=I$ and $A(t^*)=\frac{t^*}{t_0}$.

[c] The biologic decay of Ru^{106} was found by Walton [22] to be $I(t)=.38e^{-.0038t}+.62e^{-.122t}$. This was used to describe the time-course of exposure.

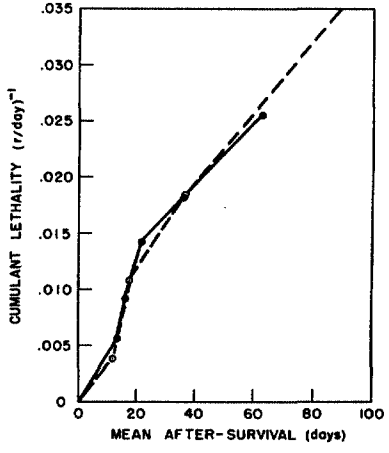

FIGURE 6.—*Cumulant lethality functions for Carworth female mice. Solid line—directly determined from data on survival at constant daily X-ray dosages. Dashed line—calculated from data on the survival following dosages of* Ru^{106}*, by use of Equation 32.*

Ru^{106} cumulant to the daily X-ray cumulant was found to be

$$1\ \mu c/g \text{ equivalent to } 38.5 \text{ rep/day}$$

The tissue dose received from retained Ru^{106} was estimated by Walton to be

$$1\ \mu c/g = 41.6 \text{ rep/day}$$

The estimated RBE of Ru^{106} with respect to 200 kvp X-rays is therefore

$$\text{RBE}=0.925$$

The two estimates of the cumulant function also agree in shape, although the C_L value from Ru^{106} at 140 days is perhaps somewhat high.

It would appear from these results that the linear model, despite its shortcomings, is useful in predicting the lethal effects of an unknown exposure pattern from the effects of a known pattern, if the patterns do not differ too greatly in form. This comparison is of some interest, because Ru^{106} has a fairly uniform distribution in the body. However, experiments with time-dependent exposures to external radiations are needed.

Fractionated exposure patterns are particular cases of time-dependent exposure, to which the methods described here can equally well be applied. However, the argument [23] that *only* fractionated exposure patterns should be used in lethality studies, in order to avoid the "wasted radiation" received in the last days of life, has no basis. The lethality functions exhibited above are estimates of the actual amount of injury present as a function of time after exposure. Hence, the injury arising from exposures received shortly before death makes its properly weighted contribution to the lethal injury. Inspection of Figure 4 will show also that this contribution in the first few days is actually comparatively small. Fractionated exposure, like time-dependent exposures in general, have an important role in the development of the theory of lethality, but this contribution will come from considerations quite unrelated to the wasted radiation concept.

CONCLUSION

The present status of the theory of radiation lethality was discussed briefly. The formal theory of lethality developed here was presented as an approach devised for the purpose of obtaining information about lethality, regarded as a physiologic process. It was shown that the lethality process is polyphasic, and that the several species studied appear to show considerable independent variation in the amplitudes of the different phases. The construction of an adequate lethality function for man requires knowledge of several independent parameters. The estimation of these parameters by nondestructive methods will be possible when they can be given a correct physiologic interpretation. The linear model may have utility for prediction of the effects of time-dependent exposure patterns, but its range of validity must first be determined by experiments with such patterns.

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RECOVERY FROM LATENT RADIATION INJURY IN RELATION TO PERMISSIBLE HUMAN EXPOSURE[1]

By H. A. BLAIR

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It is well established that following whole body exposure to ionizing radiation recovery from the consequent latent injury frequently occurs nearly exponentially with a half-time in the range from 3 to more than 20 days in the species which have been studied. The criterion used to measure recovery directly is the increase in size of a second dose sufficient to produce lethality as this dose is applied at greater intervals after a first sublethal dose. The injury so measured is called latent because it precedes the clinical syndrome of radiation injury and is measurable at present only in terms of radiation dose.

That recovery does not go to completion but leaves an irreparable residual is evidenced in either of two ways, by a permanent decrease of the lethal dose, or, by a shortening of life-span [1].

According to all indications recovery takes place similarly during, as well as following, exposure. For this reason it is a determining factor in how often successive doses may be given, or a protracted dose such as a fallout field may be sustained, without exceeding a given level of injury such as that caused by a single brief dose of selected magnitude. Application of this type of calculation to human populations requires a knowledge of recovery rate in man; but this is not known and no direct nonlethal method has yet been devised to obtain it. Presumably recovery in man resembles that in some of the other species but there is no way yet known to become assured on this point and there is a further complication in that measurements on animals present difficulties of interpretation which will be discussed now.

Until recently it was assumed that an animal subjected to whole body irradiation would recover in all parts, except the skin, at the same rate. However, Carsten and Noonan have shown in the rat that exposure of the abdomen and lower levels only, leads to recovery with half-time just over 1 day [2], while exposure of the remainder of the body only, leads to recovery with half-time of 3 to 4 days [3]. Hagen and Simmons, [4] showed that the wholly exposed rat recovers with half-time about 7 days. These data suggest that recovery rate is possibly a function of volume irradiated. Data by Storer [5] in which the whole body of the mouse is exposed, but in which the dose is adjusted to give so-called intestinal death in about 4 days, also show a fast phase of recovery presumably associated with the abdominal region. Similar observations have been made by others. These data, contrary to those cited above, indicate the possibility that segments of the body recover, or tend to recover, at their own rates independently of whether or not other segments are irradiated.

Non-homogeneous recovery raises problems of measurement and interpretation which are illustrated in Figure 1. Assuming for simplicity that there are but two segments of the animal with different recovery rates it will be seen that the recovery curve for the whole animal as defined by test doses will fall rapidly, initially, because of the fast segment A and

[1] This paper is based on work performed under contract with the United States Atomic Energy Commission at The University of Rochester Atomic Energy Project, Rochester, New York.

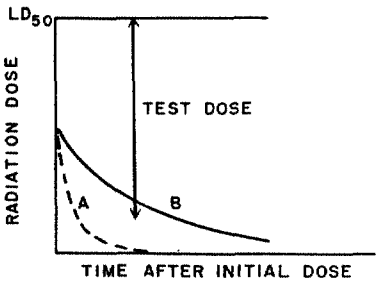

FIGURE 1.—*Following an initial whole body dose, about one-half LD_{50} in this case, recovery of the fastest segment is represented by curve A and of the slowest by curve B. Test doses adjusted to produce LD_{50} will define a recovery curve somewhere between A and B strongly influenced by A initially and later tending to run parallel to B. There are implications in these statements with respect to the summation of radiation effects produced in different segments of the body. This problem has been discussed (11).*

will later run parallel to, but below, B. The effect will be to make recovery appear too fast in the early stages and too complete in later stages. The first will indicate too short a half-time and the second will tend to obscure the residual injury. If curves A and B are simply exponential the experimental curve will not be.

There is suggested, perhaps, some peculiarity of abdominal radiation which alters its relative importance in some strains or species or under certain conditions since some observers find a fast early, presumably abdominal, component of recovery from whole body irradiation while others do not. Possibly recovery of segments is less independent in some species or under certain conditions, one of which may be dose size. In the gastro-intestional tract, for example, latent injury, as defined here, may have no meaning with respect to those doses which are sufficiently great to kill cells which are normally undergoing rapid replacement. Restoration of normal cell division and proliferation is presumably a process quite different from those involved in recovery of persisting cells. In any case, at this time, it is safe to assume only that recovery as determined experimentally by paired doses may be faster than that of the slowest recovering tissue and that the use of this recovery to predict the levels of injury for prolonged or intermittent exposures may underestimate them considerably. For this reason in choosing a tentative value for man probably it is advisable, in the absence of other information, to select a recovery half-time somewhat in excess of the longest known in mammals, which, at present, is that of the guinea pig—some 20 days.

There is another problem raised by Mole [6], who asserts that recovery rate per unit of injury is not a constant, as required by an exponential recovery hypothesis, but is a function of the level of injury and is slower with high initial doses, a result perhaps contrary to that of Storer discussed above. Mole's analysis of his data is not definitive but if his conclusion should be correct much more investigation of recovery from different dose levels would be required before the results could be applied to unknown situations. The bulk of the present evidence indicates that recovery is not a function of initial dose for doses less than about one-half LD_{50}.

Since this paper was presented verbally Davidson [7] has issued a report in which he shows a linear relation between whole body recovery rates in various mammals and the time course, following irradiation, of changes in white cells of the blood. Because blood data are available in man this relation permits a prediction of recovery half-time in man to be about one month. Although there is no known biological basis for Davidson's correlation it may be a sound one and also it may give a lead for search of similar empirical relationships. In any case the half-recovery time of 28 days chosen by Davidson appears to be a fairly conservative choice for man, even in the light of the problems raised above.

The objective for which the recovery half-time is used for human exposure calculations is that of avoiding a level of acute injury which will be dangerous or lethal. If recovery went

to completion this factor alone would be wholly determining and it would be reasonable, when necessary, to permit exposures to levels as high as possible without incapacitation.

The partial irreversibility of radiation injury precludes adoption of this simple point of view and also raises the question whether it is more practical to adopt a total dose as a permissible level independently of the time, for at least a month or two, over which it is sustained.

It is reasonable in comparison to other species and is indicated by the Rongelap incident [8] that 200 roentgens of whole body gamma radiation is sublethal for young adult man and probably for most of the very young and for the moderately old. In the young adult man this dose is not seriously incapacitating even when received promptly. It appears worthwhile then to consider the probable effects of 200 roentgens as a permissible dose in single episodes lasting for durations of minutes up to a month or more.

Observations on rodents [1] indicate that life is shortened about 7 percent per LD_{50} or about 1 percent per 100 roentgens for accumulated doses whose daily components do not exceed 120 roentgens. The effect with doses administered in less than a few hours is about 3 percent per 100 roentgens in the 200 to 500 roentgen range and is greater with larger doses.

This difference is not attributable to dose rate *per se* but to total dose within a given time. For example, using the same dose rate, Hursh *et al* [9] showed that 600 r shortened the life of the rat some 20 percent when administered in one day but gave a much smaller effect when administered in 10 daily doses of 60 r. These relationships require much additional study but in the rodents, at least, it appears safe to assume that doses less than 100 r per day give the smaller effect on life span and that doses of 200 r per day or more give the larger effect.

The only evidence that man may suffer fractional shortening of life span similar to that of rodents is that presented by Warren [10] whose data show an average loss of 5.2 years in longevity of American radiologists in comparison to unexposed physicians. The average ages of death are 60.5 and 65.7 years, respectively. If these radiologists dying in the period 1930 to 1954 sustained on the average the equivalent of about 800 roentgens of whole body radiation in divided doses their loss of life span would be similar to that in the rodent. Because this dose is in the range to be expected it is unlikely that man and rodent can differ by more than a small factor such as 2 or 3.

The effect on life-span of large prompt doses in man is not known but presumably it will be greater than that of distributed doses as in the rodents.

Assuming man and rodent to be alike 200 roentgens will shorten life about 2 percent when delivered at rates not exceeding about 100 r per day and shorten it as much as 6 or 7 percent when delivered promptly.

Existing data indicate that the after effects of successive exposures are additive. Therefore, two exposures of 200 r widely separated would shorten life twice as much as one. However, 400 r in a single prompt dose, if this is very near LD_{50} for man, would be expected to shorten life as much as 30 or 40 percent because life-shortening in rodents increases rapidly with the magnitude of the single prompt dose as the dose approaches the lethal range.

CONCLUSIONS

It appears that a limit of 200 roentgens for emergency exposures for any period up to 30 days will not entail acute lethality or significant incapacity. Consequent life-shortening would be as much as 6 percent, about 4 years, if man is like the rodent and if the dose is received over a short period. If the dose is less than a given amount, possibly about 100 r on any one day, life shortening will be about 2 percent. However, there are no definitive data for any species on how small the daily level must be to cause the lesser effect.

The suggested use of recovery with half-time of 28 days by Davidson to determine "effective dose" appears to be a conservative practice. It is not clear at present, however, what effec-

tive dose should be permitted in man because the concepts employed are based on lethality. It is not clear, for example, whether an effective dose of 200 r remaining from a greater total dose would at all times lead to the same degree of incapacity, even though it would presumably entail the same danger of lethality, as 200 r received promptly. Evidence on this point might be obtainable on the dog or some other species in which post-radiation blood changes may persist for months. At present it would probably be safer to limit the effective dose for prolonged exposures to a level somewhat less than 200 r or such other level as is permitted for short exposure.

For adequate control of prolonged or multiple exposures at substantial levels it is necessary to employ considerations of recovery rather than of total dose and the parameters and methods developed by Davidson appear to be the best available at this time.

The lethal dose for partial body exposure is higher than for whole body [11] and recovery is faster according to present indications [2]. Consequently safe estimates for whole body exposure will be even more conservative for partial body exposure.

Depending on facilities for radiation measurement and other factors it may be advisable to set permissible emergency limits for both total dose and effective dose and to use the one most feasible at the time.

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DISCUSSION

H. A. Blair

Capt. O'Donoghue (Bureau of Medicine and Surgery). I have often looked at figures like the last slide and instance of leukemia in radiologists and physicians, and thought it was very interesting, but we did not do anything about the dose sustained by the people. I have been very curious how Dr. Blair arrived at his thousand roentgen figure.

Dr. Berlin. I think that is an interesting question, Dr. Blair. Would you like to answer that?

Dr. Blair. I don't have a good answer. I have talked with a number of radiologists who were fairly well agreed that it didn't likely differ from 1,000 by a factor of more than two or so. If the daily dose rate got very high over long periods, there might have been more clinical manifestation of injury. Radiologists have not complained much about anything except burns of one kind or another. There have been instances of anemia, but they are not very common.

Dr. Cronkite. The rapidity of recovery of the peripheral blood of many species has been studied and varies considerably. It is also known to a great extent in man from the Marshallese studies.

I would like to ask, Dr. Blair, has the half time for recovery of the various species been correlated with the rapidity of the recovery of the blood picture in the various species?

Dr. Blair. I suppose the two extremes we know now are either guinea pigs or burro versus mouse. They have the shortest and longest recovery half times, I think, of the animals we know. It would be interesting to see if there was a relation between recovery rate and clinical manifestations in these species.

Dr. Robertson (Brookhaven). I would just like to make a point that has been mentioned by Dr. Berlin in a recent publication, which I think deserves more emphasis. That is, that Dr. Blair and Dr. Sacher, too, in considering the shortening of life span use the average for a group, whereas if mortality rates are plotted on a Gompert's type function, the displacement of the lines for an irradiated group from the normal group is a little greater. I take this as meaning that using the average does not take into account the automatic increase in death rate that is occurring with age, and therefore the average is not truly applicable to the individual, that is, the effect on the individual is a little greater than is deduced from the average.

Perhaps Dr. Blair's theory is flexible enough to make a bit of correction for this, and I wonder if he has thought about it in these terms.

Dr. Blair. I don't think this has anything to do with theory. Animals being currently irradiated are storing up irreversible injury, but on top of that they have acute injury from the doses gotten recently. The kind of data I was talking about here are the after effects of exposure. The radiation is stopped long before death so any acute injury that may have occurred has been healed. You have to be careful about this, because there are not very many data in the literature for which chronic radiation has stopped short of death. At a high level, such as 5 or 10 roentgens per day, the animal may die half from acute injury and half from residual injury. Allowance must be made for this in calculating shortening of life span per roentgen as an after-effect of radiation.

Dr. Sacher (Argonne). In regard to Dr. Robertson's question, I believe that you, Dr. Berlin, could probably discuss this to good advantage, because you have already, I believe, looked at relations between the average survival time estimates and the Gomperts thing.

Dr. Berlin. The Chair will not enter into a discussion at this time.

Dr. Sacher. I am afraid the burden falls on me. I believe that the Gompert's analysis is the most unbiased analysis that we can bring to lethality data, because it considers the lethality as a process that is going on continuously in an irradiated population. However, in looking at data today in terms of mean survival times, I did this out of sheer necessity, because in the daily dose studies we are usually dealing with very small populations of animals. I have not actually given serious consideration to correcting for bias in the mean survival time estimates, because I have not used them for the estimation of parameters. However, I showed two curves; one the estimate of cumulative lethality function and the derivative, two sets of curves. The first was based on mean survival times and the second based on an analysis using the Gompert's function. I think if you recall these you will recall that they were of the same form and my problem essentially is to find a scaling factor for them. Dr. Blair has mentioned the point that causes serious concern in the application of these theories to lethality under conditions when the radiation is being received up to the time of death, and that is the accumulation of injury due to the latest increments of dose received. I should say that when you use the deduced empirical function approach that I have used, you note that the injury curve takes on the order of 10 or 20 days to build to a maximum.

Therefore, when this is applied to the data, the dose as received in the last 10 or 20 days before death is given its proper weight in the contribution to the lethality. This leaves unresolved the question of whether the acute injury and the irrecoverable chronic injury combine and simply add to the death. I am quite convinced that they do not do so precisely. However, to think so and to know what to do about it are two entirely different things.

DISCUSSION ON TOPIC III

Biological Repair Factor

Dr. BERLIN. To start off the general discussion, I would like to ask Dr. Jones if he would initiate this discussion for us. We have heard from Dr. Blair and Dr. Sacher, and I think we should hear what Dr. Jones has to say on this field. I think we are fortunate in having all three in the auditorium at the same time. Perhaps we can arrive at some synthesis of mutual thought with them present.

Dr. JONES (University of California, Berkeley). I think it is a remarkable thing that all of us who have talked either here or recently elsewhere who are expressing opinions on radiation effects and particularly radiation effect upon the life span have an essentially coherent viewpoint about the thing, and are in essential agreement with regard to all major factors that I understand. Where we differ are differences in fine points of interpretation which are very important to our current work, but it is perhaps as useful to us to survey at this time some of the overall aspects of the radiation effect problem from the standpoint of what things fit together and what things perhaps do not.

Let me try to do this in about a 2-minute thumbnail sketch. In the first place, historically in radiation effects, I think everyone was first impressed by the gross aspects of radiation injuries. Things that had to do with burn, ulcer, tissue necrosis and the like. These things have enormous threshold effects. There are doses of radiation below which you do not see these effects at all. Between the range of out and out total killing of cells from which there may be no recovery because the cells don't exist any more to exhibit recovery and the threshold effect, you get regions where there are great reparative processes. So as John Storer expressed it, if you wait long enough below the level where you get frank burn, the reparative processes will give you a tissue that looks like a tissue that was not irradiated.

If you look at the problem from the standpoint of the genetic effect or the long-term effects of radiation, you have apparently another coherent viewpoint which seems to be at the opposite end of the scale. I wonder if these two viewpoints can't be brought together by the consideration of a radiation effect on a cellular basis.

In the first place, I think our concepts of threshold effects and reversible effects of radiation are largely the effects of radiation upon complex organisms such as mammals, where many cells are involved, and you have the potentiality of replacement of injured cells by cells which are not so much injured. You can divide and very rapidly, and take the place of injured tissue. As we get to the cellular level, I think the classical example is that cells do show a recovery effect such as Dr. Henshaw's early papers on the subject. Even at the same time and subsequent time since effects of radiation at the cellular level turn to be more quantum effect of radiation so we have the hit theory, and the like.

Below the cellular level at the chemical level and structural level of tissues, one finds overwhelming evidence for quantum interaction between radiation and matter and radiation effects that are largely irreversible in nature.

Now, let us look for a moment at the radiation effect in mammalian tissue. If we take a fairly uniform set of tissues such as the marrow, lymphatic tissue, and so on, there exists from the laboratories of quite a number of different observers, quantitative effects of radiation upon these tissues, either in terms of estimating

the total cellular mass that is left with respect to time after dose, perhaps the concentration of these cellular elements in the blood, or perhaps direct measures of mytosis or turnover of these cells in a measuring system.

If you take all these together with respect to dose you have a range of dose that extends from about 2,000 r at the upper end, down to about 15 r at the lower end, where you can get significant results. You find that over this whole range, even though you are dealing with different species, for these three tissues, the effects between the mouse, rat, and rabbit and man are that per roentgen on a log scale of surviving tissue as a function of dose, you have a linear effect of about 0.3 percent per roentgen, if you put it on a per roentgen basis. This, as I say, is over an enormous range of radiation exposure.

Now, this means, then, if you transform a little further that approximately in terms of the hit theory you have about 2 to 3 cells injured per 1,000 cells per 1 r of radiation exposure. If you test this out a little bit further in terms of what we know about the genetic effects of radiation, the genetic effects of radiation in terms of mammalian system, gives you at the level of 50 roentgens a mutation induced in about 1 cell out of 10 germinal cells. Then you multiply these two together, and you find per roentgen this means about 1 mutation in 500 cells or per 1,000 cells this is an induction of 1 new mutation of 2 cells out of every 1,000 cells exposed at the level of 1 roentgen. So that you see in terms of a system that we know that leads to immediate radiation damage in terms of the killing of cells associated with radiation effect, that both the genetic effect and the killing effect of cells per roentgen are the same order of magnitude, and thus we can very easily see a unifying bridge between these two systems of information that we can study.

In one case the manifestations per surviving cell are rather subtle in character, and in another effect with relatively large number of cells killed as you would have about the 50 percent lethal dose of radiation exposure which extends from about 200 to about 500 roentgens, depending upon the species, a very large number of cells killed, and of course quite a great physiologic generation of symptoms involved in such effect.

In terms of the recovery potentiality of these particular tissues, the lymphatic cells, the marrow, you have a great capacity of these cells to regenerate and replace the damaged ones that are killed. As a matter of fact, the daily replacement of such tissues anyway is of the order of 10 percent replacement per day. So even at the levels of one r, 10 r or 100 r, the radiation induced damage is not an enormous burden compared with the ordinary replacements of such cells in such tissues. So if this were the level that we could view radiation effect, I think we could be quite comfortably assured by the fact that the tissue potentiality of replacement is one that would lead us into a threshold effect of radiation and a very comfortable one, because we ought to be able to replace these cells. The trouble in the problem as far as radiation effect at subtle levels is concerned, that the cells that do survive very likely will carry with them the same quantitative transformation of the nucleic protein structure as the germinal cells in terms of mutation.

This would then be per roentgen at the generation of 1 or 2 new mutations per 1,000 cells. So that the surviving cells that fill and replace the cells that are damaged supposedly survive with this kind of a transformation of their inherent vitality. I think that this is where the genetic effects of radiation have a great deal in common. As far as we know, in critically testing these systems, we can be uncertain as to whether the life subtracting effect of radiation is entirely linear in terms of whether a divided dose or a single dose give the same effect.

Dr. Blair has just shown you some results on this. There is an entirely allowable viewpoint that a single dose may have 2 or 3 or 4 times the effect of a smaller rate of dose. But the statistics that all of us have to work with are so limited in their character that it would still permit a more unifying viewpoint that it does not make any difference for the life subtraction

effect as to whether the effect is given all in one dose or is fractionated.

This, of course, is the viewpoint that one largely takes for the total mass formation of the total genetic effects from radiation where the total transformation of the genetic information is per roentgen and does not depend upon dosage rate.

How we finally interpret the life subtracting effects of radiation, I do not know. It would be very, very tempting at this time to place the whole aging phenomenon in terms of acquisition of transformations of the cellular information on a mutational basis so that we could explain it on the basis of somatic cell mutation, accumulating with age. It is such a tempting system, indeed, because almost all the data that we have to work with fit. However, there is still another thing that we can work with from the standpoint of change with age on irreversible effect, and that is the absolute numbers of cells that may be involved. The amounts may be a qualitative difference in the kinds of cells that are left on the average after radiation exposure, and are left on the average after the aging effect proceeds.

There may be a change in the absolute number of cells that survive within a given individual either as a function of age or radiation exposure. The best information we have along this line is the information that Nathan Schott of Baltimore has collected for man, which strongly suggests that for such tissues as the kidney, and perhaps the body as a whole, that there is a decrease in active cell numbers amounting to about a 6 percent decline per decade for human tissues. This follows quite linearly over the whole of the measurable life span. So a combination of this perhaps with the change in the vigor of cells would certainly

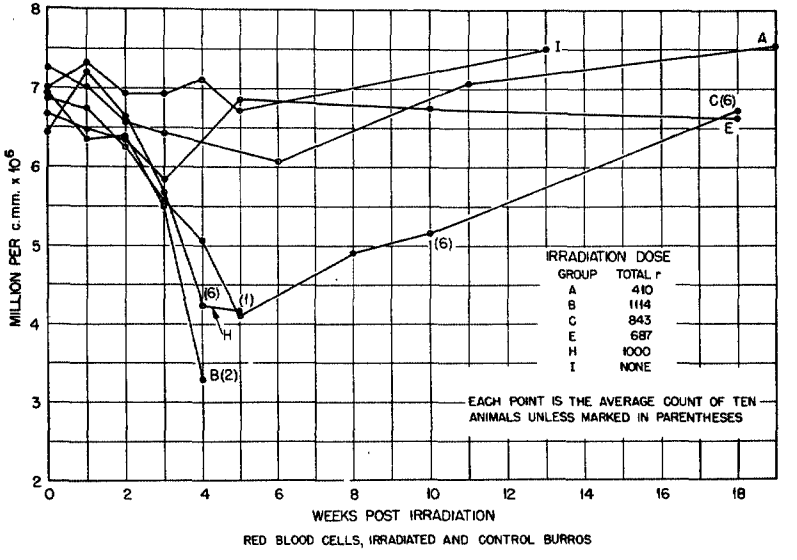

FIGURE 1.—*Red blood cells, irradiated and control burros.*

account for what we know about the life span and permit us to have a unifying attitude.

Dr. BERLIN. Colonel Trum is up here from Oak Ridge, and informs me he has some material which is pertinent at this time. I will now call on him.

Col. TRUM (Division of Biology and Medicine, AEC). Before I can make up my mind that there is a single common denominator to all of this, I must at least note that individual animals and individual tissues of animals, as well as specific species differences suggest a series of unrelated damages. Everyone who has spoken on this has put their finger on this at one time or another in some offhand statement.

Because I happen to have available, and I know from conversation that at least Bond, Sacher, and Storer are interested, I would like to present a few slides.

The doses shown on the slide are "free in air doses." They are so stated because at the time the experiment started we believed this to better represent the conditions found in a true "fallout field." We were unaware that there may be a flat depth dose existing during the critical high intensity period, as demonstrated by Vic Bond yesterday. We are still

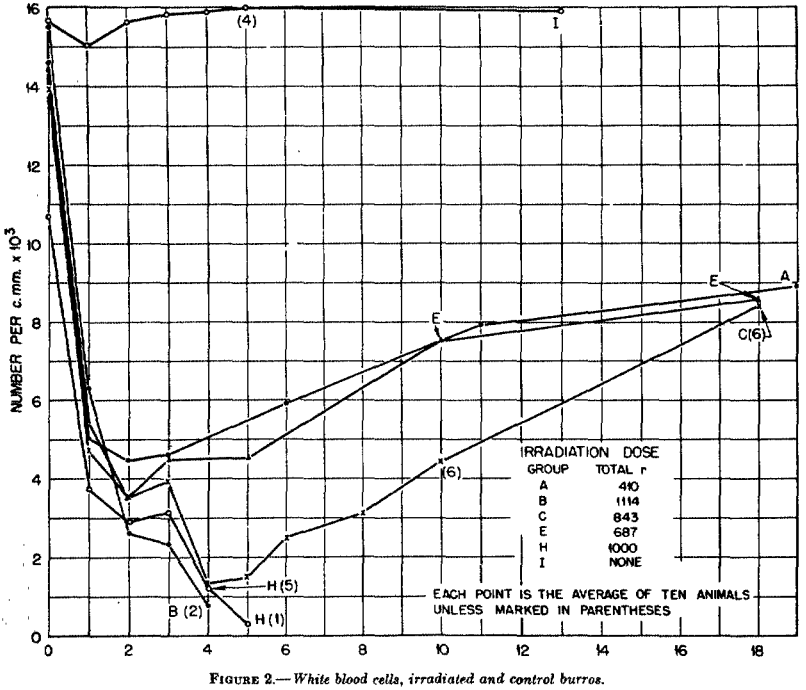

FIGURE 2.—*White blood cells, irradiated and control burros.*

looking for more information of this type. However, if you use these dose data it must be kept in mind that they are "free in air doses."

On Figure 1 you will note that the decrease in number of erythrocytes has reached normal levels in survivors of LD–50 studies at the end of several weeks.

However, as may be seen on Figure 2, in the same group of animals the lymphocytes had returned only to 50 percent normal at the same time, and as we can see in the following slide, the lymphocyte count did not approach normal for 2 years post irradiation.

These happen to be the results of work on 20 burros and yet this is true of all survivors. We know of no similar data on groups of animals with such a long life expectancy.

Now, note that two animals, survivors of LD–50/30 studies, and apparently on the road to recovery, suffered reverses. Although one had received 300 r and the other 530 r, both were in radiation groups of 10 in which no acute radiation deaths occurred—in other words, nonlethal doses. These animals died of radiation sickness 2½ and 3 years after exposure (fig. 3). At this time it was predicated from the post irradiation history that another animal that received 350 r at increments of 25 r/wk would probably die within the next year. Col. Rust informs me that this animal died about 4 years postirradiation.

The results of an experiment in which swine were given 600 r (air dose) of gamma radiation is illustrated in Figure 4. They were allowed

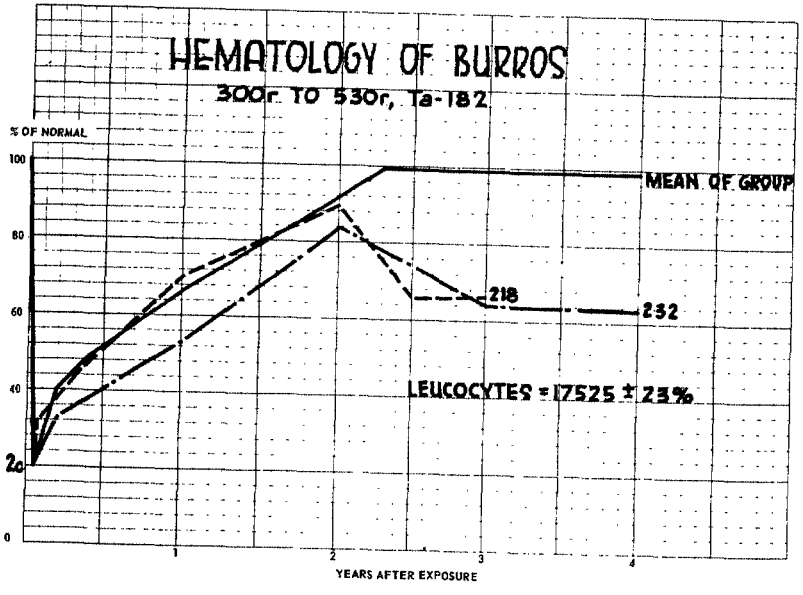

FIGURE 3.—*Hematology of burros—Years after exposure.*

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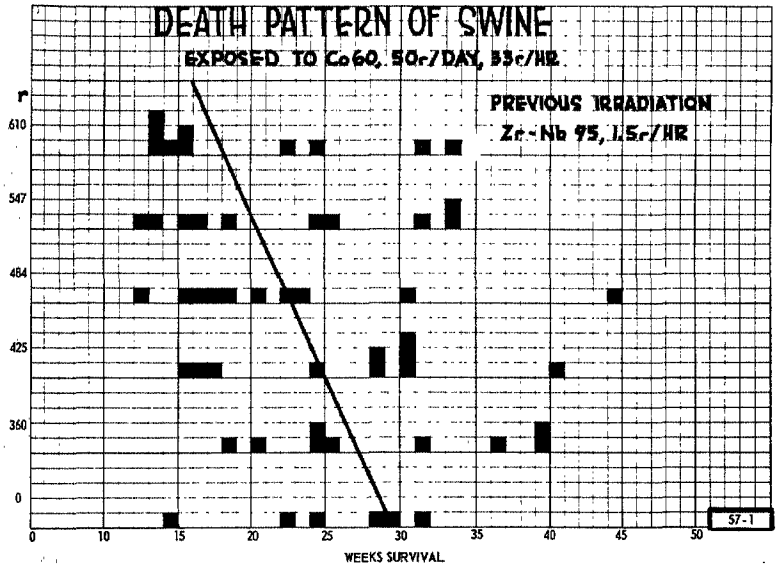

FIGURE 4.—*Death pattern of swine following irradiation.*

100 days recovery and reexposed at 50 r a day until death. There are several interesting things here: The previous dose has an effect at least on early deaths; the previous dose affected the spacing of deaths, bunching those with the higher previous dose; spacing those with the least previous dose; one animal was able to accumulate nearly 18,000 r 50 r/day before death.

There is no question of the swine's radioresistance, for both the Navy Group and UT-AEC group have found that the swine responds to acute radiation exposures similar to all other comparable animals.

In summary, can all of these variables be lumped and statistically treated as a single factor called "life shortening"?

Topic IV

External Beta Radiation

MATHEMATICAL AIDS IN THE UNDERSTANDING OF THE BIOLOGICAL HAZARDS OF RESIDUAL RADIATION

By Lt. Col. James T. Brennan, MC

Walter Reed Army Medical Center

In attempting to cope with radiation hazard problems, many a biologist has, like the writer, found that a meager working knowledge of mathematics places a frustrating upper limit on one's insight into many important situations. The mathematical treatment of the idealized contaminated plane surface is an example of this difficulty. A reference which is commonly cited in this connection is "Effects of Atomic Weapons," page 432 ff. The treatment given therein is by and for mathematicians, and as such is beyond most biologists and nearly all physicians. In 1951 C. S. Maupin [1] developed an expanded version of the analysis which appears useful in that it might significantly increase the number of biologists who can follow the derivation. This expanded version has not heretofore been published and is shown below (see fig. 1).

Consider a point P at a height h above a uniformly contaminated circular disk of radius a. Let the concentration of radioactivity be such that there are k photons, each of m (Mev) energy emitted (equally in all directions) per cm^2 of surface. Then the number of Mev emitted from the infinitesimal area $rdrd\theta$ is $kmrdrd\theta$. The number of Mev reaching point P per unit time from this small area will be

$$\frac{kmre^{-\mu\sqrt{r^2+h^2}}drd\theta}{4\pi(r^2+h^2)}$$

where μ is the total narrow beam absorption coefficient in air for photons of energy m. From this point on, no attempt is made to follow the fate of scattered photons. Ultimately this causes the estimate of dose at point P to be

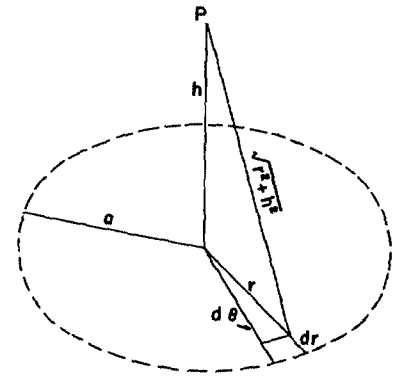

Figure 1.

low (30 percent low when h=6 meters, 10 percent low when h=1 meter). But to return to the analysis, the energy flow reaching P from the entire disk will be

$$I=\frac{km}{4\pi}\int_0^{2\pi}\int_0^a \frac{re^{-\mu\sqrt{r^2+h^2}}}{r^2+h^2}drd\theta$$

Integrating with respect to θ,

$$I=\frac{km}{2}\int_0^a \frac{re^{-\mu\sqrt{r^2+h^2}}}{r^2+h^2}dr$$

Conversion to dose in roentgens at point P may be made at any time by means of simple assumptions such as that one roentgen is delivered by a flux of 10^9 photons/cm^2.

The problem remaining then is to evaluate the geometric factor:

$$\int_0^a \frac{re^{-\mu\sqrt{r^2+h^2}}}{r^2+h^2}\,dr$$

Let $\mu\sqrt{r^2+h^2}=Z$. Then $\mu^2r^2+\mu^2h^2=Z^2$ and $2\mu^2rdr=2ZdZ$, or $rdr=\frac{ZdZ}{\mu^2}$.

This change in variable requires a change in limits as follows:

When $r=0$, $z=\mu h$
When $r=a$, $z=\mu\sqrt{a^2+h^2}$
When $r=\infty$, $z=\infty$.

$$\int_0^a \frac{re^{-\mu\sqrt{r^2+h^2}}}{r^2+h^2}\,dr=\int_{\mu h}^{\mu\sqrt{a^2+h^2}} \frac{ze^{-z}dz}{\frac{\mu^2}{z^2/\mu^2}}$$

$$=\int_{\mu_h}^{\mu\sqrt{a^2+h^2}} \frac{e^{-z}}{z}\,dz.$$

From the theory of limits

$$\int_{z_1}^{z_2} f(z)dz=\int_{z_1}^{\infty} f(z)dz-\int_{z_2}^{\infty} f(z)dz.$$

The two integrals on the right are of the form

$$\int_{z_1}^{\infty} \frac{e^{-z}}{z}\,dz=-Ei(-z_1)$$

which has been evaluated by Jahnke and Emde. Therefore:

$$\int_{\mu h}^{\mu\sqrt{a^2+h^2}} \frac{e^{-z}}{z}\,dz=\int_{\mu h}^{\infty} \frac{e^{-z}}{z}\,dz-\int_{\mu\sqrt{a^2+h^2}}^{\infty} \frac{e^{-z}}{z}\,dz$$

$$=Ei(-\mu\sqrt{a^2+h^2})-Ei(-\mu h).$$

The physical meaning of the limits is that the area of the disk in question is the difference in areas between $r=0$ to ∞, and $r=a$ to ∞.

A specific application of this analysis is given in Figure 2.

A useful conclusion to be drawn from Figure 2 is that 50 percent of the total dose at P comes from an area of radius 8 meters. This is rather less area than one might guess considering that the mean free path of the photons in air is $\sim$ 100 meters.

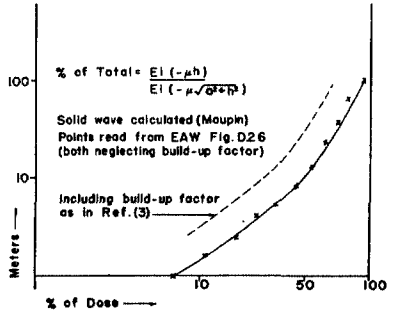

FIGURE 2.—*Percent of total dose as a function of r (h=1 meter).*

The figure of 8 meters is more plausible if one considers the case of two narrow rings of width Δr as in Figure 3.

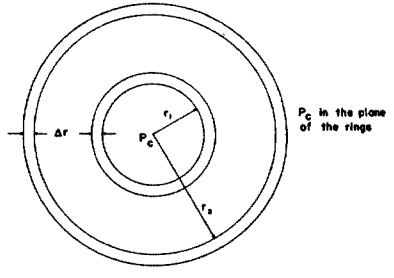

FIGURE 3.—P_c *in the plane of the rings.*

The ratio of the area of the two rings is:

$$\frac{A_1}{A_2}=\frac{(r_1+\Delta r)^2-r_1^2}{(r_2+\Delta r)^2-r_2^2}=\frac{2r_1\Delta r+\Delta r^2}{2r_2\Delta r+\Delta r^2}$$

and $\lim_{\Delta r\to 0}=\frac{r_1}{r_2}$

Thus the area of a thin ring is proportional to its radius, but the contribution to dose at P_c is

$$\propto \frac{A_1r_2^2}{A_2r_1^2}\propto \frac{r_1r_2^2}{r_2r_1^2}=\frac{r_2}{r_1}.$$

Thus, dose-wise, the relative importance of a ring is inversely proportional to its radius. The effect of air absorption is to further depress the importance of distant rings. This latter depression is only partially compensated for by scattered rays going through P_c. This may help to make the figure of 8 meters seem more reasonable.

The similar case of two thin spherical shell sources is of interest. If the shell volumes are V_1 and V_2,

$$\frac{V_1}{V_2}=\frac{4/3\pi[(r_1+\Delta r)^3-r_1^3]}{4/3\pi[(r_2+\Delta r)^3-r_2^3]}$$

$$=\frac{3r_1^2+3r_1\Delta r+\Delta r^2}{3r_2^2+3r_2\Delta r+\Delta r^2} \text{ and } \lim_{\Delta r\to 0}=\frac{r_1^2}{r_2^2}$$

Contribution to dose at the center of the sphere is $\propto \frac{V_1r_2^2}{V_2r_1^2}=1$.

Thus, spherical shells of equal thickness make equal contributions to dose at the center of a sphere, regardless of how large r may be. This conclusion is geometrical only, of course, and neglects scattering and absorption. Hence in the case of internally deposited gamma emitters, scatter is much more important than it is with the plane source.

In the case of a one-dimensional, or line source, the relative contribution by any increment of line is inversely proportional to the square of its distance from the point of measurement.

To those schooled in the exact sciences this sort of explanation may amount to belaboring the obvious. It is hoped that biologists who find such exposition illuminating will be forgiven.

In April 1949 Condit, Dyson, and Lamb [2] made the first calculation of the ratio of beta dose to gamma dose near a plane contaminated with fission products. The approach was very simple and amounts to saying that if two betas are emitted per gamma photon, and if the energy loss per unit path length for the beta particle is about 75 times that for the gamma, then the energy absorbed per unit volume ($\propto$ dose) will be about $2\times75=150$.

Slightly modified the derivation was as follows:

$E_x=E_0e^{-\tau x}$ for both betas and gammas

where E_x=Energy flux at a distance x from the source
E_0=Energy flux at the source
τ=Energy absorption coefficient
x=distance from source

Dose is closely related to the space rate of energy loss:

$$\frac{dE_x}{dx}=-E_x\tau$$

For betas, τ is replaced by the energy absorption coefficient μ_a which is obtained from empirical formulas

$$\frac{\mu_a}{d}=\frac{22}{(E_{max})^{1.33}}$$

where d=density of absorber in gms/cm^3.

For gammas, τ is replaced by $\sigma_a=3.5\times10^{-5}$ cm^{-1}.

Then the desired ratio of doses is:

$$\frac{\Phi_\beta}{\Phi_\gamma}=\frac{E_\beta\mu_a}{E_\gamma\sigma_a}\approx 130 \qquad \text{near the ground.}$$

This result was not widely known until about the time of Operation Greenhouse in 1951. In general, it was known from the early radium and radon days that gamma dose near a beta-gamma emitter is apt to be relatively negligible by comparison with beta dose.

Using the same geometrical analysis outlined in reference 1 above, reference 2 continued on

showing that at about 6 feet above a contaminated field

$$\frac{\Phi_\beta}{\Phi_\gamma} \quad \text{dropped off to about } \frac{130}{10}=13.$$

This result was the cause of a great deal of uneasiness within the Radiological Safety group during Operation Greenhouse because the instruments were, as usual, measuring and recording gamma dose only. It was suggested that if reference 2 had included the effect of Compton scattering of gamma rays in air, perhaps the beta/gamma dose ratio would be less alarming. For this reason, in reference 4 an attempt to include buildup or multiple scattering factors was made during Operation Greenhouse. Simple analytical treatments are not possible if buildup factors are to be considered. The approach used in reference 3 was a laborious numerical integration using certain empirical measurements of a buildup factor (4) that had become available. An example of how gamma dose at point P was obtained is given in Table I.

The contaminated ground was divided into unequal ring increments as in column 1.

ϕ in column (2) is the net geometrical attenuation factor.

Column (4) is the good geometry, narrow beam absorption factor.

Column (5) was obtained from White [4]. In that work White used a Co^{60} source and a water absorber, but the results were used in [3] without modification.

TABLE I.—GAMMA DOSE h=6 meters

(1) r_1 to r_2 meters	(2) ϕ	(3) Slant distance d in cm.	(4) Air absorption factor	(5) Buildup factor	(6) Relative contribution of increment	(7) Relative contribution per meter of radius	(8) Dose in r
0–1	0.09	602	0.94	1.0	0.08	0.08	0.72
1–2	.25	608	.94	1.0	.24	.24	2.16
2–3	.37	618	.94	1.0	.35	.35	3.15
3–4	.45	632	.94	1.0	.42	.42	3.78
4–5	.50	650	.94	1.0	.47	.47	4.23
5–6	.52	671	.94	1.0	.49	.49	4.41
6–8	1.03	922	.91	1.0	.94	.47	8.46
8–10	.97	1081	.90	1.1	.96	.48	8.64
10–20	.37	1616	.86	1.1	3.50	.35	31.5
20–30	.24	2571	.79	1.2	2.28	.24	20.5
30–40	.18	3551	.71	1.3	1.66	.16	14.9
40–50	.14	4540	.65	1.4	1.27	.14	11.4
50–70	2.09	6059	.56	1.5	1.76	.09	15.8
70–90	1.57	8022	.47	1.7	1.25	.06	11.3
90–120	1.80	10479	.37	2.0	1.33	.04	12.0
120–150	1.42	13514	.28	2.3	.89	.03	8.0
150–180	1.14	16511	.21	2.6	.62	.02	5.6
180–210	.97	19509	.16	3.0	.47	.009	4.2
210–250	1.09	24007	.10	3.5	.38	.006	3.4
250–300	1.14	27500	.07	4.0	.32	.005	2.9
300–350	.97	32500	.05	4.8	.23	.003	2.1
350–400	.84	37500	.03	5.4	.14	.002	1.3
400–450	.68	42500	.02	5.7	.08	.001	.7
450–500	.66	47500	.01	6.4	.04		.4
Total							182.0

A similar table is necessary for each height desired.
Comparison of column (5) with column (8) indicates that scatter accounts for about 30 percent of the total dose at P.

Column (7) takes into account the fact that *the increments chosen are not of equal width.*

In column (7) note that for a height of 6 meters, maximum dose delivering efficiency occurs at about the 6-meter radius.

In column (8) about half of the dose comes from inside the circle $r=30$ meters. For $h=1$ meter 50 percent comes from inside 12 meters (see fig. 2). Thus the net result of [3] was to show that inclusion of multiple scattering makes a militarily significant change in the total gamma dose but does not radically change the conclusions of Condit, Dyson, and Lamb regarding the beta/gamma dose ratio.

Comparison of columns (4) and (5) shows how buildup only partially compensates for absorption.

Beta dose in reference 4 was calculated using the method of Parker [5]. This is again a numerical integration method using, this time, equal ring increments. Distances greater than 6 meters were not considered significant. The distance for each ring is taken as d_m, the distance to the midpoint (see fig. 4).

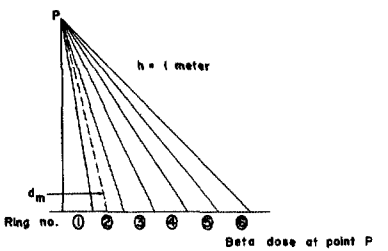

FIGURE 4.—*Beta dose at point P.*

Calculating beta dose in this manner, and gamma dose as in Table I, gave beta/gamma ratios which were not significantly different from those in reference 2.

Operation Greenhouse marked the end of what might be called the primitive era, since immediately afterward the AFSWP staff in Washington began to expend greater effort on the mathematics of fallout radiation.

For some years prior to 1952, the National Bureau of Standards group (Fano, Spencer, *et al.*) had been developing a mathematical theory concerning the penetration of X-rays through thick barriers. At the request of, and in cooperation with, the AFSWP mathematicians, the NBS theory of multiple scattering has been applied to the calculation of gamma fluxes in air at points above a plane, in a foxhole, and so forth. This work continues even now, and the writer has the impression that the theories used are fundamentally powerful enough to give satisfactory mathematical solutions for any foreseeable military medical problems due to fallout hazard.

In 1955 the multiple scattering theory was applied to beta particles [6] and another theoretical treatment of the same subject appeared [7].

These later, and professionally competent mathematical approaches yield results which agree with the physical measurements that have been made to date. So far as comparison is possible the results are not in disagreement with the conclusions reached in the crude attempts previously discussed.

British and Canadian documents have become available in recent years which show that their theoretical conclusions and field measurements are essentially the same as ours. There is a wide spectrum of opinion regarding the operational implications of these conclusions.

The mathematical methods evolved by the NBS group include the use of an electronic computer and, on the whole, appear to be beyond the ken of any biologist or physician now available to work on fallout hazard. In this situation, any progress on medical problems will require that:

A. The biologists concerned will have to accept on faith mathematical conclusions which they do not really understand.

B. The mathematician and the radiological physicist will have to be patient and endure diffuse and frustrating discussions of what really needs to be calculated and measured in order to develop an adequate medical policy.

With regard to (A) above, acceptance "on faith" has many precedents and is not, *per se*, undesirable. The difficulty is that a lack of understanding of dosimetry often has a curiously ennervating effect on the biologist doing radiation hazard work. He begins to feel that all he does is feed the mice and count the number dead at the end of 30 days. Someone else does the brainwork. Consequently, he drifts off into some other field of endeavor. Career-wise this is probably a sound instinct as far as the biologist is concerned; but if the proper solution of the fallout problem is, potentially, a condition of national survival (as some say), then the necessary minimum of capable biologists and physicians must be kept in the effort. One positive step that any biologist can take is to make a renewed attempt to understand the mathematics involved. Even the "crude" methods discussed earlier provide a degree of understanding that can be had in no easier way.

With regard to (B) above, it is offered as one opinion that, in order to arrive at a complete medical policy regarding a fallout hazard, it will ultimately be necessary not only to calculate and measure total beta and gamma fluxes, but also to:

(1) Calculate and measure the polar distribution (i. e., direction) of those fluxes in air.

(2) Calculate and measure beta and gamma depth dose in human sized animals in a fallout field.

If this seems to be asking a great deal, then it should be recalled that the problem is important enough to warrant the use of whatever scientific resources are necessary to solve it completely.

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DISCUSSION

James T. Brennan

Col. Brennan. I should like to stop here and give the people in the audience who have knowledge of other mathematical efforts that are relevant a chance to please stand up and mention them, and indicate what application and significance they have to the hazard problem.

I know of several. I know Naval Radiological Defense Laboratory has direction measurements on it. They are trying to get the theory to go with it. I know Mr. Joseph Lindwarm has mathematicians hitting that direction. I today have been informed that the National Bureau of Standards, who have by far the greatest resources in this matter have under consideration a general treatment of beta ray penetration. They will come out with a general theory. Whether or not this includes direction, I don't know. I wonder if I could ask the gentleman from the Bureau of Standards, Dr. Wyckoff, to say a word about that.

Dr. Wyckoff. I relayed the few notes I had from Dr. Spencer, but I am afraid I don't have anything to add other than the fact that they have coded up some of the beta penetration problems for the Standards Eastern Automatic Computer and apparently are able to put in the spectra of beta particles going into a shielding situation, and will be able to obtain both the dose distribution in the shielding and the flux coming out. I don't know if that includes the angular distribution that you are interested in.

Col. Brennan. The theory for gamma rays on the other hand I do know does permit calculations of polar distribution. It would perhaps be an enormous job and extended effort in applied mathematics to reduce this to tables that could be used for a simple situation by simple people for such things as a foxhole. This is a goal worth striving for, and I think one well worth financing. A good mathematical theory eliminates a lot of bad experiments, and makes it certain that money will never be wasted. Mathematics is about the cheapest type of research you can do, I believe, in return for dollar expended.

This is about all the ground I wanted to cover with respect to how mathematics has been applied and might be applied to the residual problem. Are there any further questions or comments? Particularly, does anyone know of mathematical efforts that are relevant?

Capt. Zellmer (School of Aviation Medicine). I believe there are efforts being directed to measure the angular distribution of gamma and neutrons, at least, in the forthcoming field test, using columnators with solid angles trying to obtain the angular distribution in the hemisphere.

Col. Brennan. Is this prompt radiation or residual?

Capt. Zellmer. Prompt. I imagine there will be some delayed and scattered radiation also, and some immediate fallout, because they won't be able to get to the columnators for at least two hours.

Col. Brennan. We might have a word from NRDL. I know they have a definite interest in this activity.

Dr. Teresi (NRDL). We wrote about two or three technical memoranda on this particular subject of the beta to gamma ratios, both the beta particles to gamma photons, and the beta radiation dose in terms of rep to the roentgen. I might say that we discussed there this mathematical treatment for the gamma and also pointed out the fact that you do have variation in the dose due to the variation in energy with time. I don't think Dr. Brennan pointed this out. However, I think it is obvious when you try to go from the equation to the determination of dose rate that you would need the actual gamma energy there to determine this. That does change with time.

We discussed this particular thing in these papers that we wrote up, and also the fact that your beta to gamma—that is beta particle to gamma photon—ratio will change with time. As a matter of fact, if you calculate the beta to gamma ratio for time about 2 to 3 years you will find that there will be about 8 beta particles to a gamma photon. This is approximate. Therefore, going back to the simplest relationship, 2 times 75, that would be 8 times 75. So that for very long times after detonation, you will get or should get tremendous ratios of beta rep to gamma roentgen.

I think that is about all I would like to discuss at the present time.

Col. Brennan. Thank you very much, Dr. Teresi.

The British have emphasized this. I omitted to say that these calculations do not apply except at times between the beta-gamma ratio is two particles per photon. There are two British articles. Have you seen them?

Dr. Teresi. Yes.

Col. Brennan. They emphasize the fact that after one year the beta goes up by a factor of about four. They had a report in which they had measurements which support this.

THE EFFECTS OF FALLOUT RADIATION ON THE SKIN

By Robert A. Conard, M. D.

Brookhaven National Laboratory

Fallout may be classified as the "worldwide" or the "close-in" type

Worldwide fallout results from the dissemination of minute particles of radioactive material from nuclear detonations which slowly settle out from the stratosphere and troposphere over the world. Due to the great dilution of this type of fallout and to the loss of activity with time evolved it does not impose a hazard to the skin but may result in a long-term hazard from internal deposition and possible genetic effects from low level irradiation.

Close-in fallout is most likely to result from large atomic detonations in which the fireball comes in contact with the ground, causing large amounts of material to be drawn up into the cloud where the radioactive products adhere to the ground particles. Due to the relatively large size of these particles they may then be deposited within several hundred miles of the detonation. With this type of fallout there is a real hazard not only to the skin, but also from whole body penetrating radiation and from internal absorption of radioactive materials. The nearer the site of detonation that fallout occurs the greater is the hazard. The nearer fallout takes place earlier and is therefore more active due to having undergone less radioactive decay and it is more concentrated since larger amounts (particularly larger particles) tend to fall out first.

The accidental exposure of some 240 Marshallese, 28 Americans and 23 Japanese fishermen during Operation Castle, March 1954, affords our most extensive experience with fallout effects on the human skin and in this talk frequent references will be made to data obtained on these people [1, 2]. Several other cases of human exposure to fission products or beta emitting material either accidentally or experimentally have been reported [3–8]. Skin lesions in cattle and horses have also occurred from fallout following experimental detonations at Alamogordo and in Nevada [9, 10]. Rather numerous experiments on the effects of beta radiation on the skin of animals have been reported and these data will be referred to, also [11–15].

First, some of the physical and biological factors related to skin damage from fallout will be discussed. The chemical and physical makeup of fallout will vary according to the type of terrain or soil over which the detonation occurs. All fallout is particulate in nature, but the size of the particles will depend to some extent on the physical and chemical characteristics of the soil. The fallout associated with the Castle detonation, March 1, 1954, was a white, powdery material largely composed of incinerated coral. Aside from the radioactive component the calcium oxide of the material was in itself irritating to the skin due to its caustic nature. Moreover it was probably partly dissolved in the perspiration on the skin thus increasing its irritating action. (Incidentally, this may have enhanced the radiation to the skin by bringing the radioactive materials in closer contact with the skin.) Fallout produced from other types of soil, other than predominantly coral, might vary considerably in chemical and physical makeup and irritation to the skin. Color and particle size would also vary. For instance siliceous type soils would probably form much less irritating fallout.

It goes without saying that for fallout to result in gross skin damage it would have to be

sufficiently concentrated. It seems likely that the occurrence of fallout would have to be visible to result in such damage. For example, in the Marshall Island experience, the extent and severity of the skin lesions were directly related to the amount of visible fallout and on Utirik, the least contaminated island of the inhabited group no fallout was visible and no beta lesions of the skin developed.

The particulate nature of the material produces spotty distribution on the body. The Marshallese claimed that the material adhered closely to the skin and was difficult to brush off. This was borne out by the difficulties of complete decontamination. Areas of the body where perspiration is greater such as the neck folds, axillae, antecubital fossae etc. caused the material to stick and lesions were more predominant in these areas. The hair tended to collect the material also, particularly in view of the cocoanut oil hair dressing used by these people, which made decontamination extremely difficult. Clothing, even a single layer of cotton material, afforded almost complete protection as evidenced by the fact that almost all of the skin lesions developed on exposed parts of the body. The loose clothing worn would not have accounted for more than about a 25 percent attenuation of the radiation so that the protection must have been due in part to the fact that the loosely fitted clothing tended to hold the radioactive material away from the skin. It is also possible that the material did not stick to the clothing as well as to the skin.

There are certain *biological factors* known to influence the sensitivity of the skin to radiation. In addition to species differences, it is known that the skin of certain parts of the body is more sensitive than that of others. In general the thinner-skinned flexor surfaces of the body are more sensitive than the thicker-skinned extensor surfaces [16]. This was found to be true in the Marshallese since lesions were more prevalent on the front and sides of the neck, axilla and antecubital fossae. Another factor is associated with pigmentation of the skin. Darker-skinned people, brunettes, are known to be less sensitive to radiation than blonds or people with ruddy complexions [17]. A factor which was pointed out earlier is that areas of the body where perspiration is more profuse cause the fallout to collect resulting in greater skin effects.

Sources of radiation to the skin.—Damage to the skin results largely from the beta component of the fallout in view of the fact that the beta-gamma ratio is quite high. All of the energy of the beta particles entering the skin is absorbed in the skin. Soft gamma rays accounts for some of the radiation dose to the skin, and the harder gamma rays contribute least since they are more penetrating. The skin dose results from two sources of beta radiation, the fallout material in direct contact with the skin and the material on the ground.

1. *Contact source.*—The spotty distribution and particulate nature of the fallout in contact with skin results in multiple point sources on the skin. By far the greatest part of the skin dose comes from this source. Radiation is largely from the skin surface. However, the possibility must be considered that a certain amount of percutaneous absorption may take place and some penetration into the dermal region via the hair shafts, sebaceous and sweat glands may occur. The Castle fallout contained about *10 percent water soluble fission* products, some of which might conceivably have been absorbed percutaneously. Whitten *et al.* [18] have shown that thorium-x applied to the skin results in some percutaneous absorption and entry into the hair shafts and glands. We intend to investigate this problem with fission products on the skin by means of autoradiography.

2. *Ground source.*—A certain amount of the skin dose may result from beta radiation from the fallout material on the ground. This contribution is likely to be far less than that from the contact source. The lower parts of the body will receive the greater part of this radiation since the beta particles are completely stopped in 2 meters of air.

Estimation of beta doses to the skin from fallout is an exceedingly complicated problem and I will leave the main discussion of the subject to other speakers. The degree of skin reaction and damage is more dependent on the depth dose than on the surface dose of beta radiation and the depth dose is dependent on the energies of the beta particles of the component isotopes. Thus soft radiation confined largely to the dead horny layer and upper epidermis would be relatively ineffective in producing a reaction in the skin; more energetic radiation, penetrating through the epidermis, could result in transepidermal necrosis; and deeper penetration into the dermis could result in more severe ulcerating lesions. Each radioisotope has its own characteristic spectrum of energies with a maximum energy, but since relatively few particles are of this energy, the average energy, which is roughly one-third of the maximum energy and the 50 percent attenuation in tissue are more meaningful in estimating skin effects.

Figure 1 shows roughly the 50 percent attenuation in skin of several isotopes. With the same surface dose the more energetic beta emmiting isotopes will naturally result in greater damage to the skin.

Table 1 is made up of data from animal studies from several investigators and shows the energy dependence of betas from various isotopes in producing recognizable skin reactions. Note that the surface doses for threshold reaction (erythema, epidermal atrophy) are fairly dependent on the energy of the beta particles of the various isotopes. Thus it takes 20,000–30,000 rep from S^{35} (average energy 0.1 Mev.) to produce a reaction while it only takes 1500–2000 rep of Sr^{90}–Y^{90} or Y^{91} (average energy 0.5–0.6 Mev.) to produce the same reaction. It is of interest that Moritz and Henriques found that the dose at 0.09 mm. depth of the pigskin (estimated to be the epidermal thickness) was constant within several hundred rep to produce transepidermal injury [15]. Wilhelmy has also noted that it takes roughly the same dose of electrons and soft X-rays at the level of the subpapillary layer to produce erythema [19]. On this basis

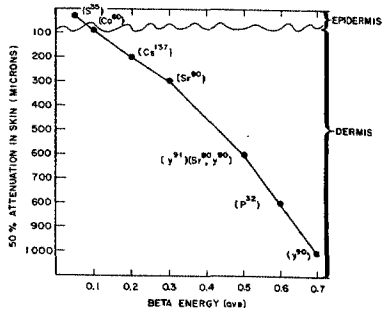

FIGURE 1.—*50 percent attenuation in skin (microns).*

TABLE 1.—SURFACE DOSE REQUIRED TO PRODUCE RECOGNIZABLE EPIDERMAL INJURY

Investigator	Animal	Isotope	Average energy (Mev.)	Surface dose (rep)
Henshaw, et al.	Rats	P^{32}	0.5	1,500–4,000
Snider and Raper	Mice	P^{32}	.5	2,500
Raper and Barnes	Rabbits	P^{32}	.5	5,000
Lushbaugh	Sheep	S^{90}	.3	2,500–5,000
Moritz and Henriques	Pigs	S^{35}	.05	20,000–30,000
Do	do	Co^{60}	.1	4,000–5,000
Do	do	Cs^{137}	.2	2,000–3,000
Do	do	Sr^{90}	.3	1,500–2,000
Do	do	Y^{91}	.5	1,500–2,000
Do	do	Y^{90}	.7	1,500–2,000

Parker has advocated the use of beta detecting instruments with chamber walls corresponding in milligrams per square centimeter to the thickness of the relatively inert epidermal layer [20]. Thus in expressing skin dosage it is probably more informative to use the depth dose at the depth of the epidermal layer of the skin.

The above table also indicates the species difference in skin sensitivity to beta radiation. Rabbits and sheep required larger doses than mice to produce the same effect with roughly the same energy beta. Porcine skin, which is reputedly more like human skin than other animals, apparently is more sensitive than the rabbit or sheep skin. Some of these differences, aside from species differences, may be due to variation in thickness of the skin of different species and differences in techniques used.

Table 2 shows beta dosage data from some human experiments and accidents found to produce various effects on the skin. These data must be interpreted with great caution due to differences in experimental techniques and dosimetry. The severity of the skin reactions is represented by degrees. A first degree reaction implies erythema and/or dry desquamation; a second degree, transepidermal necrosis with ulceration; and third degree, further breakdown of the skin with the development of chronic radiation dermatitis. It can be seen that there is a considerable variation in dose reported to produce the various reactions.

In the Marshallese the skin dose could not be estimated with any degree of accuracy due to the complicated smear of beta spectra varying with time and the uneven distribution of the material on the skin.

The beta component of the fallout was found to have two major peaks of energy, one at 100 kev which accounted for 50–80 percent of the activity and one at 600 kev which accounted for 20–50 percent of the activity [1]. Fifty percent attenuation of the 100 kev component occurs at about 80 microns, about the depth of the epidermis. Fifty percent attenuation of the 600 kev component occurs at about 800 microns, fairly deep in the dermis; deep enough to irradiate many of the hair follicles. The relatively soft nature of the radiation was borne out by the superficial nature of most of the lesions that developed.

A very rough biological estimate of the dose to the scalp of the Rongelap people might be made by using the index of epilation. It is known that with 200 kvp X-ray a dose of about 400 r is necessary to produce epilation, and doses above about 700 r produce permanent epilation. Since regrowth of hair took place in the epilated Marshallese the dose to the hair follicles must have been in the above range. This dose must have been largely from the 600 kev component. Therefore the surface dose from this component must have been 4 to 5 times higher or in the range of 1,600–3,500 rep. The surface dose from the more abundant 100 kev component must have been much higher, by

TABLE 2.—HUMAN EXPOSURE TO BETA RADIATION

Investigator	Radiation	Est. dose (rep)	Reaction
Wirth and Raper	P^{32}	635	1st degree (threshold).
Do	P^{32}	1, 180	2nd degree (threshold).
Low-Beer	P^{32}	*143	1st degree (threshold).
Do	P^{32}	7, 000–17, 000	2nd degree.
Robbins *et al*	Cathode rays (1,200 kv)	1, 000– 2, 000	3rd degree.
Knowlton *et al*	Fission products	3, 000– 4, 000	2nd degree.
Do	(1 Mev. ave. energy)	5, 000–10, 000	3rd degree.
Do	do	5, 000–10, 000	3rd degree.
Do	do	8, 000–16, 000	3rd degree.

*Estimated dose in 1st mm. layer.

a factor of 5, 10 or more, but with very little penetration.

Estimations of the dose of skin irradiation from ground source beta has been made by Sondhaus [1].

If no shielding occurred and exposure is considered continuous the dose at the level of the dorsum of the feet was calculated to be about 2,000 rep, at hip level 600 rep and at head level 300 rep. This source of radiation was apparently insufficient, alone, to produce any lesions, though it probably contributed significantly to the severity of the foot lesions observed. With larger amounts of fallout, radiation from the ground source could be sufficient in itself to produce skin lesions.

Acute effects of beta radiation on the skin.—In general beta radiation effects on the skin are similar to effects produced by more penetrating radiation such as gamma or X-radiation. However, the less penetrating beta radiation produces more superficial lesions with less damage to the dermis. Consequently they are usually less painful and heal more rapidly.

The time sequence of development of beta lesions from fallout varies considerably with the dose to the skin. A primary erythema may or may not be observed beginning a few hours after exposure. This was not seen in the Marshallese, perhaps due to the dark color of their skin. During the first day or so itching, burning, or tingling of the affected skin may be experienced. As was pointed out these symptoms might in part be due to the chemical nature of the fallout. These early signs and symptoms are usually followed by an asymptomatic latent period before full-blown lesions develop. The length of the latent period may vary from a few days to several weeks which is usually related to the dose to the skin; the higher the dose the shorter the latent period. In the Marshallese the more heavily exposed group developed skin lesions about a week before less heavily exposed groups. Due to the particulate nature and uneven distribution of the fallout on the skin the developing lesions are likely to be spotty. A secondary wave of erythema may be seen along with gross changes in the skin. These changes may be in the form of simple tanning, pigmentation, and mild desquamation with low doses. This reaction might be classed as a *first degree reaction*. With higher doses vesiculation, complete epidermolysis and ulceration may occur. This severity of reaction might be classed as a *second degree reaction*. Spotty epilation may occur along with lesions of the scalp. Regrowth of hair is likely with a second degree lesion. Healing is usually accomplished within a week or two with repigmentation of the skin in milder lesions. Deeper lesions may heal with some scarring and lack of repigmentation.

Chronic radiation effects.—With larger doses of radiation chronic radiation dermatitis may develop. These lesions do not heal well and on healing may break down and ulcerate again. Badly scarred skin with telangiectatic vessels may result. These severe reactions might be classed as *third degree reactions*. Repeated repair and breakdown may occur due to instability and poor vascularity of the dermis. It is in skin of type that malignant change may later take place.

Malignant changes in the skin has been observed in animals as a late effect of beta irradiation of the skin and presumably could also occur in the human skin. Though malignancy usually develops at the site of chronic radiation dermatitis, as a result of repeated exposure to radiation it has been reported to occur in animals following a single exposure to beta radiation with little or no chronic change in the skin.

Treatment of acute beta lesions is mainly symptomatic. With mild lesions, daily cleansing, application of bland antipruitic ointments and lotions may be all that is necessary. For more severe ulcerating lesions, cleaning with daily dressings, splinting and use of antibiotic ointments or antibiotics parenterally in case of secondary infections may be indicated. The use of Aloe Vera plant applications is claimed by some to enhance healing of radiation burns [21]. Lesions of chronic radiation dermatitis may be quite painful and the only effective therapy in such cases is early skin grafting [22].

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Figures 2, 3, 4, and 5 illustrate typical lesions in the Marshallese people.

In conclusion I would like to summarize a few things we have learned about the effects of fallout on the skin, largely as a result of our Marshallese experience:

1. The best prophylactic measure, of course, is avoiding getting the fallout on the skin by taking shelter or covering as much of the body as possible with clothing. Prompt decontamination of the skin by thorough scrubbing with soap or detergent and water is of extreme importance. If the hair is seriously contaminated and difficulty is encountered in decontamination, shaving of the head is indicated.

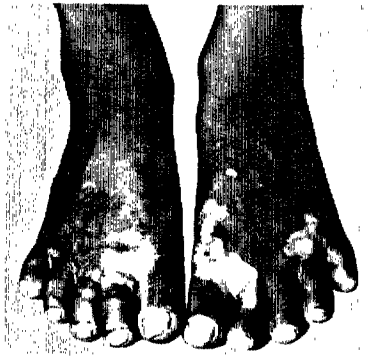

FIGURE 2.—*Beta radiation lesions of the feet at 4 weeks after exposure.*

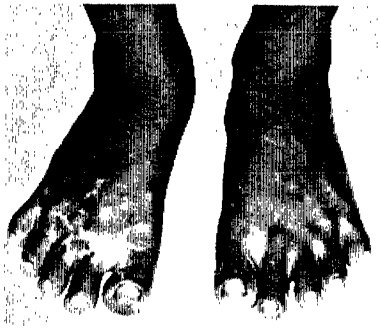

FIGURE 3.—*Same case in Figure 2 at 6 months after exposure.*

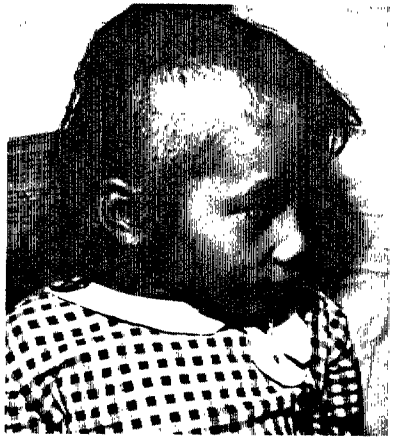

FIGURE 4.—*Epilation in 7-year old girl at 28 days. Case No. 72.*

In the Marshallese certain factors afforded protection against the development of lesions: (1) Shelter, (2) Bathing, swimming, wading, (3) Clothing. Certain factors also favored the development of lesions: (1) As pointed out areas where perspiration is more profuse, (2) Delay in decontamination, and (3) Difficulties in decontamination.

2. Moderately severe beta lesions of the skin and epilation may result from fallout situations in which the whole body penetrating dose of radiation is sublethal. With such doses the skin lesions do not appear to complicate the radiation syndrome.

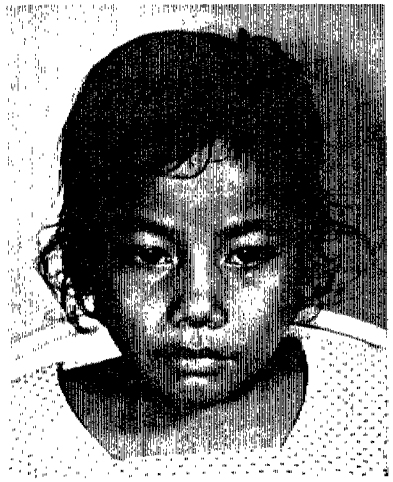

FIGURE 5.—*Same case as in Figure 4 six months after exposure showing complete regrowth of normal hair.*

3. However, in situations where skin lesions are associated with larger whole body doses of radiation i. e. in the lethal range or above, with greater hematopoetic depression, the lesions would become more easily infected, possibly affording portals of entry, leading to bacteremia or septicemia.

4. Severe skin irradiation with minimal whole body irradiation might result in situations where promp evacuation from the contaminated area occurred, but skin decontamination was delayed.

5. Early skin and eye symptoms might be mildly disabling during the first day or two after exposure to fallout and later symptoms associated with full blown lesions might be quite disabling. Late effects on the skin in the form of chronic radiation dermatitis and malignancy are possible complications.

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DISCUSSION ON TOPIC IV

External Beta Radiation

Dr. HENSHAW. A few years ago when at Oak Ridge, we were studying the effects of beta rays on rats and mice, and we saw lesions so very much like this which were followed by different kinds of neoplasias of the skin. The interesting thing was that we saw these abnormalities and deformities of the skin of a variety of types much as described here, and then the lesions after recovery and repair of the skin had taken place there were points where tumors began to form. These tumors were different in type. They represented every conceivable level of maturation of skin tissue. That combined with the fact that there were these other abnormalities of the skin directed our attention to the matter of the guiding forces in the skin that tend to make it behave in one way or another.

We had called to our attention rather forcefully this morning the possibility of the effects on the nucleus as being an explanation, first, of tissue degeneration when there are extreme mutations in the nucleus, and then in lesser mutations the kinds of persistent effects. We could see in these lesions this afternoon evidence of a behavior of tissues which will certainly differ from that of the usual traumatic kind of lesions, such as a cut with a knife or a burn. It was as though the guiding forces of the cells worked differently and here were some that were trying to do one type of thing, and others that were attempting to do other kinds of things.

If we should think of somatic mutations as being a partial explanation of what is happening and then think what would be the situation if the radiation were distributed throughout the body, perhaps we begin to get some basis of an explanation of what the aging processes are, and in relation to this, perhaps within the same structure of explanation, some basis of an explanation of the changes that led toward malignancy. That mutations take place there is no question. But the dynamics of those changes—the dynamics of the tissue behavior changes—with the modification in physiologic gradients are things which may be very strong and important forces for us to take into account in attempting to explain these various kinds of processes that we are seeing.

Col. BRENNAN. Thank you very much, Dr. Henshaw. As I recall, you have examples of tumors from all different layers of the skin.

Dr. HENSHAW. Yes.

Col. BRENNAN. Every layer gave its own type of tumor.

Dr. HENSHAW. Yes. Not only that, but there were hair folicle tumors, gland tumors, and other kinds of tissue expressions. There were a few connective tissue tumors. When we used more penetrating radiations those were more frequent. Then the related observations that when you have bone seekers you get the bone tumors, so it is largely a matter of distribution of the radiation as to the kinds of malignancies that occur.

Col. BRENNAN. Thank you. Certainly malignancy is the big question to be watched here. Perhaps when Dr. Conrad makes his 27th semiannual visit to the Marshallese, we will have the answer. (Laughter.)

Mr. Joseph Lindwarm of the Chemical Corps has some remarks which are pertinent to the general subject of beta, particularly with regard to what the consequences would be to take steps to avoid this sort of thing. Would you please give us the benefits of your remarks.

Mr. LINDWARM (Army Chemical Center). My comments on the beta hazard are being

made not from the point of view of a biologist or physicist, but someone who has been exposed to Army operational requirements concepts, and thinking over the past few years in the field of radiological defense in general.

My feeling is that many of the practical problems of radiological defense of which beta is one can be resolved most successfully by a joint attack on the problem by research people and the operational people with the research people providing the basic information. Where the operational people provide their capabilities and limitations, which serve in many cases as a framework in which part of the research and development effort at least should be directed to provide solutions to these very simple problems. This is no more apparent than in the case of beta hazard. I think if we assume for the moment that as a result of the studies that Col. Brennan recommended as to the beta hazard, let us assume that they proceed to the point where they indicate that in a fair number of tactical or practical situations in the field, there will be a beta hazard relative to the gamma hazard.

The very real operational problem then comes to the fore is, do you have to assess this hazard in the field, and if you do, how do you go about doing it? There are two schools of thought on this particular problem. One says that you have to have beta detection capability or measuring capability in the field, and the other school says the way to approach this thing is to do some research and development based on simplified geometry situations, and by means of gamma measurements plus factors based on field geometry, you can come up with a fairly decent estimate of what the beta hazard will be in these various situations.

The question as to which approach should be taken appears to be dictated at the present moment by operational limitations, rather than technical limitations. If you can assume for the moment that you do have radiological equipment which can give you information by means of a beta window reading, or what have you, it still brings up the point of how many of these instruments will be required to give you a meaningful reading. The Army can only support so many of these instruments, and so many different types to do a given survey job.

At the present time their concept is that two gamma measuring instruments per company will give them an indication of the contours in the company area. I wonder how many instruments it would take of a beta detecting ability to do the same thing in view of the fact that you have such marked variations. In the beta hazard part of this thing, there is so much variation of the beta dose within a given area, if you are going to get a meaningful survey, it seems to me you would have to take an awful lot of instrumentation to do it. If you were going to use beta detection for the other type of beta hazard, the point contact which results from personnel contamination, again the question comes if you take a simple company with 250 men distributed in a forward area, how do you go about monitoring every individual, finding out whether he is contaminated, and to what level?

There are other practical limitations, and that is the availability of personnel to do the monitoring. The present concept in the Army is that monitoring will be taught as part of the basic soldierly skill. It will be taught to enlisted men in basic training. It is not simple now-a-days to get enlisted men to do ordinary simple gamma measuring in the field. The question of getting meaningful beta readings I think is recognized even among people who know what they are doing as a quite difficult thing. Just how to interpret an instrument reading with the window closed and open takes quite a bit of interpreting.

Then the last consideration as far as the practical limitation is concerned is that we know that the instrument can do rugged work. We know even in the hands of technical people these windows have a habit of being punctured. If you distribute these types of instruments to personnel in the field, you stand a very good chance of winding up with no beta detection capability but without any gamma instrumentation as well.

I think certain operational capabilities and limitations must be thrown into the picture fairly early in the game so it indicates the direction in which the research and development effort might be more profitably directed.

Col. Brennan. Thank you. That was very illuminating. Certainly no one can say in view of the Marshallese and the other data are available that there is no such thing as a beta hazard as we used to think. The answers to the problems that Mr. Lindwarm poses certainly I don't know. I suspect from an efficiency point of view, the Army and the Armed Forces and the Civil Defense people should emphasize prophylaxis with regard to beta rays. The Marshallese tend to maximize this information for us by wearing few clothing, living out of doors, a hot climate where they perspire and so forth. One can look at this and realize the undesirability and seriousness of it, and perhaps take care of it by enforcing simple measures, keep your sleeves down, your helmet on, don't go in contaminated areas, and so forth.

The point contact can largely be avoided for at least military personnel by simply battlefield hygiene measures. The external beta component, whether this has to be allowed for or routinely measured or measured once in a while, I think it is impossible to say at the present time without more experimental data, and a good deal more development of doctrine and philosophy. I think the beta problem is going to be with us militarily and civilianwise for quite some time.

There are many, many industrial hazard situations in which the beta hazard has likewise been sort of shoved in the background, and not solved, because it was hard to approach. There are many instances in which you have insoluble particles in the air, many industrial hazards that are regarded as gaseous and liquid, which are really not. If the truth were known they are particulate and give the point contact for a beta hazard if they are inhaled.

Does anyone have any further comment?

Mr. Greene (FCDA). For some time we have felt that there was a need for making beta measurements, especially for certain types of civil defense operations. The most obvious that I can think of would be rescue workers who are working in debris and who would have their faces and hands close to the sources of radiation. It would certainly be important for them to know whether they are working in an area that actually has the contamination in the debris where they are working or whether the main source of radiation is from the outside.

From that standpoint we have felt that there is a requirement for the measurement—and I use the term rather loosely—of beta radiation and we have incorporated that into our specifications. We actually have an instrument that is now beginning to come off the production line which measures beta. The problem of fragility is certainly a serious one. We therefore have not attempted to have a beta window as thin as the 7 milligrams per square centimeter that one might ideally want. What we have done is used a thicker window, and from work in Nevada, and work with Dr. Failla, we believe we can get a portion of the beta radiation which is relatively constant with time and from that portion with calibration curve get some idea of the total beta radiation dose.

Mr. Lindwarm. This fight has been going on for so many years that it is funny. I question the requirement why you have to know you are operating in a contaminated area. You have a gamma reading to tell you that. You mean if you find beta, you will take gloves off or if there is none, you will take them off.

Mr. Greene. You are working with the Chemical Corps. You ought to know more about it than anybody else. I was talking about your face and hands. If you are working in rubble, you are close to the debris and your face is close to it.

Mr. Lindwarm. I doubt that there would be any requirement at any time if you are doing emergency rescue work to go in with a gas mask for the simple purpose of protecting your face. As soon as you got out you would wash your hands and face.

Col. Brennan. Is the gas mask to protect from inhalation?

Mr. Greene. I was thinking of a mask to

protect against the radiation. Maybe it would not be a gas mask. This illustrates our difficulty of working with your face covered.

Mr. LINDWARM. I wonder about the payoff of this thing. There are lots of things you can do about it. The question in my mind is just what the payoff is when you go to the trouble of putting in something like that that might wind up giving you something useless in the long run.

Mr. GREENE. Let me mention that if one does use an ionization chamber at atmospheric pressure, and it is not punctured, you can still use it. I think we better fight this out some other way.

Col. BRENNAN. This is a very interesting angle and the whole point of looking at this. Does anybody have any other comments?

Dr. MORGAN (ORNL). I didn't want to get into this argument, but I recall away back in the Bikini days that they threatened to throw me overboard unless I kept my mouth shut and quit complaining about the beta-gamma ratio. Finally they gave me a crew of men and we went out and made measurements and as I indicated before on the topside of MFT ships, we found values as high as several hundred. In one case it was as high as 700. In such a situation it was common practice for the fellows to sleep topside with little or no clothing on, and if they relied completely on the reading of the gamma instruments, say 1 r per hour, and slept there through the night with 700 r per hour, they would have had quite a nice erythema and would have ended up with ulcers and other difficulties. So I began then the argument that under certain situations it is quite vital that we do measure the beta-gamma ratio or measure the beta dose even though it is a difficult job. We do it in the laboratory under all types of conditions. It can be done. I know that there are some problems but just because a job is hard to do, I think is no reason why you should run the risk of sacrificing the lives of people.

Col. BRENNAN. I certainly would agree with that. In general, then, the responsive action would be to either protect against it in terms of clothing or time or geometry, and be very sure you have good protection or if you can't do that, you are pretty much committed to measure it. At least measure it often enough to control the hazard, however difficult that may be. That is at least the direction one ought to go.

Topic V

Internal Emitters

INTERNAL DOSE FROM SHORT-LIVED RADIONUCLIDES

By KARL Z. MORGAN

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INTERNAL DOSE FROM SHORT-LIVED RADIONUCLIDES

The National Committee on Radiation Protection (NCRP) and the International Commission on Radiological Protection (ICRP) set the national and international standards for radiation protection. One of the important assignments of these organizations has been the establishment of the maximum permissible body burden, q, and the maximum permissible concentrations, MPC, of the radionuclides in air, water, and food. To the present time only q and MPC values for continuous exposure to the radionuclides have been published in the NCRP [1] and the ICRP [2] Handbooks. Values for single exposure are being considered but it will probably be several years before final agreement is reached on a set of values.

The ICRP Handbook, which was published about two years after the NCRP Handbook 52, differs in some respects from the earlier publication. Perhaps the most important change was the incorporation of MPC values based on the G.I. tract as the critical body organ. The importance of this is emphasized by the fact that of the 355 MPC values listed in the ICRP Handbook, 71 percent for ingestion and 41 percent for inhalation refer to the G.I. tract as the critical body organ. The bone is the second most important body organ and is the critical body organ for 11 percent of the MPC values for ingestion and 28 percent of the MPC values for inhalation. The G.I. tract was not included as a critical body organ in Handbook 52 because at the time of the publication of Handbook 52 radiation damage per roentgen to the G.I. tract was considered less significant than that to the other body organs and because insufficient data were available to estimate the absorbed dose and corresponding hazard to various portions of the G.I. tract. When Dr. E. E. Pochin [3] furnished to the ICRP Committee data on the mass and time distribution of material in the G.I. tract, it became evident that the G.I. tract receives the greatest absorbed dose from many of the radionuclides. The lower large intestine is the critical portion of the G.I. tract for all radionuclides considered in the ICRP Handbook with the exception of Mn^{56} and F^{9}, in which cases the upper large intestine and stomach are the critical portions of the G.I. tract, respectively. The lower large intestine is usually the critical portion of the G.I. tract for three reasons:

1. Only radionuclides with a radioactive half-life greater than 1 hour were considered in the preparation of the ICRP Handbook.
2. The contaminated material remains in the lower large intestine 18 of the 31 hours that it is in the G.I. tract.
3. The mass of material in which the radionuclide is diluted and to which the radiation dose is delivered is relatively small, i. e., 150 g in the lower large intestine, 250 g in the stomach, 1100 g in the small intestine, and 135 g in the upper large intestine.

[1] Handbook 52, "Maximum Permissible Amounts of Radioisotopes in the Human Body and Maximum Permissible Concentrations in Air and Water," by National Committee on Radiation Protection, *National Bureau of Standards*, Washington, D. C. (1953).

[2] ICRP Handbook, "Recommendations of the International Commission on Radiological Protection," *Br. Jr. of Radiology*, Supplement 6, British Institute of Radiology, London (1955).

[3] Private Communication from E. E. Pochin, Director, Department of Clinical Research, University College Hospital, Medical School, University Street, London, W. C. 1., Great Britain, to K. Z. Morgan, dated October 9, 1953.

Therefore, most of the absorbed dose (in ergs/g or rad units) is delivered to the lower large intestine in the case of the radionuclides with relatively long half-lives which are listed in the ICRP Handbook. The material spends 1 hour in the stomach, 4 hours in the small intestine, 8 hours in the upper large intestine, and 18 hours in the lower large intestine. If radionuclides of shorter half-life are considered in future publications and if MPC values are given for single exposure, we may expect other portions of the G.I. tract to become the critical body tissue. In the present ICRP Handbook the assumption is made that the fraction, f_1, of the radionuclide passes through the small intestine into the blood so that only $(1-f_1)$ reaches the upper and lower large intestine. Therefore, if radionuclides are considered in which $f_1 \simeq 1$, the critical organs are unlikely to be the large intestines.

Although no official MPC or q values for a short period of single exposure have been agreed upon, unofficial single exposure values have been adopted by some of the laboratories working with certain of the radionuclides in order to aid in assessing the hazards associated with accidents or "spills." Previous attempts have been made to prepare tables of MPC values for single exposure. Tables of MPC values of some 80 radionuclides were given for two cases: 1. The radionuclide is taken into the body by inhalation or ingestion over a 24-hour period,[4] and 2. The radionuclide is taken into the body by inhalation or ingestion over an 8-hour period, or by way of a contaminated wound.[5] None of these single exposure values has official status.

It is to be expected that single exposure values will be included in the Internal Dose Handbooks in the near future. Table I is a summary of the single exposure data given at the Conference on Peaceful Uses of Atomic Energy in 1955 at Geneva[5]. Single exposure values were given for 5 different sets of assumptions: 1. Soluble radioactive material is inhaled; 2. A wound is contaminated with soluble radioactive material; 3. A wound is contaminated with insoluble radioactive material; 4. Insoluble radioactive material is inhaled with the lung as the critical body organ; and 5. Insoluble radioactive material is inhaled with various portions of the G.I. tract as the critical body organ. The calculations were made for three criteria relative to permissible exposure and only the lowest maximum permissible values are listed in Table I. The three assumed permissible exposures were 0.3 rem/wk, 15.7 rem/yr or 150 rem/70 years following the exposure. We should note that in Tables I and II the 0.3 rem/wk is the limiting case—gives the smallest maximum permissible values—with the exception of 5 bone-seeking radionuclides ($Sr^{90}+Y^{90}$, Sm^{151}, Ra^{226}, natural thorium, and Pu^{239}) in the soluble form. In these cases, 150 rem/70 years is the limiting case. In the case of a wound contaminated with insoluble radioactive material it was assumed that all the contamination remained in 1 mg of tissue at the wound site. In the cases of inhalation of radioactive material, the μc values given in Tables I and II correspond to the amount of the radionuclides initially present in the air—for example, as the result of an accident—which if inhaled over a period of 8 hours would deliver the indicated dose. The radioactive material decays with a half-life T_r in this period, i. e., both before and after inhalation, and is eliminated with a biological half-life T_b while in the body. In the case of a wound all the radioactive contamination enters the wound at time zero. Values were not given in the Geneva paper for ingestion because experience had indicated that in cases of single exposure, inhalation in general presents a much greater hazard than ingestion. However, in some accident cases one may be concerned with the ingestion problem and so Table II is added to give the μc that are considered permissible to ingest in an 8-hour period. Tables I and II list values of μc present initially at the time of an accident, e. g., spill, explosion, failure of a

[4] Morgan, K. Z. and M. R. Ford, "Developments in Internal Dose Determinations," *Nucleonics*, Vol. 12, No. 6, 32 (June 1954).

[5] Morgan, K. Z., W. S. Snyder, and M. R. Ford, "Maximum Permissible Concentration of Radionuclides in Air and Water for Short Period Exposure," International Conference on the Peaceful Uses of Atomic Energy, Geneva, Switzerland (1955).

TABLE I.—MAXIMUM PERMISSIBLE OCCUPATIONAL EXPOSURE

Single Exposure Values for Radionuclides for Inhalation and for Contaminated Wounds

Z	Isotope	Inhalation [1] of soluble radioactive material (μc initially available to be inhaled during succeeding 8 hrs) [2]	Wound contamination		Inhalation [1] of insoluble radioactive material	
			(μc of soluble radioactive material initially in wound) [2]	Wound critical tissue (μc of insoluble radioactive material initially in wound)	Lung critical organ (μc initially available to be inhaled during succeeding 8 hrs)	GI tract critical organ (μc initially available to be inhaled during succeeding 8 hrs) [2]
(1)	(2) [3]	(3)	(4)	(5)	(6)	(7)
1	H^3 (HTO or H^3_2O)	1.5×10^4 (0.3, TB)	1.1×10^4 (0.3, TB)	1.4×10^{-4} (0.3)	1.1×10^4 (0.3)	1.2×10^4 (0.3, LLI)
4	Be^7	7.7×10^3 (0.3, B)	2.0×10^3 (0.3, B)	8.1×10^{-4} (0.3)	1.9×10^3 (0.3)	4.2×10^3 (0.3, LLI)
6	C^{14} (CO_2)	4.7×10^2 (0.3, F)	3.4×10^2 (0.3, F)	1.6×10^{-3} (0.3)	3.3×10^2 (0.3)	1.4×10^3 (0.3, LLI)
9	F^{18}	1.3×10^4 (0.3, B)	3.0×10^3 (0.3, B)	2.2×10^{-4} (0.3)	5.3×10^3 (0.3)	1.7×10^3 (0.3, S)
11	Na^{24}	2.8×10^3 (0.3, TB)	1.7×10^3 (0.3, TB)	9.8×10^{-4} (0.3)	23 (0.3)	57 (0.3, S)
15	P^{32}	53 (0.3, B)	34 (0.3, B)	1.5×10^{-2} (0.3)	12 (0.3)	19 (0.3, LLI)
16	S^{35}	4.7×10^2 (0.3, sk)	2.5×10^2 (0.3, sk)	1.6×10^{-1} (0.3)	1.3×10^2 (0.3)	1.6×10^2 (0.3, LLI)
17	Cl^{36}	3.7×10^2 (0.3, TB)	2.6×10^2 (0.3, TB)	3.3×10^{-6} (0.3)	26 (0.3)	2.9×10^2 (0.3, LLI)
19	K^{42}	3.3×10^3 (0.3, M)	1.9×10^3 (0.3, M)	5.6×10^{-6} (0.3)	50 (0.3)	98 (0.3, S)
20	Ca^{45}	33 (0.3, B)	24 (0.3, B)	3×10^{-5} (0.3)	80 (0.3)	4.4×10^2 (0.3, LLI)
21	Sc^{46}	46 (0.3, s) 29 (0.3, L)	10 (0.3, s) 57 (0.3, L)	6.5×10^{-6} (0.3)	5.7 (0.3)	9.2 (0.3, LLI)
21	Sc^{47}	1.8×10^3 (0.3, s) 1.2×10^3 (0.3, L)	31 (0.3, s) 24 (0.3, L)	7.5×10^{-6} (0.3)	63 (0.3)	21 (0.3, LLI)
21	Sc^{48}	87 (0.3, s) 50 (0.3, L)	20 (0.3, s) 9.4 (0.3, L)	3×10^{-5} (0.3)	13 (0.3)	9.1 (0.3, LLI)
23	V^{48}	2.7×10^3 (0.3, B)	64 (0.3, B)	6.6×10^{-6} (0.3)	4.7 (0.3)	7.1 (0.3, LLI)
24	Cr^{51}	10^4 (0.3, K)	2.8×10^3 (0.3, K)	4.6×10^{-4} (0.3)	6.7×10^3 (0.3)	5.0×10^3 (0.3, LLI)
25	Mn^{56}	1.2×10^3 (0.3, K) 1.6×10^3 (0.3, L)	1.3×10^3 (0.3, K) 3.7×10^3 (0.3, L)	4.4×10^{-3} (0.3)	4×10^2 (0.3)	80 (0.3, ULI)
26	Fe^{55}	1.2×10^3 (0.3, Bl)	7.8×10^2 (0.3, Bl)	1.4×10^{-4} (0.3)	1.1×10^3 (0.3)	3.1×10^3 (0.3, LLI)
26	Fe^{59}	14 (0.3, Bl)	9.1 (0.3, Bl)	7.5×10^{-6} (0.3)	9 (0.3)	68 (0.3, LLI)
27	Co^{60}	3.7×10^2 (0.3, Li)	1.3×10^2 (0.3, Li)	7.8×10^{-6} (0.3)	4.7 (0.3)	11 (0.3, LLI)
28	Ni^{59}	5.7×10^3 (0.3, Li)	1.9×10^3 (0.3, Li)	1.7×10^{-3} (0.3)	1.4×10^3 (0.3)	94 (0.3, LLI)
29	Cu^{64}	8.3×10^3 (0.3, Li)	2.6×10^3 (0.3, Li)	7.5×10^{-3} (0.3)	3.7×10^3 (0.3)	1.1×10^3 (0.3, LLI)
30	Zn^{65}	1.5×10^3 (0.3, B)	4.5×10^2 (0.3, B)	6.6×10^{-3} (0.3)	21 (0.3)	49 (0.3, LLI)
31	Ga^{72}	2.2×10^3 (0.3, B)	46 (0.3, B)	1.5×10^{-3} (0.3)	33 (0.3)	11 (0.3, LLI)
32	Ge^{71}	9.0×10^3 (0.3, K)	2.2×10^3 (0.3, K)	1.1×10^{-4} (0.3)	8.7×10^2 (0.3)	3.9×10^3 (0.3, LLI)
33	As^{76}	4.3×10^3 (0.3, K)	10^3 (0.3, K)	3.6×10^{-6} (0.3)	25 (0.3)	5.4 (0.3, LLI)
37	Rb^{86}	1.4×10^3 (0.3, M)	10^3 (0.3, M)	1.6×10^{-6} (0.3)	11 (0.3)	1.3×10^3 (0.3, LLI)
38	Sr^{89}	16 (0.3, B)	5.5 (0.3, B)	1.6×10^{-6} (0.3)	13 (0.3)	17 (0.3, LLI)
38	$Sr^{90}+Y^{90}$	5 (150, B)	2.7 (150, B)	8.5×10^{-7} (0.3)	7 (0.3)	21 (0.3, LLI)
39	Y^{91}	16 (0.3, B)	4.0 (0.3, B)	1.6×10^{-4} (0.3)	13 (0.3)	6.6 (0.3, LLI)
40	$Zr^{95}+Nb^{95}$	1.1×10^3 (0.3, B)	27 (0.3, B)	4.9×10^{-6} (0.3)	7.3 (0.3)	16 (0.3, LLI)
41	Nb^{95}	1.7×10^3 (0.3, B)	79 (0.3, B)	1.9×10^{-6} (0.3)	14 (0.3)	46 (0.3, LLI)
42	Mo^{99}	9.3×10^3 (0.3, B)	5.9×10^3 (0.3, B)	1.4×10^{-5} (0.3)	28 (0.3)	72 (0.3, LLI)
43	Tc^{96}	4.7×10^3 (0.3, K)	2.6×10^3 (0.3, K)	5.5×10^{-3} (0.3)	8 (0.3)	24 (0.3, LLI)
44	$Ru^{106}+Rh^{106}$	20 (0.3, K)	5.1 (0.3, K)	6.3×10^{-7} (0.3)	4.7 (0.3)	2.7 (0.3, LLI)
45	Rh^{105}	1.4×10^3 (0.3, K)	51 (0.3, K)	10^{-3} (0.3)	53 (0.3)	22 (0.3, LLI)
46	$Pd^{103}+^{109}$	1.4×10^3 (0.3, K)	56 (0.3, K)	2.7×10^{-3} (0.3)	1.1×10^3 (0.3)	1.2×10^3 (0.3, LLI)
47	Ag^{105}	2.0×10^3 (0.3, L)	6.8×10^2 (0.3, L)	9.0×10^{-3} (0.3)	4.3 (0.3)	9.4 (0.3, LLI)
47	Ag^{111}	5.0×10^3 (0.3, L)	1.6×10^3 (0.3, L)	3.1×10^{-5} (0.3)	26 (0.3)	11 (0.3, LLI)
48	$Cd^{109}+Ag^{109}$	2.0×10^3 (0.3, L)	49 (0.3, L)	2.1×10^{-4} (0.3)	97 (0.3)	1.3×10^3 (0.3, LLI)
50	Sn^{113}	9.3×10^3 (0.3, B)	2.4×10^3 (0.3, B)	4.4×10^{-4} (0.3)	23 (0.3)	44 (0.3, LLI)
52	Te^{127}	53 (0.3, K)	18 (0.3, K)	3.2×10^{-6} (0.3)	22 (0.3)	18 (0.3, LLI)
52	Te^{129}	18 (0.3, K)	5.9 (0.3, K)	1.4×10^{-6} (0.3)	5.3 (0.3)	6.1 (0.3, LLI)
53	I^{131}	0.70 (0.3, T)	0.52 (0.3, T)	6.3×10^{-6} (0.3)	22 (0.3)	2.9×10^2 (0.3, SI)
55	$Cs^{137}+Ba^{137}$	1.4×10^3 (0.3, M)	1.1×10^3 (0.3, M)	4.7×10^{-6} (0.3)	12 (0.3)	2.2×10^3 (0.3, SI)
56	$Ba^{140}+La^{140}$	8.7 (0.3, B)	2.4 (0.3, B)	1.3×10^{-6} (0.3)	4.3 (0.3)	7 (0.3, LLI)
57	La^{140}	20 (0.3, B)	16 (0.3, B)	4.8×10^{-6} (0.3)	15 (0.3)	6.6 (0.3, LLI)
58	$Ce^{144}+Pr^{144}$	9.3 (0.3, B)	2.3 (0.3, B)	6.9×10^{-7} (0.3)	5 (0.3)	2.9 (0.3, LLI)
59	Pr^{142}	73 (0.3, B)	18 (0.3, B)	3.3×10^{-6} (0.3)	27 (0.3)	13 (0.3, LLI)
61	Pm^{147}	2.0×10^3 (0.3, B)	50 (0.3, B)	1.3×10^{-2} (0.3)	1.0×10^3 (0.3)	56 (0.3, LLI)
62	Sm^{151}	2.4×10^3 (150, B)	60 (150, B)	4.3×10^{-3} (0.3)	3.3×10^3 (0.3)	1.9×10^3 (0.3, LLI)
63	Eu^{154}	57 (0.3, B)	14 (0.3, B)	4.1×10^{-6} (0.3)	8.3 (0.3)	11 (0.3, LLI)
67	Ho^{166}	1.3×10^3 (0.3, B)	37 (0.3, B)	5.7×10^{-6} (0.3)	47 (0.3)	10 (0.3, LLI)
69	Tm^{170}	21 (0.3, B)	5.4 (0.3, B)	2.7×10^{-6} (0.3)	22 (0.3)	12 (0.3, LLI)
71	Lu^{177}	2.4×10^3 (0.3, B)	58 (0.3, B)	9.2×10^{-6} (0.3)	57 (0.3)	28 (0.3, LLI)

See footnotes at end of table.

TABLE I.—MAXIMUM PERMISSIBLE OCCUPATIONAL EXPOSURE—Continued

Single Exposure Values for Radionuclides for Inhalation and for Contaminated Wounds

Z	Isotope	Inhalation [1] of soluble radioactive material (μc initially available to be inhaled during succeeding 8 hrs) [2]		Wound contamination (μc of soluble radioactive material initially in wound) [3]		Wound critical tissue (μc of insoluble radioactive material initially in wound)		Inhalation [1] of insoluble radioactive material Lung critical organ (μc initially available to be inhaled during succeeding 8 hrs)		GI tract critical organ (μc initially available to be inhaled during succeeding 8 hrs) [2]	
(1)	(2) [3]	(3)		(4)		(5)		(6)		(7)	
73	Ta^{182}	47	(0.3, L)	11	(0.3, L)	5.1×10^{-4}	(0.3)	8.7	(0.3)	12	(0.3, LLI)
74	W^{181}	9.3×10^{2}	(0.3, B)	2.1×10^{2}	(0.3, B)	2.9×10^{-3}	(0.3)	6.3	(0.3)	16	(0.3, LLI)
75	Re^{186}	1.4×10^{2}	(0.3, T)	7.2×10^{2}	(0.3, T)	2.9×10^{-4}	(0.3)	11	(0.3)	50	(0.3, LLI)
		7.3×10^{2}	(0.3, sk)	1.1×10^{2}	(0.3, sk)						
77	Ir^{190}	2.9×10^{2}	(0.3, K)	98	(0.3, K)	3.1×10^{-3}	(0.3)	43	(0.3)	81	(0.3, LLI)
		4.0×10^{2}	(0.3, s)	28	(0.3, s)						
77	Ir^{191}	31	(0.3, K)	12	(0.3, K)	4.0×10^{-4}	(0.3)	7.3	(0.3)	12	(0.3, LLI)
		47	(0.3, s)	3.6	(0.3, s)						
78	Pt^{191}	48	(0.3, K)	12	(0.3, K)	2.9×10^{-4}	(0.3)	12	(0.3)	18	(0.3, LLI)
78	Pt^{193}	40	(0.3, K)	11	(0.3, K)	2.8×10^{-4}	(0.3)	12	(0.3)	21	(0.3, LLI)
79	Au^{196}	53	(0.3, L)	13	(0.3, L)	2.6×10^{-4}	(0.3)	28	(0.3)	51	(0.3, LLI)
		43	(0.3, K)	13	(0.3, K)						
79	Au^{198}	31	(0.3, L)	7.4	(0.3, L)	5.8×10^{-4}	(0.3)	27	(0.3)	14	(0.3, LLI)
		20	(0.3, K)	5.8	(0.3, K)						
79	Au^{199}	73	(0.3, L)	18	(0.3, L)	1.6×10^{-3}	(0.3)	63	(0.3)	40	(0.3, LLI)
		53	(0.3, K)	15	(0.3, K)						
81	Tl^{200}	4.0×10^{2}	(0.3, M)	1.6×10^{2}	(0.3, M)	3.3×10^{-3}	(0.3)	26	(0.3)	31	(0.3, LLI)
81	Tl^{201}	1.4×10^{2}	(0.3, M)	5.8×10^{2}	(0.3, M)	2.9×10^{-3}	(0.3)	90	(0.3)	2.2×10^{2}	(0.3, LLI)
81	Tl^{202}	6.7×10^{2}	(0.3, M)	2.8×10^{2}	(0.3, M)	1.0×10^{-2}	(0.3)	40	(0.3)	1.2×10^{2}	(0.3, LLI)
81	Tl^{204}	4.7×10^{2}	(0.3, M)	1.9×10^{2}	(0.3, M)	3.4×10^{-3}	(0.3)	27	(0.3)	27	(0.3, LLI)
82	Pb^{203}	1.3×10^{3}	(0.3, B)	4.1×10^{2}	(0.3, B)	2.1×10^{-2}	(0.3)	37	(0.3)	46	(0.3, LLI)
82	Pb^{210}+dr.	0.21	(0.3, B)	7.1×10^{-2}	(0.3, B)	1.5×10^{-5}	(0.3)	0.12	(0.3)	39	(0.3, LLI)
84	Po^{210}	0.60	(0.3, s)	0.12	(0.3, s)	1.6×10^{-5}	(0.3)	0.12	(0.3)	6.4×10^{-2}	(0.3, LLI)
85	At^{211}+dr.	0.10	(0.3, T)	5.3×10^{-2}	(0.3, T)	1.8×10^{-7}	(0.3)	3.1	(0.3)	2.4	(0.3, S)
88	Ra^{226}+55% dr.	0.32	(150, B)	0.11	(150, B)	5.3×10^{-5}	(0.3)	2.8×10^{-2}	(0.3)	5.1×10^{-2}	(0.3, LLI)
89	Ac^{227}+dr.	4.7×10^{-3}	(0.3, B)	1.2×10^{-3}	(0.3, B)	2.5×10^{-6}	(0.3)	2.1×10^{-3}	(0.3)	1.1	(0.3, LLI)
90	Th-nat	2.6×10^{-3}	(150, B)	6.7×10^{-4}	(150, B)	2.3×10^{-6}	(0.3)	1.9×10^{-3}	(0.3)	0.03	(0.3, LLI)
90	Th^{234}+Pa^{234}	7.7	(0.3, B)	1.9	(0.3, B)	1.1×10^{-6}	(0.3)	8.3×10^{-3}	(0.3)	4.4	(0.3, LLI)
92	U-nat.	3.7×10^{-3}	(0.3, K)	8.7×10^{-4}	(0.3, K)	9.1×10^{-6}	(0.3)	7.3×10^{-2}	(0.2)	0.04	(0.3, LLI)
92	U^{233}	0.43	(0.3, B)	7.1×10^{-2}	(0.3, B)	1.7×10^{-5}	(0.3)	0.14	(0.3)	7.5×10^{-3}	(0.3, LLI)
94	Pu^{239}	2.0×10^{-2}	(150, B)	5.2×10^{-3}	(150, B)	1.6×10^{-5}	(0.3)	0.13	(0.3)	7.1×10^{-3}	(0.3, LLI)
95	Am^{241}	0.33	(0.3, B)	8.4×10^{-2}	(0.3, B)	1.5×10^{-5}	(0.3)	0.12	(0.3)	6.7×10^{-3}	(0.3, LLI)
96	Cm^{242}	0.29	(0.3, B)	7.6×10^{-2}	(0.3, B)	1.4×10^{-5}	(0.3)	0.11	(0.3)	6.0×10^{-3}	(0.3, LLI)

[1] The values μc inhaled (as given in columns 3, 6, and 7) can be converted to MPC in μc/cc of air by multiplying by 10^{-7}.

[2] TB—total body, B—bone, F—fat, sk—skin, M—muscle, s—spleen, L—liver, K—kidney, Bl—blood, T—thyroid, LLI—lower large intestine, ULI—upper large intestine, S—stomach, SI—small intestine. The letters given in parentheses indicate the critical body organ.

[3] The s in column 2 indicates daughter products that are isomers in an excited state.

NOTE.—The 0.3 or 150 in parentheses refers to the limiting dose rate of 0.3 rem/wk or 150 rem/70 yrs—whichever gives the smaller maximum permissible value.

chemical hood, etc., which if inhaled (Table I) or ingested (Table II) *for an 8-hour period would result in the indicated dose.* The values in Table I, columns 3, 6, and 7, can be converted to MPC in μc/cc of air by *multiplying them by* 10^{-7}. The values in Table II, columns 3 and 4, were converted to MPC in μc/cc of water by *dividing them by 1,100 cc.* These values of μc/cc of water are entered for convenience in columns 5 and 6 of Table II.

It is to be noted that maximum permissible values for wounds contaminated with insoluble radioactive material are several orders of magnitude smaller than the values for the other cases. This should serve to emphasize that perhaps contaminated wounds are the greatest radiation hazard in the laboratory. However, when applying this information to the problem of fallout material from the testing of nuclear weapons, it is probably safe to

TABLE II.—MAXIMUM PERMISSIBLE OCCUPATIONAL EXPOSURE

Single Exposure Values for Ingestion of Radionuclides

Z	Isotope	Ingestion of soluble radioactive material (μc initially available to be ingested during succeeding 8 hrs) [1]		Ingestion of insoluble radioactive material (μc initially available to be ingested during succeeding 8 hrs) [1]		Ingestion of soluble radioactive material (μc/cc in water ingested in 8 hrs) [2]		Ingestion of insoluble radioactive material (μc/cc in water ingested during 8 hrs) [1]	
(1)	(2) [3]	(3)		(4)		(5)		(6)	
1	H^{3} (HTO or $H^{3}_{2}O$)	1.1×10^{4}	(0.3, TB)	7.5×10^{3}	(0.3, LLI)	10	(0.3, TB)	6.8	(0.3, LLI)
4	Be^{7}	1.7×10^{4}	(0.3, B)	2.6×10^{3}	(0.3, LLI)	1.5×10^{3}	(0.3, B)	0.24	(0.3, LLI)
6	C^{14} (CO_2)	3.4×10^{3}	(0.3, F)	8.8×10^{2}	(0.3, LLI)	0.31	(0.3, F)	0.80	(0.3, LLI)
9	F^{18}	9.8×10^{3}	(0.3, B)	1.1×10^{3}	(0.3, S)	8.0	(0.3, B)	1.0	(0.3, S)
11	Na^{24}	2.2×10^{3}	(0.3, TB)	36	(0.3, SI)	0.20	(0.3, TB)	3.3×10^{-3}	(0.3, SI)
15	P^{32}	53	(0.3, B)	12	(0.3, LLI)	4.3×10^{-2}	(0.3, B)	1.1×10^{-2}	(0.3, LLI)
16	S^{35}	4.3×10^{3}	(0.3, sk)	10^{3}	(0.3, LLI)	0.39	(0.3, sk)	9.1×10^{-2}	(0.3, LLI)
17	Cl^{36}	2.8×10^{3}	(0.3, TB)	1.8×10^{3}	(0.3, LLI)	0.25	(0.3, TB)	0.16	(0.3, LLI)
19	K^{42}	2.5×10^{3}	(0.3, M)	61	(0.3, S)	0.23	(0.3, M)	5.5×10^{-2}	(0.3, S)
20	Ca^{45}	54	(0.3, B)	2.8×10^{3}	(0.3, LLI)	4.9×10^{-2}	(0.3, B)	0.25	(0.3, LLI)
21	Sc^{46}	1.5×10^{4}	(0.3, s)	5.8	(0.3, LLI)	14	(0.3, s)	5.3×10^{-3}	(0.3, LLI)
		1.7×10^{4}	(0.3, L)			15	(0.3, L)		
21	Sc^{47}	6.8×10^{4}	(0.3, s)	13	(0.3, LLI)	62	(0.3, s)	1.2×10^{-2}	(0.3, LLI)
		7.2×10^{4}	(0.3, L)			65	(0.3, L)		
21	Sc^{48}	$3.3\times10_{4}$	(0.3, s)	5.7	(0.3, LLI)	30	(0.3, s)	5.2×10^{-3}	(0.3, LLI)
		3.0×10^{4}	(0.3, L)			27	(0.3, L)		
23	V^{48}	1.4×10^{4}	(0.3, B)	4.4	(0.3, LLI)	13	(0.3, B)	4.0×10^{-3}	(0.3, LLI)
24	Cr^{51}	5.7×10^{4}	(0.3, K)	3.1×10^{3}	(0.3, LLI)	52	(0.3, K)	0.28	(0.3, LLI)
25	Mn^{56}	6.6×10^{3}	(0.3, K)	50	(0.3, ULI)	6.0	(0.3, K)	4.5×10^{-3}	(0.3, ULI)
		1.4×10^{4}	(0.3, L)			13	(0.3, L)		
26	Fe^{55}	9.8×10^{3}	(0.3, Bl)	1.9×10^{3}	(0.3, LLI)	0.89	(0.3, Bl)	1.7	(0.2, LLI)
26	Fe^{59}	11	(0.3, Bl)	43	(0.3, LLI)	10^{-2}	(0.3, Bl)	2.9×10^{-3}	(0.3, LLI)
27	Co^{60}	6.5×10^{3}	(0.3, L)	6.9	(0.3, LLI)	0.59	(0.3, L)	6.3×10^{-3}	(0.3, LLI)
28	Ni^{59}	10^{4}	(0.3, L)	59	(0.3, LLI)	9.1	(0.3, L)	5.4×10^{-2}	(0.3, LLI)
29	Cu^{64}	1.2×10^{4}	(0.3, L)	69	(0.3, LLI)	1.1	(0.3, L)	6.3×10^{-3}	(0.3, LLI)
30	Zn^{65}	3.5×10^{3}	(0.3, B)	31	(0.3, LLI)	3.2	(0.3, B)	2.8×10^{-3}	(0.3, LLI)
31	Ga^{72}	5.5×10^{3}	(0.3, B)	6.9	(0.3, LLI)	50	(0.3, B)	6.3×10^{-3}	(0.3, LLI)
32	Ge^{71}	2.3×10^{4}	(0.3, K)	2.4×10^{3}	(0.3, LLI)	2.1×10^{3}	(0.3, K)	0.22	(0.3, LLI)
33	As^{76}	3.9×10^{3}	(0.3, K)	3.4	(0.3, LLI)	3.5	(0.3, K)	3.1×10^{-3}	(0.3, LLI)
37	Rb^{86}	1.1×10^{3}	(0.3, M)	81	(0.3, LLI)	0.10	(0.3, M)	7.4×10^{-3}	(0.3, LLI)
38	Sr^{89}	8.8	(0.3, B)	11	(0.3, LLI)	8.0×10^{-3}	(0.3, B)	10^{-2}	(0.3, LLI)
39	Sr^{90}+Y^{90}	4.4	(150, B)	13	(0.3, LLI)	4.0×10^{-3}	(150, B)	1.2×10^{-3}	(0.3, LLI)
39	Y^{91}	7.5	(0.3, B)	4.1	(0.3, LLI)	6.8	(0.3, B)	3.7×10^{-3}	(0.3, LLI)
40	Zr^{95}+Nb^{95}	6.4×10^{3}	(0.3, B)	10	(0.3, LLI)	58	(0.3, B)	9.1×10^{-3}	(0.3, LLI)
41	Nb^{95}	1.6×10^{3}	(0.3, B)	29	(0.3, LLI)	0.15	(0.3, B)	2.6×10^{-2}	(0.3, LLI)
42	Mo^{99}	9.3×10^{3}	(0.3, B)	45	(0.3, LLI)	85	(0.3, B)	4.1×10^{-3}	(0.3, LLI)
43	Tc^{96}	4.7×10^{3}	(0.3, K)	15	(0.3, LLI)	0.43	(0.3, K)	1.4×10^{-2}	(0.3, LLI)
44	Ru^{103}+Rh^{103}	10^{4}	(0.3, K)	1.7	(0.3, LLI)	9.1	(0.3, K)	1.5×10^{-3}	(0.3, LLI)
45	Rh^{105}	2.8×10^{3}	(0.3, K)	14	(0.3, LLI)	0.25	(0.3, K)	1.3×10^{-2}	(0.3, LLI)
46	Pd^{103}+Rh^{103}s	2.8×10^{3}	(0.3, K)	75	(0.3, LLI)	0.25	(0.3, K)	6.8×10^{-3}	(0.3, LLI)
47	Ag^{105}	4.0×10^{4}	(0.3, L)	5.9	(0.3, LLI)	36	(0.3, L)	5.4×10^{-3}	(0.3, LLI)
47	Ag^{111}	10^{4}	(0.3, L)	6.9	(0.3, LLI)	91	(0.3, L)	6.3×10^{-3}	(0.3, LLI)
48	Cd^{109}+Ag^{109}s	1.9×10^{4}	(0.3, L)	81	(0.3, LLI)	17	(0.3, L)	7.4×10^{-3}	(0.3, LLI)
50	Sn^{113}	2.7×10^{4}	(0.3, B)	28	(0.3, LLI)	25	(0.3, B)	2.5×10^{-3}	(0.3, LLI)
52	Te^{127}	1.5×10^{4}	(0.3, K)	11	(0.3, LLI)	1.4	(0.3, K)	10^{-2}	(0.3, LLI)
52	Te^{129}	5.2×10^{3}	(0.3, K)	3.8	(0.3, LLI)	0.47	(0.3, K)	3.5×10^{-3}	(0.3, LLI)
53	I^{131}	0.58	(0.3, T)	1.8×10^{3}	(0.3, SI)	4.8×10^{-4}	(0.3, T)	0.16	(0.3, SI)
55	Cs^{137}+Ba^{137}s	1.1×10^{3}	(0.3, M)	1.4×10^{3}	(0.3, SI)	0.10	(0.3, M)	0.13	(0.3, SI)
56	Ba^{140}+La^{140}	25	(0.3, B)	4.4	(0.3, LLI)	2.3×10^{-2}	(0.3, B)	4.0×10^{-3}	(0.3, LLI)
57	La^{140}	7.0×10^{3}	(0.3, B)	4.1	(0.3, LLI)	6.4	(0.3, B)	3.7×10^{-3}	(0.3, LLI)
58	Ce^{144}+Pr^{144}	4.7×10^{3}	(0.3, B)	1.8	(0.3, LLI)	4.3	(0.3, B)	1.6×10^{-2}	(0.3, LLI)
59	Pr^{143}	4.6×10^{3}	(0.3, B)	8.1	(0.3, LLI)	4.2	(0.3, B)	7.4×10^{-3}	(0.3, LLI)
61	Pm^{147}	9.0×10^{3}	(0.3, B)	35	(0.3, LLI)	82	(0.3, B)	3.2×10^{-2}	(0.3, LLI)
62	Sm^{151}	4.1×10^{3}	(150, B)	1.2×10^{3}	(0.3, LLI)	3.7×10^{3}	(150, B)	0.11	(0.3, LLI)
63	Eu^{154}	2.6×10^{4}	(0.3, B)	6.9	(0.3, LLI)	24	(0.3, B)	6.3×10^{-3}	(0.3, LLI)
67	Ho^{166}	9.1×10^{4}	(0.3, B)	6.3	(0.3, LLI)	83	(0.3, B)	5.7×10^{-3}	(0.3, LLI)
69	Fm^{170}	9.5×10^{3}	(0.3, B)	7.5	(0.3, LLI)	8.6	(0.3, B)	6.8×10^{-3}	(0.3, LLI)
71	Lu^{177}	9.1×10^{4}	(0.3, B)	18	(0.3, LLI)	83	(0.3, B)	1.6×10^{-2}	(0.3, LLI)

See footnotes at end of table.

TABLE II.—MAXIMUM PERMISSIBLE OCCUPATIONAL EXPOSURE—Continued

Single Exposure Values for Ingestion of Radionuclides

Z (1)	Isotope (2) [2]	Ingestion of soluble radioactive material (μc initially available to be ingested during succeeding 8 hrs) [1] (3)	Ingestion of insoluble radioactive material (μc initially available to be ingested during succeeding 8 hrs) [1] (4)	Ingestion of soluble radioactive material (μc/cc in water ingested in 8 hours) [1] (5)	Ingestion of insoluble radioactive material (μc/cc in water ingested during 8 hrs) [1] (6)
73	Ta^{182}	1.8×10^{1} (0.3, L)	7.5 (0.3, LLI)	16 (0.3, L)	6.8×10^{-3} (0.3, LLI)
74	W^{181}	2.8×10^{1} (0.3, B)	10 (0.3, LLI)	25 (0.3, B)	9.1×10^{-3} (0.3, LLI)
75	Re^{183}	1.4×10^{2} (0.3, T) 7.3×10^{2} (0.3, sk)	31 (0.3, LLI)	1.3 (0.3, T) 6.6 (0.3, sk)	2.8×10^{-2} (0.3, LLI)
77	Ir^{190}	4.0×10^{2} (0.3, K) 8.0×10^{2} (0.3, s)	51 (0.3, LLI)	0.45 (0.3, K) 7.3 (0.3, s)	4.6×10^{-2} (0.3, LLI)
77	Ir^{192}	53 (0.3, K) 9.4×10^{2} (0.3, s)	7.5 (0.3, LLI)	4.8×10^{-2} (0.3, K) 0.85 (0.3, s)	6.8×10^{-3} (0.3, LLI)
78	Pt^{191}	1.4×10^{2} (0.3, K)	11 (0.3, LLI)	0.13 (0.3, K)	10^{-2} (0.3, LLI)
78	Pt^{193}	1.3×10^{3} (0.3, K)	13 (0.3, LLI)	0.12 (0.3, K)	1.2×10^{-2} (0.3, LLI)
79	Au^{198}	1.4×10^{1} (0.3, L) 1.3×10^{2} (0.3, K)	32 (0.3, LLI)	1.3 (0.3, L) 0.12 (0.3, K)	2.9×10^{-2} (0.3, LLI)
79	Au^{199}	8.1×10^{1} (0.3, L) 60 (0.3, K)	8.8 (0.3, LLI)	0.74 (0.3, L) 5.4×10^{-3} (0.3, K)	8.0×10^{-3} (0.3, LLI)
79	Au^{200}	1.9×10^{1} (0.3, L) 1.6×10^{2} (0.3, K)	25 (0.3, LLI)	1.7 (0.3, L) 0.15 (0.3, K)	2.3×10^{-2} (0.3, LLI)
81	Tl^{200}	4.4×10^{2} (0.3, M)	19 (0.3, LLI)	0.40 (0.3, M)	1.7×10^{-2} (0.3, LLI)
81	Tl^{201}	1.5×10^{3} (0.3, M)	1.4×10^{2} (0.3, LLI)	1.4 (0.3, M)	0.13 (0.3, LLI)
81	Tl^{202}	7.4×10^{2} (0.3, M)	75 (0.3, LLI)	0.67 (0.3, M)	6.8×10^{-2} (0.3, LLI)
81	Tl^{204}	5.2×10^{2} (0.3, M)	17 (0.3, LLI)	0.47 (0.3, M)	1.5×10^{-2} (0.3, LLI)
82	Pb^{203}	2.6×10^{3} (0.3, B)	29 (0.3, LLI)	2.4 (0.3, B)	2.6×10^{-2} (0.3, LLI)
82	Pb^{210}+dr	0.42 (0.3, B)	24 (0.3, LLI)	3.8×10^{-4} (0.3, B)	2.2×10^{-2} (0.3, LLI)
84	Po^{210}	4.0 (0.3, s)	4.0×10^{-2} (0.3, LLI)	3.6×10^{-3} (0.3, s)	3.6×10^{-2} (0.3, LLI)
85	At^{211}+dr	7.1×10^{-2} (0.3, T)	1.5 * (0.3, S)	6.5×10^{-5} (0.3, T)	1.4×10^{-2} (0.3, S)
88	Ra^{226}+55% dr	*0.54* (*150*, B)	*3.2×10^{-3} (0.3, LLI)*	4.9×10^{-4} (*150*, B)	2.9×10^{-2} (0.3, LLI)
89	Ac^{227}+dr	0.39 (0.3, B)	0.69 (0.3, LLI)	3.5×10^{-4} (0.3, B)	6.3×10^{-3} (0.3, LLI)
90	Th-nat	1.3 (150, B)	1.9×10^{-2} (0.3, LLI)	1.2×10^{-3} (150, B)	1.7×10^{-2} (0.3, LLI)
90	Th^{234}+Pa^{234}	3.9×10^{1} (0.3, B)	2.8 (0.3, LLI)	3.5 (0.3, B)	2.5×10^{-2} (0.3, LLI)
92	U-nat	15 (0.3, K)	3.0×10^{-2} (0.3, LLI)	1.4×10^{-3} (0.3, K)	2.7×10^{-2} (0.3, LLI)
92	U^{233}	2.2×10^{2} (0.3, B)	4.7×10^{-2} (0.3, LLI)	0.20 (0.3, B)	4.3×10^{-2} (0.3, LLI)
94	Pu^{239}	36 (150, B)	4.4×10^{-2} (0.3, LLI)	3.3×10^{-3} (150, B)	4.0×10^{-2} (0.3, LLI)
95	Am^{241}	2.1×10^{2} (0.3, B)	4.2×10^{-2} (0.3, LLI)	0.19 (0.3, B)	3.8×10^{-2} (0.3, LLI)
96	Cm^{242}	1.8×10^{2} (0.3, B)	3.8×10^{-2} (0.3, LLI)	0.16 (0.3, B)	3.5×10^{-2} (0.3, LLI)

[1] TB—total body, B—bone, F—fat, sk—skin, M—muscle, s—spleen, L—liver, K—kidney, Bl—blood, T—thyroid, S—stomach, SI—small intestine, ULI—upper large intestine, LLI—lower large intestine.

[2] The s in column 2 indicates daughter products that are isomers in an excited state.

NOTE.—The 0.3 or 150 in parentheses refers to the limiting dose rates of 0.3 rem/wk or 150 rem/70 yrs—whichever gives the smaller maximum permissible value. The letters given in parentheses indicate the critical body organ.

assume that the risk of damage from contaminated wounds is relatively unimportant. In most cases the inhalation of insoluble radioactive material gives lower maximum permissible values than inhalation of soluble radioactive material. Usually the lower large intestine is the most critical portion of the G.I. tract except where radionuclides of very short radioactive half-life are considered or where $f_1 \cong 1$.

In the cases where the G.I. tract is the critical organ (column 7 of Table I and columns 4 and 6 of Table II), it is assumed the material is swallowed (62 percent for inhalation and 100 percent for ingestion) and that it irradiates various portions of the G.I. tract in direct proportion to the time spent in each section. Some of the radioactive material passes through the wall of the small intestine determining a biological half-life. In case daughter products are produced, their contribution to the absorbed dose is included.

Figure 1 indicates the absorbed dose distribution in the G.I. tract for three cases, $Sr^{90}+Y^{90}$, $Ra^{228}+Ac^{228}$ and Pr^{144}. In the case of $Sr^{90}+Y^{90}$

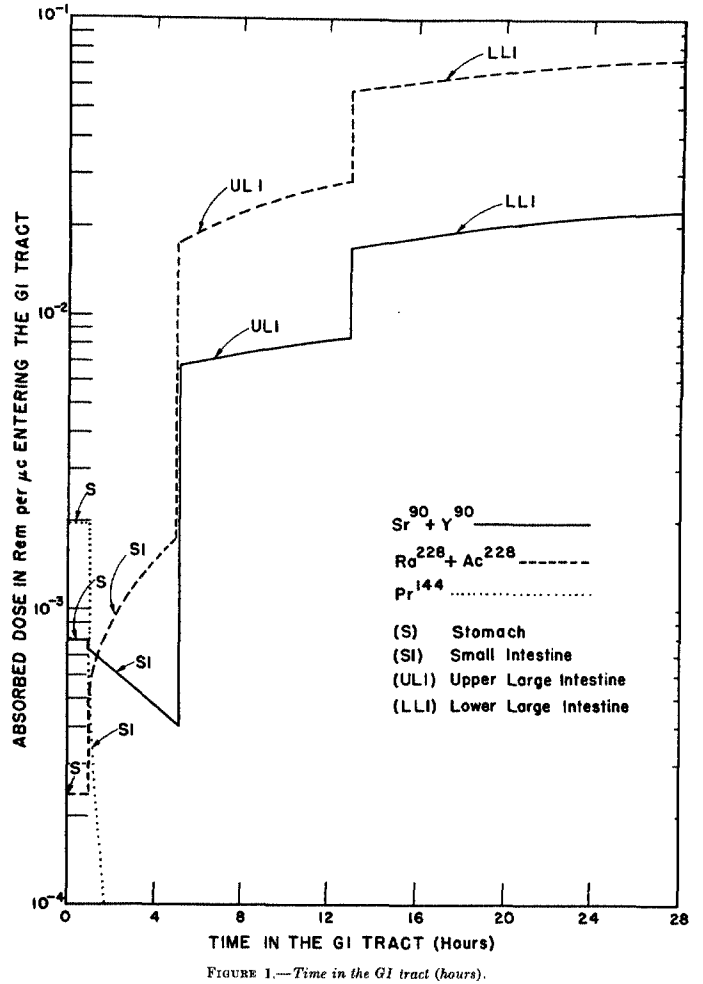

FIGURE 1.—*Time in the GI tract (hours).*

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the absorbed dose delivered to the stomach in 1 hour is small. The absorbed dose in the top portion of the small intestine is the same as that to the stomach, but decreases during the 4 hours there due to the 60 percent uptake into the bloodstream. The absorbed dose to the large intestine is much greater than that to the small intestine because the radioactive material spends 8 hours in the upper and 18 hours in the lower large intestine compared with only 4 hours in the small intestine. Also, the mass of material in the upper or lower large intestine is about one-eighth of that in the small intestine ($m_{ULI}=150$ g, $m_{LLI}=135$ g, $m_{SI}=1100$ g, $m_{stomach}=250$ g). In the case of Ra^{228} the 6-hour daughter, Ac^{228}, makes a large contribution to the dose. The rise in the dose in the small intestine is accentuated by the fact that the effective energy of Ac^{228} is 80 times that of the parent, Ra^{228}, and there is only 20 percent absorption from the small intestine into the blood. The slow rise in the absorbed dose in the upper and lower large intestines in the case of $Sr^{90}+Y^{90}$, and $Ra^{228}+Ac^{228}$ is the result of the growth of the 61-hour Y^{90}, and 6-hour Ac^{228} respectively. In the case of the 17.5-minute Pr^{144}, the absorbed dose is delivered mostly to the stomach. The dose to the large intestine is negligible because Pr^{144} passes through 17 radioactive half-lives in the stomach and small intestine.

The foregoing tables of MPC values may be useful in dealing with hazards associated with the fallout material from the testing of nuclear weapons as well as the contamination resulting from a laboratory spill or accident. However, many of the radionuclides of great interest that comprise fallout during the early periods following the detonation of an atomic weapon were not included in these tables. There are many factors that determine the type of fallout material from the detonation of a nuclear weapon, e. g., height of burst, distance from ground zero, type of weapon, weapon yield, meteorological conditions, etc. Likewise it has been found that there may be factors (physical, chemical, and biological) which tend to fractionate and concentrate certain of the radionuclides. For example, at the first Bikini underwater test I made a number of surveys on the target ships and nearby islands of the β/γ dose rate ratio and found it to range from 1 to several hundred. This high β/γ dose rate ratio was, in part, a consequence of the fact that on the average there are about twice as many beta particles as gamma rays emitted per disintegration of the U-fission mixture, and most of the beta particles have a range of less than a meter in air whereas a large fraction of the gamma photons have a range in air of many meters, i. e., the fraction of photons with an absorption coefficient λ that travel a distance greater than x is given approximately by the equation $(1-e^{-\lambda x})$. In addition, many common materials such as tar, resin, rust, paint, metals, etc., seemed to retain selectively certain of the beta-emitting radionuclides. Under certain circumstances this fractionation may be of considerable importance because overexposure to beta radiation can lead to serious erythema, burns, ulcers, and even death. Yet the most commonly used field survey equipment is designed to measure the absorbed dose from relatively hard gamma radiation and may give little or no response to beta radiation. Following the test of a thermonuclear weapon by the United States in the South Pacific in 1954, the more serious cases of radiation damage among the natives and operating personnel from the United States resulting from contact with the fallout materials were the consequence of exposure to beta radiations. It is sometimes stated that beta exposure is of little importance compared to the gamma dose from fallout material and that one would have to be partly naked or lie prone on the ground before the beta exposure should be a matter of concern.* I do not agree with this point of view and dare say some among the Marshallese, the Japanese fishermen, and the Americans who received painful and disfiguring beta burns as a consequence of exposure to fallout material in the South Pacific would not be inclined to underestimate the seriousness of exposure to beta radiation. In any case, the record should speak for itself- namely, the damage to man and animals (cattle, horses, deer, etc) that has been observed from the fallout material from nuclear tests to date has resulted not from exposure to hard gamma radiation but from exposure to beta radiation. In assessing the hazard from fallout, therefore, one must be cautious not to overlook the seriousness of exposure to beta radiation, and one should not rely on a theoretical estimate of the isotopic distribution or one should not reach final conclusions regarding the radiation hazard unless measurements have been made of the absorbed dose from β and soft γ radiation.

*The reader is referred to the final summary of this Conference by Dr. E. P. Cronkite.

Having called attention to the many factors which may change the isotopic distribution, I have risked setting up Table III which lists the more important U-fission radionuclides that would be present as a function of time following the detonation of a weapon if there were no fractionation. The radionuclides are listed in order of decreasing availability (assuming no selective deposition or separation of the radioelements) for 5 time intervals—1 hour to 1 day, 1 day to 1 week, 1 week to 1 month, 1 month to

TABLE III.—AVAILABILITY OF U-FISSION RADIONUCLIDES

(Listed in order of decreasing yield)

1 hour to 1 day		1 day to 1 week		1 week to 1 month		1 month to 1 year		1 year to 70 years	
Radionuclide	Yield	Radionuclide	Yield	Radionuclide	Yield	Radionuclide	Yield	Radionuclide	Yield
Y^{92}	559	Mo^{99}	360	La^{140}	337	Nb^{95}	585	Sr^{90}	430
La^{141}	519	Ce^{143}	360	Ba^{140}	293	Zr^{95}	433	Y^{90}	430
I^{135}	458	Nb^{97}	250	Pr^{143}	284	Y^{91}	374	Cs^{137}	406
Y^{93}	454	I^{135}	241	Ce^{141}	216	Sr^{89}	311	Ba^{137}	406
Sr^{92}	451	I^{132}	237	I^{131}	160	Ce^{141}	284	Pm^{147}	215
Pr^{146}	444	Zr^{97}	229	Nd^{147}	128	Ce^{144}	271	Ce^{144}	214
Ba^{139}	427	Te^{132}	228	Zr^{95}	127	Pr^{144}	271	Pr^{144}	214
Ir^{194}	423	Ba^{140}	160	Y^{91}	122	Ru^{103}	197	Sm^{151}	44
Sr^{91}	414	La^{140}	128	Sr^{89}	109	Rh^{103}	189	Nb^{95}	31
Cb^{97}	387	I^{131}	122	Mo^{99}	102	Pr^{143}	154	Ru^{106}	27
La^{142}	376	Pr^{143}	111	Ru^{103}	96	La^{140}	150	Rh^{106}	27
Zr^{97}	368	Y^{93}	110	Rh^{103}	93	Ba^{140}	135	Zr^{95}	14
Ce^{145}	351	Sr^{91}	96	I^{132}	87	Pm^{147}	40	Y^{91}	9
Rb^{86}	314	Pm^{149}	92	Te^{132}	79	Nd^{147}	39	Sr^{89}	4
Cs^{138}	294	Nd^{147}	77	Cb^{95}	41	I^{131}	34		
I^{133}	250	Rh^{105}	70	Ce^{144}	28	Ru^{106}	22		
Te^{133}	233	I^{135}	48	Pr^{144}	28	Rh^{106}	22		
Ce^{143}	222	Ce^{141}	40	Ce^{143}	14	Sr^{90}	13		
Y^{91}	206	Zr^{95}	39	Pm^{149}	12	Y^{90}	13		
Pr^{146}	164	Y^{91}	37			Cs^{137}	10		
Y^{95}	163	Y^{92}	37			Ba^{137}	10		
Te^{134}	161	Sr^{89}	35						
Mo^{99}	126	Ru^{103}	32						
Y^{94}	72	Rh^{103}	31						
Te^{132}	71	Pr^{145}	17						
Ba^{141}	70								
I^{132}	67								
La^{143}	61								
Mo^{101}	37								
Rb^{89}	34								
Te^{131}	29								

TABLE IV.—RELATIVE HAZARD AFTER DETONATION**

1 hour to 1 day				1 day to 1 week				1 week to 1 month				1 month to 1 year				1 year to 70 years			
Radionuclide	Critical organ δ	Lungs	GI tract	Radionuclide	Critical organ	Lungs	GI tract	Radionuclide	Critical organ	Lungs	GI tract	Radionuclide	Critical organ	Lungs	GI tract	Radionuclide	Critical organ	Lungs	GI tract
Y^{92}				Mo^{99}	3.9×10^{-4}	13	5.0	La^{140}	4.8	22	51	Nb^{95}	3.4	42	13	Sr^{89}	96	61	20
La^{140}				Ce^{141}				Ba^{140}	24	68	42	Zr^{95}	3.9	69	27	Y^{91}			
I^{131}				Nb^{95}				Pr^{143}	3.9	11	22	Y^{91}	23	29	57	Cs^{137}	2.9	24	1.8
Y^{91}				I^{131}				Ce^{141}	2.3×10^{3}	7.3	.55	Sr^{89}	31	24	18	Ba^{131}			
Sr^{89}				Zr^{97}				I^{131}				Ce^{144}	29	54	93	Pm^{147}	1.1	2.2	3.8
Pr^{143}				Te^{132}				Nd^{147}	1.2	17	7.9	Pr^{144}				Ce^{144}	23	43	74
Ba^{140}				Ba^{140}	18	37	23	Zr^{95}	7.6	9.4	18	Ru^{103}				Sm^{151}	.18	.13	.23
Ir^{194}				La^{140}	1.8	8.5	19	Y^{91}	11	8.4	6.4	Rh^{103}				Nb^{91}	.18	2.2	.67
Sr^{91}				I^{131}	170	5.5	.42	Sr^{89}	1.1×10^{-2}	3.6	1.4	Pr^{143}	2.1	5.7	12	Ru^{106}	1.4	5.8	10
Cs^{137}				Pr^{143}	1.5	4.1	8.5	Mo^{99}				La^{140}	2.1	10	23	Rh^{106}			
La^{142}				Ru^{103}				Ru^{103}				Ba^{140}	15	31	19	Zr^{95}		1.9	.85
Zr^{97}				Rh^{103}				Rh^{103}				Pm^{147}	20	.40	72	Y^{91}	.12	.69	1.3
Cs^{138}				I^{132}				Te^{132}		2.9	.80	Nd^{147}		1.5	.12	Sr^{89}	.42	.32	.25
Cs^{139}	2.2	29	2.4	Cb^{95}	.24	5.6	9.7	I^{131}	48	4.7	8.2								
Pm^{149}				Ce^{144}	3.0			Ru^{106}	1.1										
I^{131}				Pr^{144}				Rh^{106}	2.6	1.8	.62								
Te^{132}				Ce^{141}				Sr^{89}											
Cs^{141}				Pm^{149}	.35	5.3	2.4	Y^{91}	1.8										
Y^{91}	13	16	31	Zr^{97}	2.3	2.9	5.6	Cs^{137}	.069	81	.044								
Pr^{144}				Y^{93}				Ba^{131}											
Y^{93}				Te^{132}	3.5	2.7	2.0												
Sr^{89}				Sr^{89}															
Ru^{103}				Ru^{103}															
Mo^{99}	1.4×10^{-4}	4.5	1.8	Rh^{103}															
Y^{93}				Pr^{144}															
Te^{132}																			
Ba^{131}																			
I^{132}																			
La^{140}																			
Mo^{99}																			
Rh^{106}																			
Te^{131}																			

*In these cases the values of the relative hazard are based on the assumption that the parent radionuclide is taken into the body and the daughter grows while the parent is contained in the critical body organ contributing its proportionate share of the dose during the indicated time intervals.

**The many voids in this Table indicate where essential data are missing and impress on one the desirability of conducting additional experiments

δ Critical organ as listed in column 3 of table I.

1 year, and 1 year to 70 years. These values were obtained by summing the integration of the rate of radioactive decay of each radionuclide over each of the 5 time intervals. Data of Hunter and Ballou [6] were used in making these calculations.

Table IV lists the relative hazards* in the 5 time intervals. The relative hazards were obtained by dividing values of availability given in Table III by the single exposure MPC values given in Tables I and II. Many voids appear in Table IV, especially in the case of the 1 hour to 1 day interval because the MPC values are not available for many short-lived radionuclides. The transuranic nuclides or the induced radioisotopes are not included in these tables of relative availability or relative hazard because their availability depends upon the type and efficiency of the weapon and the elevation of the weapon at the time of detonation. However, their contribution to the possible hazards of fallout must not be overlooked.

Summarizing the data available in Table IV, the radionuclides presenting the major hazard in each interval may be listed in order of decreasing hazard as follows:

1 hour to 1 day - (insufficient data)
1 day to 1 week - I^{131}, Ba^{140}, La^{140}, Mo^{99}
1 week to 1 month—I^{131}, Ba^{140}, La^{140}, Pr^{143}, Y^{91}, Zr^{95}, Sr^{89}, Ce^{144}
1 month to 1 year—Ce^{144}, Zr^{95}, Y^{91}, I^{131}, Nb^{95}, Ba^{140}, Sr^{89}
1 year to 70 years Sr^{90}, Ce^{144}, Cs^{137}, Ru^{106}

DISCUSSION

Karl Z. Morgan

Dr. LANGHAM. I would like to give just a little opportunity for questions to Dr. Morgan, primarily because I have been at two of his presentations recently, and every time I found that there were questions to be asked, that we never did really get to him. I would like to start the questioning by asking, Dr. Morgan, what do you really feel is the practical significance that you intend to place on these maximum permissible values calculated on the basis of the gastrointestinal tract? Do you really feel these numbers which, as I understand it, will lower air in water maximum permissible values, will be pushed as being the accepted value over those calculated from other critical organs?

Dr. MORGAN. Dr. Langham, I should be asking you or Dr. Henshaw or one of the rest of this group this question, rather than you asking me. The British on the International Commission from the start I think felt rather strongly that we should not make any distinction between the permissible dose to the gastrointestinal tract and to other body organs. This matter was discussed somewhat in our committee in the early part of the week, and I gathered from the discussion there that the consensus of opinion is again that we should not make any distinction with one exception. In the case of alpha emitters, I believe the National Committee feels rather strongly that the alphas would not reach the mytotic layer in the lower large intestine, and therefore would not constitute a hazard. So with that one exception, it is very likely in the revised handbook 52 that the gastrointestinal tract will appear as one of the critical organs in many cases, with the exception of the alpha emitters.

As to whether or not this is a safe assumption, I would rather for you or Dr. Henshaw or someone else to answer.

Dr. LANGHAM. Thank you. Do we have any other questions on this subject?

Dr. HOLLAND (AEC). I would like to ask whether there is any plan to incorporate in these values the variation in radiosensitivity of the various organs and tissues, that is, to set up maximum permissible doses on a sort of an organ by organ basis.

Dr. MORGAN. That has been considered. At present we make a distinction in the case of one organ only, that is for the thyroid. We permit 600 rems per week, rather than 300

[6] HUNTER, H. F. and N. E. BALLOU, "Simultaneous Slow Neutron Fission of U^{235} Atoms. I. Individual and Total Rates of Decay of the Fission Products," Report ADC-65, April 11, 1949.

*The relative hazards listed in Table IV do not take into account the concentration of certain radionuclides in the food cycle, e. g. the concentration of iodine and strontium in milk. Such factors may increase the relative hazard from certain radionuclides.

rems per week. The External Dose Committee makes several exceptions, especially in the case of the skin. There are many other variations perhaps that are even more important. For example, we should have values for the various chemical forms of the radionuclides. So there is a lot of work yet to be done and we will be happy when we finish the present set of values for about 150 new radionuclides that we are including in the revised handbook.

The answer is "No," in the forthcoming revision of the Handbook we do not plan to make any distinction between the various organs with the exception of the thyroid. MPC values are based on two principal criteria: 1), 0.3 rems per week to the organ, or 2) in the case of bone seekers, an amount that will give a dose corresponding to that received from 0.1 microgram of radium deposited in the bone.

Dr. LANGHAM. I think it is pretty obvious that part of the difficulty on this particular subject centers around the fact that the experimental biologist and his experiments can not keep up with Dr. Morgan and his pencil. One more question.

Capt. BENNETT (BuShips Navy). We are currently considering adapting our meters to measure the external hazard to a considerable extent by not a beta-gamma ratio, but a ratio of shallow dosage to deep dosage which will be based on a mean depth of the mytotic layer of the skin. I would like to ask Dr. Morgan whether such a meter would be properly used for contamination hazard determination in view of varying depths of the dangerous areas in the internal organ?

Dr. MORGAN. I think that some compromises have to be reached in designing instruments to measure the damage from beta emitters. I think the answer to your question is yes, that such an instrument would be very valuable. We are doing essentially the same thing in the revision of our film badge, so that we will have one very thin window that will give us readings that will correspond very closely to the dose delivered at a depth of about 10 mg/cm^2 tissue equivalent. I think in any monitoring system one should have a device that will indicate the exposure from the beta radiation. I don't believe this has much direct relationship to the beta dose.

I was not quite sure of the implication here relative to the internal emitters. Along with the external monitoring system, one has to monitor the urine and try to determine what the internal dose is. I am not sure that I got your question.

Capt. BENNETT. We consider the contamination meter as the device which measures the probable hazard from internal dosage, and we wondered whether the standards that we are setting for the external meter would be equally applicable for a contamination meter.

Dr. MORGAN. I want to look into the detailed standards before I could answer that question. Perhaps we could get together.

UPTAKE OF IODINE-131 IN HUMAN AND BOVINE THYROIDS FOLLOWING DETONATION OF NUCLEAR WEAPONS

By MARGARET R. WHITE and HARDIN B. JONES

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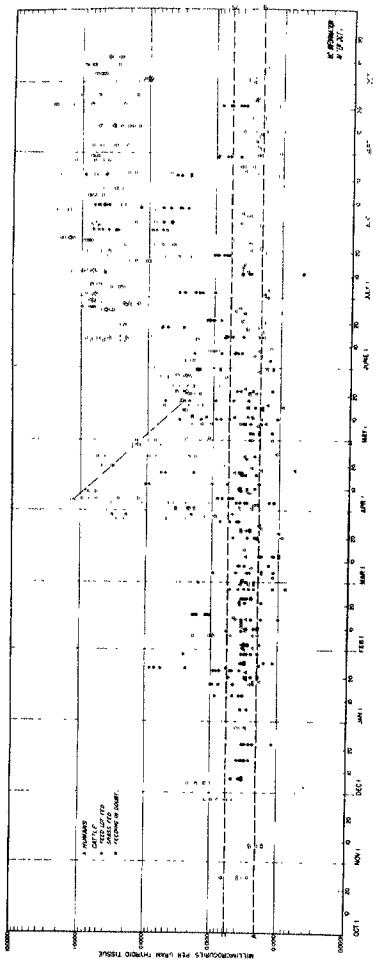
Beginning with the finding of measurable uptake of radioactive iodine (I^{131}) in thyroid tissue in the periods following nuclear explosions [Van Middlesworth 1, 2], Donner Laboratory has maintained a routine assay of I^{131} content of beef thyroids obtained from local slaughter houses and more recently of human thyroids which could be obtained on autopsy in the San Francisco area. The I^{131} concentrations of beef thyroids were reported for 1955 Northern California and Western cattle (U. C. R. L. Report 3355. March 1956). This report confirmed the Van Middlesworth observation and established the maximum uptake between March and September 1955 as 6.4 millimicrocuries of I^{131} per gram of beef thyroid and a total integrated maximum dose to the thyroid of cattle of about 1 rep for the Spring and Summer of 1955. This activity apparently was the result of the several detonations at the Nevada testing site. After the last test in mid-May 1955, the maximum activity in beef thyroids declined following closely the natural half-life of I^{131}. Upon this evidence and the additional evidence of a prompt rise in thyroid activity following nuclear explosion, it may be concluded that beef thyroids are in rapid equilibrium with iodine fallout. Van Middlesworth has recently shown that human thyroid I^{131} concentration roughly parallels bovine thyroid in radioiodine content, and that the human thyroid concentration is less than approximately 1/250th of the cattle thyroid concentration. The times of maximum iodine concentration in human or cattle thyroids coincided.

This report includes 151 human thyroids and 1,000 beef thyroids assayed for I^{131} content October 1955 to October 1956. Two periods of slight I^{131} content are recorded in December 1955 and January 1956 respectively, and two periods of concentration of I^{131} approaching or exceeding 1 millimicrocurie per gram of beef thyroid which appeared in March and May 1956. The activity which appeared in March died away in detectable concentration in newly obtained beef thyroids with a half-decline period of 8 days. The radioactivity which began in May 1956 maintained a value of 1 to 2.6 millimicrocuries per gram of beef thyroid during the entire period, June to October 1956 in spite of isotopic decay. Presumedly this reflected multiple additions to the atmospheric level of I^{131}. At all times of increased I^{131} levels, some cattle appeared to have low concentrations of I^{131}. These cattle were usually described as feed-lot-fed (see Table below). Range-fed cattle during all periods of collection of samples had the greatest concentration of I^{131}. Range-fed and feed-lot-fed cattle differ by a factor of 50 to 100 in usual concentration. Some feed-lot animals appear to have appreciable concentrations of thyroid I^{131}, but the simplest assumption to explain this inconsistent uptake is the lack of reliability of information on feeding procedures preceding marketing of beef. Additionally there is the problem of difficult evaluation of



Plot of radioactivity per gram of thyroid tissue on a logarithmic scale as a function of the date of death of cattle and humans. The radioactivity is expressed as millimicrocuries of I^{131}. The dashed horizontal line, A–B, is the average radioactivity of glass vials and a representative sample of muscle. This radioactivity includes potassium and other long-lived radioactive materials. The line C–D is placed three standard deviations above the mean background radioactivity (line A–B). Thus all radioactivity in cattle thyroids above line C–D is statistically significant with respect to fallout radioactivity.

The plotted values of human thyroid radioactivity include the radioactivity of the glass vial, even though the latter accounts for most of the activity observed. Only the scintillation counter background has been subtracted. Since none of the points lie above the line C–D, the actual human burden of I^{131} was not significant.

lapse of time between last range feeding and slaughter. Thus, much of the variance in range-fed animals may be iodine decay during the pre-slaughter holding in stockyards. The cattle thyroid irradiation for the Russian explosion March 1956 was an accumulated exposure of 0.2 rep (March to May 1956).

An integral dose for the Bikini tests is estimated as including time from May to October 1956. During this period the maximum activities corresponded to 0.010 to 0.028 rep per day. The average radiation to all range-fed cattle is close to 0.01 rep per day for this entire period. Thus the maximum exposure of bovine thyroid was (June 6, 1956 to October 11, 1956) approximately *1.3 r*.

Human thyroids at all times of collection were in the range of 1/1000th the level of range-fed beef thyroid I^{131} content. During times when I^{131} content was not detectable in either humans or cattle, the thyroid gland tissue from beef or human contained similar levels of natural radioactivity. Additional beef thyroid radioactivity was identified as I^{131} by following the decay of radioactivity which uniformly gave an 8-day half-life to the radioactivity above the natural background.

Human and beef thyroid were measured for radioactivity both initially and after 4 or more half-lives of radioiodine (lapse of at least 28 days). Even though any one of the human thyroids contained too little I^{131} to be detected, it was possible to make a finer estimate of the I^{131} content in human thyroids, by combining all the human measurements according to 2 time periods: One of low cattle I^{131} content (January 1956 to March 4, 1956) and the other of the period (June to October) when range cattle thyroids were measurable as having 2 to 3 thousand counts per minute per total countable sample. By counting the human thyroid specimens initially and after decay, the following comparison fails to detect I^{131} in human thyroids and establishes that the average human thyroid concentration of I^{131} is probably less than 1/1000th the maximum level observed in thyroids from range-fed cattle.

June to October 1956

Bovine thyroids—

Thyroids having more than 1 mμc/gm:

Number	Average counts per sample	I^{131} mμc/gm	Average weight sample	Average weight whole thyroid
18	3532	1.51	4.3 gm	27 gm
1 (highest measured concentration).	6235	2.67	4.3 gm	----
Median level of thyroid I^{131} content in range-fed animals				
132	1181	0.409	4.3 gm	27 gm
Median level of thyroid I^{131} content in feed lot-fed animals				
52	28.9	0.008	4.3 gm	27 gm

Human thyroids—

Number	Average counts per sample	Average weight sample	Average weight whole thyroid
68 initial count	6.21±0.83	4.3 gm	16 gm
68 recount	5.18±0.76		
difference. (difference not significant)	1.03±1.14		

Samples were counted an average of 3.8 days after death, therefore if human thyroids had 1.03 counts of I^{131} per sample the average I^{131} burden of human thyroids would be 0.0006 millimicrocuries per gram.

Radioiodine levels in human thyroids either individually or as a group-measure were not significantly established in the same time period when cattle I^{131} content in thyroid tissue ranged up to 2.7 millimicrocuries per gram thyroid.

It is interesting to note that one human thyroid had appreciable I^{131} content comparable with beef thyroid. It measured 0.025 millimicrocuries per gram, which is about 1/20th of the bovine I^{131} concentration, during this time period. This man, upon investigation through the attending physician, was found to have been given a tracer dose (2.5 μc I^{131}) 67 days preceding death, or approximately 8 half-lives of I^{131} earlier. Thus the observed level is entirely within the expected value for this length of decay of I^{131}.

It is possible that human thyroids do not

take up I^{131} from fallout even in the order of magnitude directly measured which is 1/1000th of the level measured in bovine thyroids for the same period. However, statistical confidence is established at two defendable limits of possible I^{131} uptake by human thyroid tissue. Thus:

(*a*) The median value of range-fed cattle corrected to estimated day the cattle left the range is 0.6 millimicrocuries per gram thyroid.

(*b*) The observed mean human thyroid I^{131} content as of day of death is 0.0006 millimicrocuries per gram thyroid.

(*c*) Human I^{131} levels in thyroid *could have been* established at 0.0021 millimicrocuries per gram thyroid with a certainty of $P=0.01$.

(*d*) There is only 1 chance in 1,000 that human values could have exceeded 0.0026 millimicrocuries per gram thyroid during the observation period.

Thus it is probable that human thyroid contains less than 0.43 percent or 1/230th of the I^{131} burden in cattle thyroid following atomic detonation.

Therefore, we can accept that irradiation of thyroid from I^{131} content in man in the high I^{131} fallout period of June to October 1956 is less than

$$\frac{1.3 \text{ rep (median thyroid irradiation, range cattle June—Oct. 1950)}}{230 \text{ (confidence value for ratio of cattle to human)}} = 0.0056/\text{rep}$$

However, the probable value is estimated as

$$1.3 \text{ rep} \times \frac{1.03}{1181} = 0.001 \text{ rep}$$

for accumulated irradiation exposure of human thyroid tissue by contained I^{131} fallout.

ADDENDUM

Data collected in 1957, when *continental U. S. tests* were being conducted, shows that during the period from May 20 to July 31 statistically significant amounts of radioactivity were found in human thyroids. During this period the average value for 41 human thyroids obtained in the San Francisco area was 0.0014 millimicrocuries per gram of wet tissue while that for 87 range cattle thyroids was 0.63 millimicrocuries per gram of wet tissue. Therefore the ratio of human thyroid radioactivity to cattle thyroid radioactivity was 1/460 as compared to a ratio of less than 1/1000 during the Bikini tests of 1956. This indicates different relative uptakes of I^{131} between humans and cattle in the two periods. The highest human value was 0.0056 millimicrocuries per gram while the highest cattle value was 4.0 millimicrocuries per gram, a value approximately twice the maximum observed in cattle during the period reported above. The present concentration of I^{131} in human thyroids would still be significantly elevated at one-half the observed counting rate.

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2. Lester Van Middlesworth. *Science,* 123, 982 (1956).

DISCUSSION

Hardin B. Jones

Dr. Lindberg (UCLA). Were those cattle from the bay area also?

Dr. Jones. These cattle were from northern California.

Dr. Lindberg. But from the west.

Dr. Jones. Yes.

Dr. Lindberg. We are going to present some data this afternoon regarding the occurrence of iodine near the test site or more specifically, near the fallout pattern, which would suggest the values presented are very conservative in a short period of time anyway.

THE EXCRETION OF RADIOACTIVE FISSION FRAGMENTS BY MAN DURING CONTINENTAL AND OVERSEAS WEAPONS TESTS

By Ariel G. Schrodt, James B. Hartgering and Kent T. Woodward

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The excretion of iodine-131 and strontium-90 has been measured, during Operation Teapot, in human urine specimens collected on a routine basis at selected stations throughout the United States and in foreign countries. A complete account of the work referred to herein is given in "Recovery of Radioactive Iodine and Strontium from Human Urine—Operation Teapot", Walter Reed Army Institute of Research Document 00–55 (AFSWP–893) by J. B. Hartgering, Ariel G. Schrodt *et al.*

The excretion of several of the principal fission products is being measured during Operation Redwing. Data will be available in a forthcoming report.

Details of the chemical separation procedures and the low-level counting techniques used may be found in the AFSWP–893 Document.

The program was set up to obtain 24-hour urine specimens from 10 individuals at each of a number of stations throughout the United States and overseas. The selected continental stations (Table I) are shown on the map (fig. 1).

The data from the widely scattered overseas stations will not be presented here, but are available in AFSWP–893.

In Figure 2, the average activity of iodine-131 per group of 24-hour urine specimens is plotted versus the collection time. Along the lower abscissa are indicated the dates which correspond with the collection week numbers of the upper graph.

The x's scattered below the upper graph in Figure 2 indicate the passage of clouds at the altitude shown in relation to the shot times. No outstanding correlation is seen.

Table I.—CONTINENTAL COLLECTION STATIONS

A. Letterman Army Hospital, San Francisco, Calif.
B. Fitzsimmons Army Hospital, Denver, Colo.
C. Brooke Army Hospital, San Antonio, Tex.
D. Walter Reed Army Hospital, Washington, D. C.
E. Fairchild Air Force Base, Spokane, Wash.
H. March Air Force Base, Riverside, Calif.
J. Nellis Air Force Base, Las Vegas, Nev.
K. Luke Air Force Base, Phoenix, Ariz.
L. Lockbourne Air Force Base, Columbus, Ohio
M. Tinker Air Force Base, Oklahoma City, Okla.
N. Hill Air Force Base, Ogden, Utah
O. Scott Air Force Base, Bellville, Ill.
P. Selfridge Air Force Base, Mount Clemens, Mich.
S. Donaldson Air Force Base, Greenville, S. C.
T. MacDill Air Force Base, Tampa, Fla.
W. Westover Air Force Base, Chicopee Falls, Mass.
X. Camp Mercury Air Force Base, Nev.

The lower graph in Figure 2 shows the gummed paper data supplied by Eisenbud's group. There is not the good correlation between the biological data and the physical determinations that we had hoped might obtain.

Camp Mercury, Nevada (fig. 3) is of especial interest because of its proximity to the test site. The figure is self-explanatory.

Figure 4 presents the data from Oklahoma City. Unfortunately our collection program ceased just at the time the iodine-131 excretion reached its highest point, so we were unable to record any subsequent data.

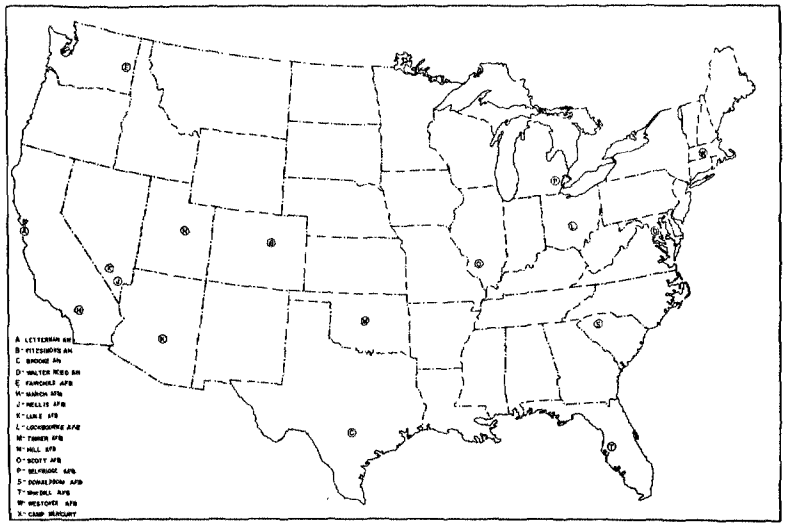

FIGURE 1.—*Map of U. S. collection stations.*

The data presented in the upper graph of Figure 5 is from Ogden, Utah, while the lower graph is from the nearest gummed paper station, Salt Lake City. There appears to be rather good correlation in this instance between the passage of clouds and the excreted iodine-131 activity.

The data obtained from the Denver samples (fig. 6) are particularly interesting from the third through the seventh week. The diminishing level of excreted activity follows closely the physical decay curve of iodine-131 over that period. There was no precipitation and no additional cloud passage over that area during the 5-week period. Individuals varied greatly, but the average of 10 specimens yields the smooth decay curve.

Table II shows the average values obtained for 24-hour specimens at several locations for the entire test period, February 22 to May 24, 1955. From this data one could roughly estimate the thyroid burden.

TABLE II.—AVERAGE IODINE-131 ACTIVITY IN 24-HOUR URINE SPECIMENS COLLECTED FEBRUARY 22 TO MAY 24, 1955

Ogden, Utah	134 dpm/24 hr spec
Camp Mercury, Nev.	98 dpm/24 hr spec
Belleville, Ill.	74 dpm/24 hr spec
Denver, Colo.	69 dpm/24 hr spec
Oklahoma City, Okla.	52 dpm/24 hr spec
Washington, D. C.	20 dpm/24 hr spec
Chicopee Falls, Mass.	12 dpm/24 hr spec
San Francisco, Calif.	11 dpm/24 hr spec

Some thyroid autopsy specimens were assayed during the test period, but most of these were so long removed from the biosphere that no significant activity was observed.

The maximum amount of iodine found in any individual 24-hour specimen was 774 disintegrations per minute. We found iodine-133

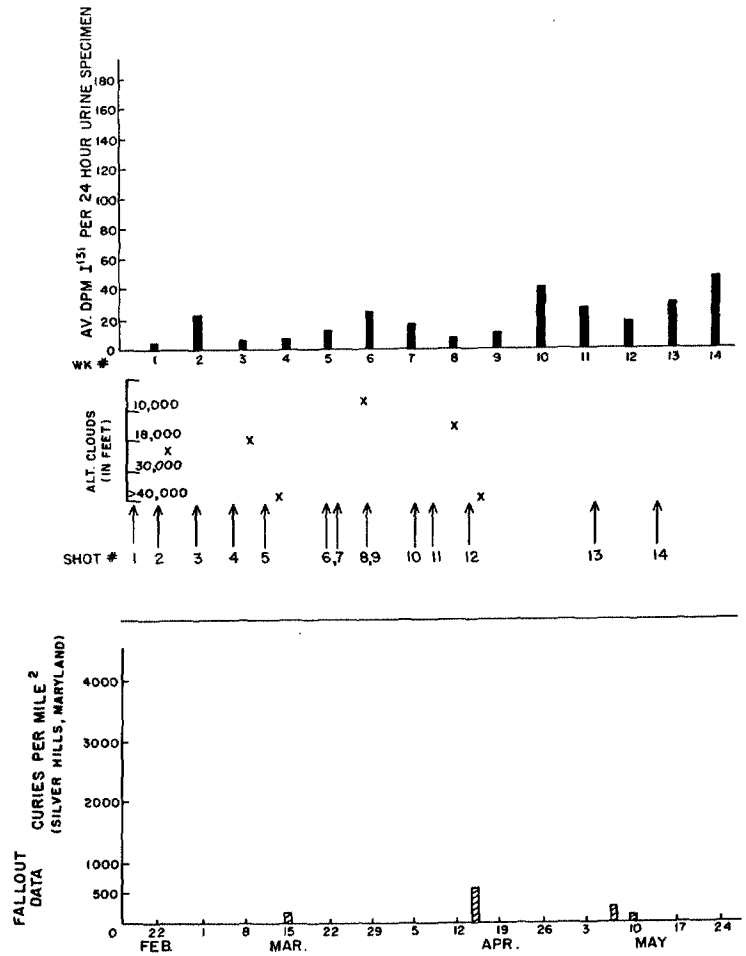

FIGURE 2.—*Fallout data—Walter Reed Army Medical Center.*

with its 22-hour half-life in some of the samples. This suggests that an appreciable amount of the radioiodine enters the body through inhalation rather than proceeding through the food cycle.

In looking for strontium-90 in these samples, we found a maximum value for any one sample of 2.3 disintegrations per minute. However, the average value of strontium-90 in the specimens collected prior to the test period was 0.09 disintegrations per minute per 24-hour urine specimen.

Figure 7 shows an attempt to correlate the iodine-131 with the strontium-90 activity in the samples. The iodine activity here was corrected to shot time rather than to collection time as is the case for the other graphs.

The strontium data could only be obtained by pooling many individual specimens. The yttrium-90 daughter separation and counting procedures used were identical with those of the Chicago Sunshine Laboratory.

In conclusion we can report that strontium-90 activity measurably increased during the interval between Operations Teapot and Redwing. This increase is in part due to foreign weapon tests. In the United States, the average level of iodine-131 excretion during Operation Redwing is not markedly different from the level observed during Teapot. Specific data from the Redwing period will be available in a forthcoming report.

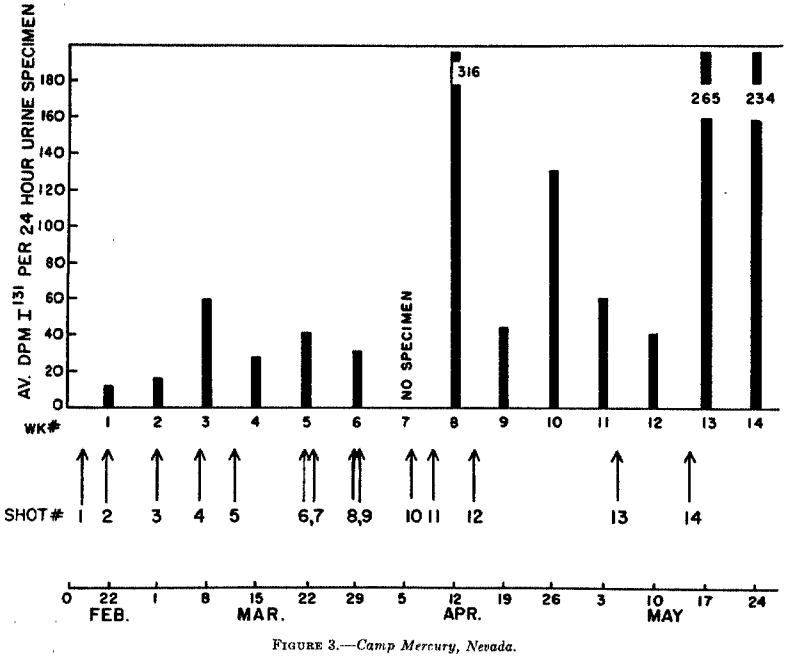

FIGURE 3.—*Camp Mercury, Nevada.*

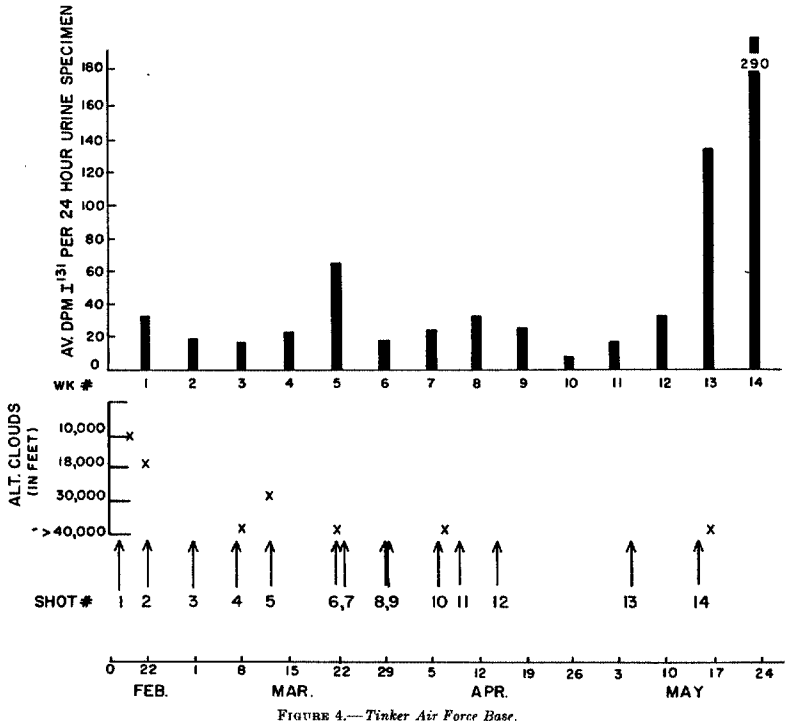

FIGURE 4.—*Tinker Air Force Base.*

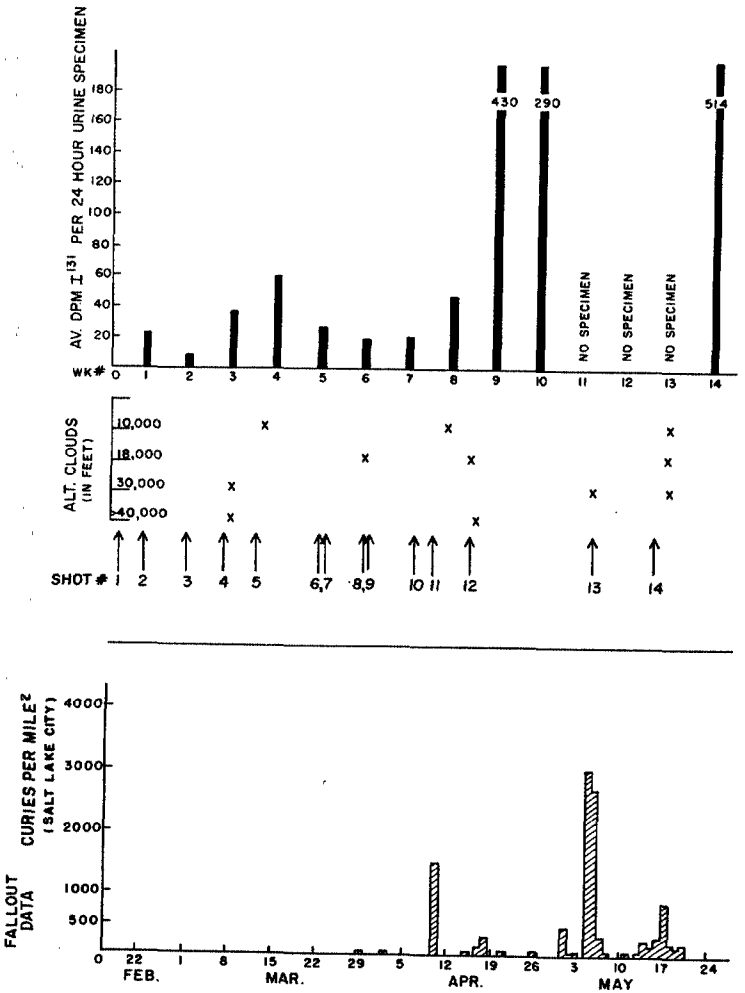

FIGURE 5.—*Fallout data—Hill Air Force Base.*

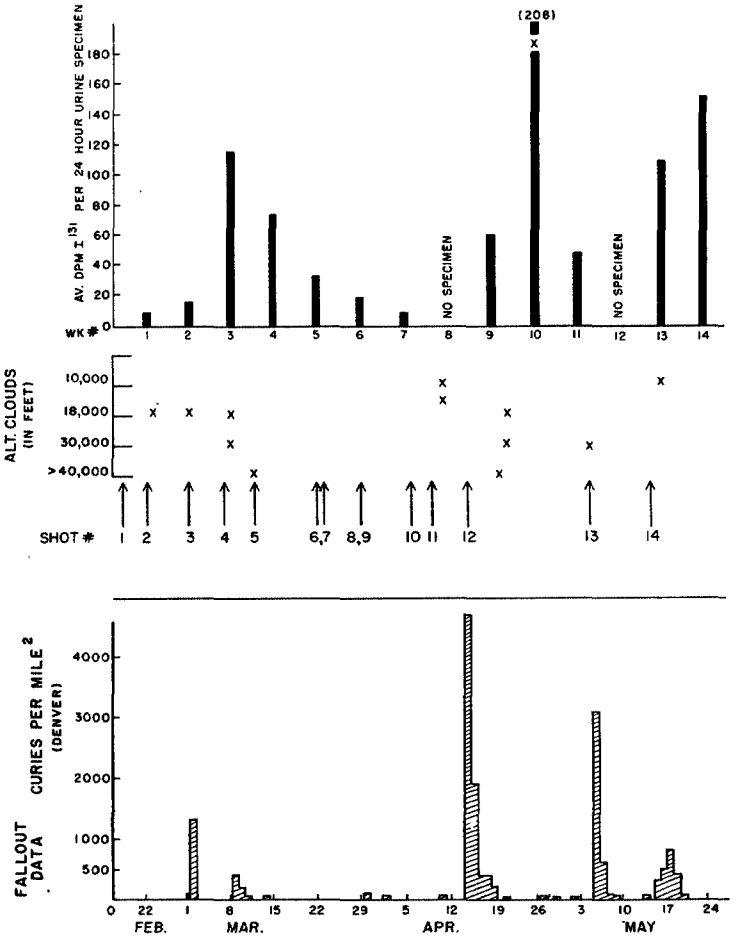

FIGURE 6.—*Fallout data—Fitzsimons Army Hospital.*

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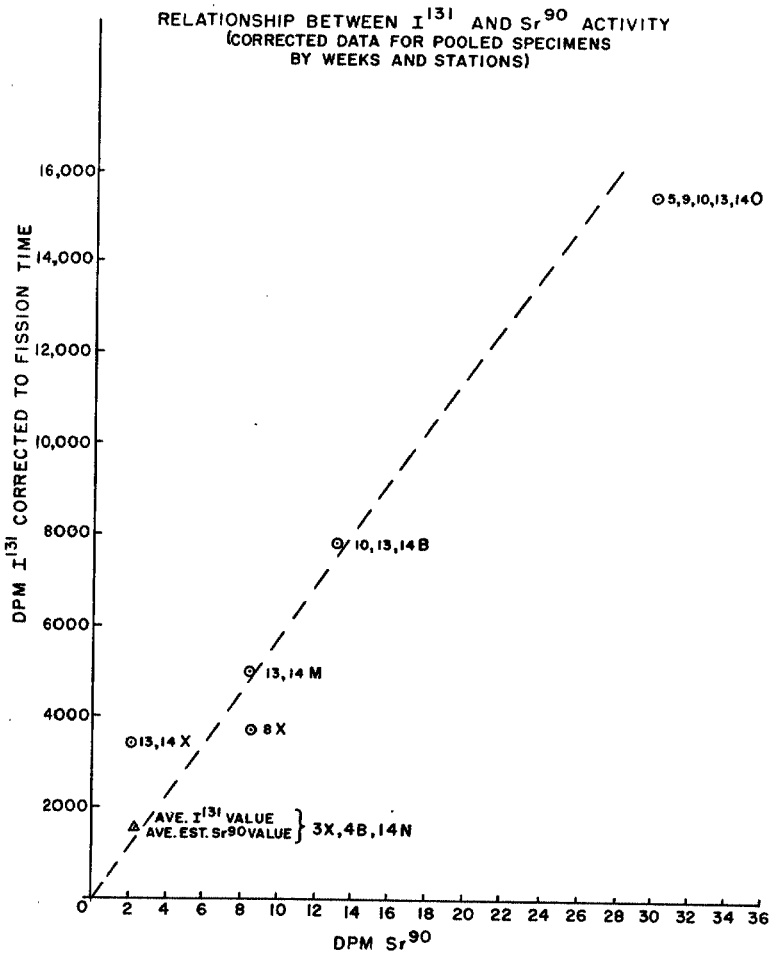

FIGURE 7.—*Relationship between I 131 and Sr 90 activity.*

METABOLIC STUDIES WITH STRONTIUM-90 IN THE RHESUS MONKEY

(Preliminary Report)

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ABSTRACT

Adult rhesus monkeys eliminated 56 percent of administered Sr^{90}, compared with 28 percent of Ca^{45}, in the urine during the first 10 days after intravenous administration.

The Sr^{90} concentration in the vertebrae was found to be reasonably representative of the skeleton as a whole in two animals whose skeletal distribution of Sr^{90} was studied. Successive amputation of caudal vertebrae is therefore recommended as the simplest and safest method of acquiring information on long-term skeletal retention of Sr^{90} in valuable animals with long life spans.

Average half times for skeletal retention of Sr^{90} were calculated for an adult male, 470 days, and for an adult female that had experienced 3 closely spaced pregnancies, 315 days.

Half times for skeletal retention of Sr^{90} of 155 and 195 days were calculated for the first 10 months of life of two offspring born to an injected mother.

One infant monkey retained an average of 18 percent of Sr^{90} administered daily by mouth for 13 weeks, whereas 6 adolescents retained on the average less than 5 percent of a daily dose during the same period of time.

A measurable amount of Sr^{90}, 23.5 dpm/g bone ash, was found in the skeleton of an uninjected control animal.

Placental transfer from a mother with a fairly well-fixed skeletal burden of Sr^{90} amounted to about 3 percent of the Sr^{90} content of the mother's skeleton at term.

The Sr^{90} concentration in milk samples from an injected female taken shortly after the birth of her second offspring (402 days postinjection) was 3 to 4 times the Sr^{90} level of a plasma sample taken a few days later.

INTRODUCTION

It is generally agreed that Sr^{90} is potentially the most dangerous of the fission products. It is produced in relatively high yield in the fission process, and has a long physical half-life Many of its compounds are quite soluble and are readily absorbed by both plants and animals. Once absorbed by an animal, Sr^{90} is retained for long periods in the skeleton [1].

Dudley [2] has compiled a survey of the literature on Sr^{90} in mammals to mid-1954; this survey is in the form of tables setting forth the animals employed and their age, the dose administered, the length of the study, and the effects observed. Numerous investigations have been made or are under way of the absorption, distribution, and elimination of strontium isotopes under varying conditions in laboratory rodents, [1–7] dogs, [8] and domestic livestock [9, 10].

The most important animal from the standpoint of human society is necessarily man himself. Data currently being applied to

human beings have been derived chiefly from three sources: (*a*) Extrapolation from experience with laboratory animals, undependable at best; (*b*) studies of the behavior of stable strontium in normal man; [11–13] and (*c*) tracer experiments with shorter-lived radioisotopes of strontium in patients with advanced diseases, usually neoplastic [14, 15]. For obvious reasons data on Sr^{90} in normal human beings can be obtained only from accidental contamination such as that reported by Cowan *et al.* [16] and the exposure of the Marshallese during Operation Castle [17–19].

It was believed that another primate, the rhesus monkey, might provide valuable clues to the behavior and effects of Sr^{90} in man despite the differences in life span—20 years vs. 65 years—and diet—herbivorous vs. omnivorous. Edington *et al.* [20] reported that 0.5 mC/kilo of Sr^{90} was lethal to monkeys in 35 to 60 days. Using microradiographic and autoradiographic techniques, Jowsey *et al.* [21] found that in the tibiae of monkeys Sr^{90} was laid down quite unevenly, and apparently only in areas of bone growth.

This report is a summary of the data obtained during the past 2½ years in the course of a series of investigations with Sr^{90} in the rhesus monkey (*Macaca mulatta*).

METHODS

General Care of the Animals

The animals used in these studies were adult, adolescent, and infant rhesus monkeys of both sexes. Adults and adolescents were maintained on the diet shown in Appendix I unless otherwise specified. They were fed early each morning; the entire daily ration was offered at that time. Upon receipt, each animal was tested for tuberculosis and X-rays were taken for age determination.[1] The TB tests were repeated at least annually. All animals in the colony were weighed once a month. Complete blood counts were also taken monthly. Periodically the colony was checked for intestinal parasites—chiefly worms. When necessary, "erystoid" tablets (0.1 g, Sharp and Dohme) and/or gentian violet capsules (3/20 gr, Lilly) were administered until very few worm eggs could be found in three consecutive daily stool samples. Miscellaneous problems in care were handled with the help of a veterinarian and a dentist. Prior to use in an experiment, all animals were maintained in the colony for a conditioning period of 3 to 6 months.

[1] Tabulation of the bone growth data is still incomplete.

Intravenous Studies

Three adults, Stupe (a normal healthy male), Tony (an older male with an "arthritislike" condition of the lower extremities), and Rosy (a 3-months-pregnant female [2]) were each given 35μC of carrier-free Sr^{90} as the equilibrium mixture of Sr^{90}-Y^{90} and 135μC of high-specific-activity Ca^{45} intravenously in isotonic sodium citrate.[3] One adolescent, Pat, received only Sr^{90}, 3 months after being placed on a low-calcium diet (the standard diet without the milk and vitamin supplements).

After injection of the radioisotopes all animals were placed in metabolism cages, and daily collections of urine and feces were made for 10 days. After the initial 10-day collection period the animals were returned to their regular cages. Fecal samples were obtained periodically without transfer of the animals from their regular cages.

Stupe and Rosy were kept for breeding and long-term study. Pat was sacrificed 94 days after injection, and Tony, 242 days after injection, both with overdoses of nembutal. Muscle was dissected from the various parts of the skeleton, which were ashed individually for radioactive assay. Muscle and soft-tissue balance were also prepared for assay. Bone biopsy samples were obtained from the two remaining adults and one uninjected adolescent female by amputation of two caudal vertebrae. The operations were performed under aseptic conditions and were followed by a course of antibiotics.

[2] Gestation period for this species is 6 months.

[3] Both isotopes were obtained from Oak Ridge National Laboratory

Breeding Experiments

To date Rosy has been successfully mated with Stupe and has borne three apparently normal offspring. The first, Willie, was born 98 days after the mother received her Sr^{90} injection, Betty was born 402 days postinjection, and Henry was born 840 days postinjection. Daily milk samples of 1.2 to 8.3 ml were obtained from the mother with a breast pump from the third to the sixth day after the birth of the second infant, Betty. The Sr^{90} levels of the red blood cells and plasma of the mother were obtained 30 days later. All three infants were removed from the mother at birth and have been raised on formula by members of the staff. The formula and dietary supplements are shown in Appendix II. A careful record has been kept of the food intake, body weight, and blood counts of the infants.

The Sr^{90} burdens of the first two infants were checked when they were 3 months old by *in vivo* counting of the Bremsstrahlung produced by the Y^{90} beta particles with two 2-inch sodium iodide scintillation counters. Bone biopsies (caudal vertebrae) were obtained from Willie and Betty at 20 and 10 months of age respectively. Fecal samples were obtained periodically from each animal for Sr^{90} assay.

The youngest, Henry, was checked for Sr^{90} content 8 days after birth by the above-mentioned *in vivo* method. This animal has worn plastic pants and a diaper (fig. 1) since he was a few days old so as to facilitate collection of excreta. Pooled excreta were collected daily from birth until age 36 days to establish the rate of elimination of the Sr^{90} acquired *in utero*. When 36 days old he was put on a long-term low-level feeding program. For the past 5 months he has received daily in his first bottle 0.0043 μC of Sr^{90} as the equilibrium mixture except on weekends and holidays. Since the initiation of the feeding program, pooled daily excreta have been collected, ashed, and assayed for Sr^{90}.[4] Retention has been measured by (*a*) calculation from excretion data, (*b*) periodic *in vivo* counting, and (*c*) caudal vertebral biopsy.

[4] Unfortunately, urine and feces are not readily separable.

FIGURE 1.—*Infant monkey with plastic pants and diaper.*

Absorption and Retention in Adolescents

Six adolescent monkeys,[5] two males and four females, have received daily 0.0066 μC of Sr^{90} as the equilibrium mixture except on weekends and holidays since June 26, 1956. A round slab of banana is scored with a knife, and 0.1 ml of a dilute saline solution of Sr^{90} is spread over the scored portion. The "spiked" banana is offered to each animal at least 10 minutes before the rest of the day's ration is presented. So far, there have been few difficulties in this feeding procedure because the animals are hungry, and banana is their favorite food. At the beginning of the feeding period the animals were housed in metabolism cages for collection of excreta. The separation of urine and feces is not complete because of the semiliquid nature of the stools, particularly after treatment for worms. Excreta are collected every other day and pooled on a weekly basis for assay. Twelve weeks after the initiation of the feeding program three of the

[5] Estimated age at initiation of feeding program: 2 years.

animals were placed on a low-calcium diet [6] consisting of fruit and vegetables; a milk substitute of butter, sugar, hydrolyzed casein and water (in the same proportions as are present in whole milk); and the usual supplement of vitamins and iron.

Radioactive Assay Procedures

Samples with very low levels of activity, such as blood and milk from injected animals and bones and excreta from infants, were sent to Nuclear Science and Engineering Corporation, Pittsburgh, Pennsylvania, for assay. Bones and excreta from injected animals and those on the feeding program were assayed according to the following procedure: After dry ashing, the samples were digested with concentrated HNO_3 or *aqua regia* until solution was nearly complete and then evaporated to dryness. Dilute HNO_3 was added so that 10 ml of the final solution represented approximately 0.5 g of ash. Small aliquots were taken from samples containing both Ca^{45} and Sr^{90}, transferred to weighed gold plates, and treated according to a procedure described previously [22]. All samples were stored for at least 30 days to allow for attainment of radioactive equilibrium. The Ca^{45} and Sr^{90} beta-particle activities were measured with a thin-end-window G-M counter by differential filtration. Aliquots of samples containing only Sr^{90} were placed in weighed porcelain ashing capsules, evaporated to dryness, and counted with a G-M counter. In each case the appropriate corrections were made for self-absorption, and corrected counts were compared with an aliquot of the administered dose.

RESULTS

Distribution and Excretion of Intravenously-administered Sr^{90}

The decline in urinary excretion rate of intravenously administered Sr^{90} is shown in Figure 2 for four adult rhesus monkeys. Because of the wide variations in the curve shapes for the individual animals, a scatter diagram with an average curve (broken line) is shown. The average urinary excretion curve (broken) has two components with half times of 0.8 and 4.2 days.

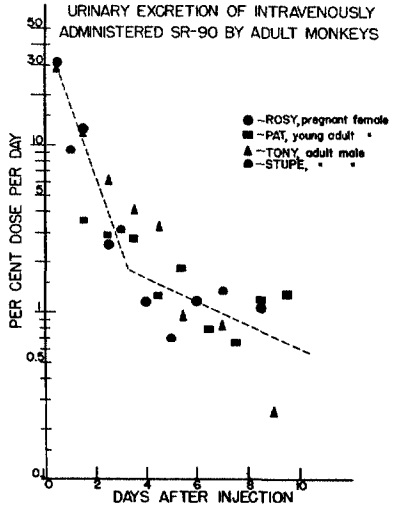

FIGURE 2.—*Urinary excretion rate of Sr^{90} by adult rehesus monkeys.*

A comparison of the cumulative urinary excretion of Ca^{45} and Sr^{90} is shown in Figure 3 for two of the adults. In contrast to the wide variation in the individual rate curves, the cumulative curves are quite similar for these two animals. Renal excretion of Sr^{90} was apparently more efficient than that of Ca^{45}. Similar results have been obtained for other species [15, 23, 24]. Figure 4 shows the fecal excretion rate of Sr^{90} in the two surviving adults to 900 days postinjection. The slope of the slowest component, which appears at about 200 days, was similar for the two animals despite the fact that the female had experienced three closely spaced pregnancies. It is quite unlikely that the urinary excretion-rate curve has a different shape, inasmuch as the Sr^{90} eliminated by either route is derived from the same source, namely, the circulating blood. Experiments are under way to test this point.

[6] The regular diet contains 1,395 mg of calcium per day and the low-calcium diet, 696 mg calcium per day.

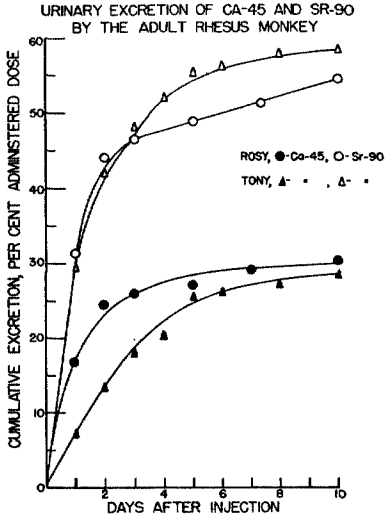

FIGURE 3.—*Cumulative urinary excretion of Ca^{45} and Sr^{90} by adult rhesus monkeys.*

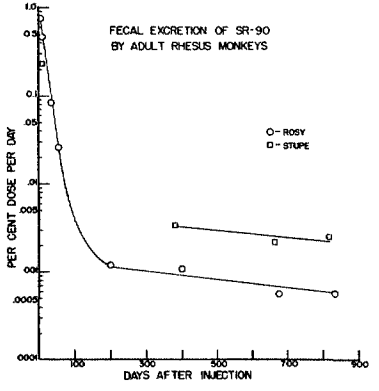

FIGURE 4.—*Fecal excretion rate of Sr^{90} by adult rhesus monkeys.*

Table I shows the distribution of Sr^{90} in the various parts of the skeletons of an adult and an adolescent monkey. The ratios Sr^{90}:Ca^{45} for the skeletal parts are shown for the adult. As might be expected on the basis of age, differences in diet, and postinjection interval, the Sr^{90} level was generally higher in the bones of the adolescent. These differences were more striking in flat bones than in the long bones. The Sr^{90} content of the vertebrae seems to be reasonably representative of the skeleton as a whole. It was for this reason, as well as the simplicity of the operative procedure, that caudal vertebrae were selected for bone biopsy.

With the exception of scapulae, paw bones, and ribs, the Sr^{90}:Ca^{45} ratios were quite similar for the various bones. The mean Sr:Ca ratio for the entire skeleton of this animal was 0.52.

The body burdens of Sr^{90} in the two surviving adults, estimated from bone biopsy, are shown in Table II. Average half times for Sr^{90} were calculated for the male and female, based on retention 10 days after injection (57.7 percent and 36.3 percent of the administered dose respectively), and on the estimated body burdens at approximately 600 days. For the male the average half time was 470 days, and for the female, 315 days. The successive pregnancies of the female (but without lactation) appeared to hasten the elimination of Sr^{90}.

A measurable amount of Sr^{90}, 1.5 ± 0.5 dpm,[7] was found in a 63.8-mg sample of vertebral ash obtained in August 1955 from Alice, an animal that had not been given Sr^{90}. This Sr^{90} does not seem to be due to contamination, because a great deal of care was exercised to avoid contamination during the operative and ashing procedures.

[7] Martell reports that the error in measurement of Sr^{90} by the "Chicago Sunshine Method" used by Nuclear Science Engineering Corporation is less than 10 percent [25]. The activity of this sample was well within their limits of sensitivity.

TABLE I

The distribution of carrier-free Sr^{90} in the rhesus monkey after intravenous administration. Each animal received 35 μC Sr^{90}; Tony also received 135 μC Ca^{45}

	Pat—3.5-yr-old female 94 days postinjection		Tony [a]—6.5-yr-old male 242 days postinjection		
	Sr^{90}		Sr^{90}		
	% dose	%/g ash	% dose	%/g ash	% Sr^{90} / % Ca^{45}
Skull	7.00	0.206	2.96	0.068	0.50
Mandible	2.76	.264	1.31	.113	.57
Scapulae	1.77	.294	1.28	.138	.62
Clavicles	.29	.290	.31	.135	.57
Sternum	.25	.295	.35	.125	.49
Ribs	2.00	.278	.90	.106	.63
Paw bones	2.53	.141	2.05	.116	.60
Humeri	2.93	.207	2.39	.136	.52
Ulnae	1.47	.217	1.14	.134	.48
Tibiae	2.88	.202	1.67	.148	.55
Femora	4.37	.197	1.56	.111	.39
Radii and fibulae	1.82	.191	1.20	.122	.42
Patellae	.18	.277	.35	.175	.50
Cervical and thoracic vertebrae	2.56	.224	2.80	.135	.54
Lumbar and caudal vertebrae	4.48	.258	8.74	.148	.52
Pelvis	4.50	.293	[b]	[b]	[b]
Teeth	.96	.106	.27	.016	.49
Muscle	.10	.007	.09	.006	.82
Soft tissue balance	.08	.009	.11	.007	.61

[a] Arthritic (i. e. pelvis and lumbar and caudal vertebrae heavily calcified and fused).
[b] Included with lumbar and caudal vertebrae.

TABLE II

Sr^{90} content of biopsy samples of caudal vertebrae from injected adult male and female rhesus monkeys, the first two offspring of an injected female, and one control female. Injected animals received 35 μC Sr^{90} intravenously

Animal	Estimated age at sampling	Days postinjection	Source	Body wt at sampling (kg)	dpm Sr^{90} per g ash of caudal vertebrae	Estimated Sr^{90} body burden [a] (μC)
Rosy	5 6 yr	638	Hooper Fdn	4.85	2.95×10^4	3.2
Stupe	5.5–6 yr	623	do	11.1	5.95×10^4	8.2
Pat	3.5 yr	94	Commercial	3.35	1.53×10^5	[b] 11.9
Willie	20 mo	(98 for mother)	Rosy and Stupe	2.95	5.5×10^2	3.6×10^{-2}
Betty	10 mo	(402 for mother)	do	1.99	8.3×10^2	3.7×10^{-2}
Alice	3–3.5 yr.	None-control	Commercial	3.84	2.35×10^1	2×10^{-3}

[a] 1 μC = 2.22×10^6 dpm; body burden based on estimate from Table I that Sr^{90} concentration in caudal vertebrae is representative of total skeletal Sr^{90}; average ash content of monkey bone (plus marrow) taken as 27.5%; percent body weight of bone estimated at 18% for infants and females and 10% for adult males.
[b] Actual measured body burden 15 μC; error of estimate of this sort is thus in the range of 15%.

Samples of milk were obtained from Rosy for four successive days shortly after the birth of her second infant. Table III shows the Sr^{90} level in the milk, the daily fecal excretion rate at that time, and the Sr^{90} content of plasma 1 month later. Except for the first sample taken 3 days postpartum, the milk concentration was from three to four times that in the plasma.

The blood-count data have proved to be of little value because of the introduction into the colony late in 1954 of a blood parasite similar to *Bartonella*. The original infected animals were destroyed, but Rosy and Stupe apparently still remain carriers, and the parasite is now endemic in the colony.

Sr^{90} in Infant Monkeys

Table IV gives the Sr^{90} content of the first two infant monkeys 3 months after birth, and 8 days after birth of the third as determined by in vivo scintillation counting. The last two lines in Table II show the Sr^{90} burdens calculated from bone biopsy samples for the two older monkeys, Willie and Betty, at 20 and 10 months of age, respectively.

The Sr^{90} burdens of these animals apparently had no ill effect upon their growth rate, as shown in Figure 5. The growth rates of the three siblings were very close to that reported by Pickering et al. [26] for infant monkeys of this species raised under similar conditions. During the first 6 months to a year the blood counts of all three infants were within normal limits.[8]

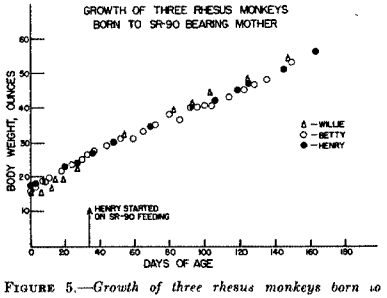

FIGURE 5.—*Growth of three rhesus monkeys born to Sr^{90} bearing mother.*

Samples of pooled urine and feces from Betty at 14 and 140 days of age contained 260 dpm/day and 38.6 dpm/day, indicating that the Sr^{90} acquired by placental transfer was eliminated fairly rapidly. Nearly a year after birth (305 days), Willie, the oldest, was still excreting Sr^{90} in the feces at a relatively high level: 205 dpm/day. More recent excretion samples from these two animals have not yet been analyzed.

On the basis of the data in Table IV, and the calculated body burdens shown in Table II, approximate half times for elimination of Sr^{90} during the first 10 months of life were calculated for the two older infants: 195 days for Willie, and 155 days for Betty. Extrapolation back to the time of birth of these two infants provides a rough estimate of the placental transfer of Sr^{90}. In the first-born, Willie, placental transfer accounted for about 0.3 μC Sr^{90}, or 3 percent of the mother's retained dose; the retention by the mother was calculated from her half-time value of 315 days. Rosy received her Sr^{90} injection halfway through the second trimester of her first pregnancy. The extrapolated Sr^{90} content for the second offspring, Betty, was slightly more than 0.15 μC, 2.9 percent of the mother's retained dose 402 days postinjection. When Henry, the third offspring, was 8 days old, an in vivo count was on the border line of the sensitivity of the counting method, and the Sr^{90} burden was estimated at something less than 0.1 μC, or slightly more than the 3 percent calculated for the other two. Although the number of individuals was small, and the measurements subject to errors of about 15 percent, placental transfer of a gradually declining burden of Sr^{90} can be estimated at something close to 3 percent of that in the mother shortly before the birth of the infant.

[8] At age 14 mo Betty's red blood cell count dropped to less than one million. A few days later she succumbed, apparently as the result of infection with the blood parasite mentioned above. Her skeleton is currently being processed for assay. Willie was infected at the same time but responded to a heroic course of treatment with "Aralen," chloroquin hydrochloride (Winthrop-Stearns), and massive injections of liver, iron, and folic acid. His red cell count has remained at from 4.1 to 4.5 million for the past 3 months, and he has continued to gain weight at an apparently normal rate.

TABLE III

The blood level, fecal excretion rate, and milk concentration of Sr^{90} in an adult female rhesus monkey, Rosy, 3 to 40 days after the delivery of her second offspring on the 402nd day after receiving 35 μC of Sr^{90} intravenously

Sample	*Days postinjection*	*Sr^{90} concentration (%/ml)*
Plasma	449	3.7×10^{-6}
Red blood cells	449	1.6×10^{-6}
	(Second offspring born 402 days postinjection)	
Milk:		
3 days post partum	405	2.6×10^{-6}
4 days post partum	406	6.5×10^{-6}
5 days post partum	407	9.1×10^{-6}
6 days post partum	408	1.7×10^{-5}
Fecal excretion rate	397 to 407	1.1×10^{-3} %/day

TABLE IV

Placental transfer of Sr^{90} in the rhesus monkey estimated by *in vivo* scintillation count of the infants

Vital statistics			Counting		
Animal	Birth date	Day after mother injected	Date	Age (days)	Estimated Sr^{90} content * (μC)
Willie	7/7/54	98	10/29/54	104	0.22
Betty	5/7/55	402	8/12/55	97	.10
Henry	5/20/56	840	5/28/56	8	$<.10$

* Low counting rate makes for probable error of at least 25%.

Table V shows the retention by the infant monkey, Henry, of orally administered Sr^{90} for the first 13 weeks of the feeding program. The daily excretion pattern is not tabulated, but is of some interest because it is so consistent. Values for a typical week were as follows: Monday, 36.5 cps; Tuesday, 39.4 cps; Wednesday, 42.3 cps; Thursday, 43.4 cps; Friday, 49.8 cps; Saturday, 6.0 cps; and Sunday, 5.1 cps. The dose averages 62 cps/day Monday through Friday. The mean weekly retention during this 13-week period was 18.2 percent of the administered Sr^{90}, or 0.048 μC. Although the level of activity in the animal is still too low for accurate *in vivo* counting, a Sr^{90} measurement was made by this method after 12 weeks of Sr^{90} that agreed fairly well with the retention calculated by difference in administered and excreted Sr^{90}.

The data on retention by the adolescent monkeys of oral Sr^{90} are being analyzed at the present time, and accurate values cannot be given as yet. Based on the initial calculations, an upper limit of something less than 5 percent can be set for the retention of oral Sr^{90} by 2- to 2.5-year-old monkeys.

TABLE V

Retention of 0.0043 μC Sr^{90} fed daily as the equilibrium mixture of Sr^{90}-Y^{90} in milk to an infant monkey (Henry). Feeding was started at age 36 days

Weeks	Sr^{90} fed dpm x 10^4	Retention	
		dpm/week x 10^3	% weekly dose
1	4.42	6.63	15
2	3.67	8.15	22.2
3	4.65	5.25	11.3
4	4.77	9.16	19.2
5	4.72	13.92	29.5
6	4.75	6.74	14.2
7	4.75	10.83	22.8
8	4.75	6.32	13.3
9	4.65	3.63	7.8
10	4.70	10.81	23.0
11	3.78	10.05	26.6
12	4.75	6.94	14.6
13	4.75	9.02	19.0

DISCUSSION

Most of the results described above were obtained from measurements on only a few individuals; nevertheless, some tentative conclusions may be drawn. The metabolism of Sr^{90} in the monkey followed qualitatively the pattern described for other species [1–10]. Early elimination was chiefly urinary; later, excretion occurred in urine, feces, and milk.

Retention was prolonged, and in the adults the half time was on the order of 400 days. The most widely quoted half time for skeletal retention of Sr^{90} (>200 days) is that derived by Hamilton [1] from experiments with adult rats. This figure was rechecked recently in this laboratory in a double labeling experiment with Ca^{45} and Sr^{90} in rats, and the half time obtained was on the order of 350 days or about one-half of the animal's remaining life expectancy [27]. The biological half life for Sr^{90} in man currently accepted by the International Commission on Radiological Protection [28] is 11.2 years and is based on the original work by Hamilton [1] and by Sullivan *et al.* [29] with rats. With corrections for the difference in life expectancy—20 to 25 years for the rhesus monkey and 65 to 70 years for man—a biological half time based on the monkey data presented in this report would be in the neighborhood of 3 years, or about one-fourth of the currently accepted value.

The turnover of Sr^{90} was much more rapid in the infant monkeys; the half time can be set tentatively at about 6 months.

Placental transfer from a mother with a relatively firmly fixed Sr^{90} burden was roughly 3 percent of the Sr^{90} retained by the mother at term. The concentration of Sr^{90} in the milk of the breeding female was 2 to 4 times the plasma level, indicating that for this species a significant amount of Sr^{90} would be transferred to the nursing young. Secretion of Sr^{90} in milk and its subsequent accumulation in the bones of the young has been demonstrated for rats and mice [4, 30] and for cows [31].

In the infant monkey with a rapidly developing skeleton, 18 percent of orally administered Sr^{90} was retained, compared with less than 5 percent for adolescent monkeys with presumably nearly complete skeletal growth. It should be noted that the diet of these latter animals was much richer in calcium, phosphorus, and protein (designed to resemble the diet of Western Man) than what would be available to them in their natural habitat.

ACKNOWLEDGMENTS

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This work was done under the auspices of the U. S. Atomic Energy Commission.

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Appendix I.- Standard monkey diet

Item	*Source*	*Daily ration* [a]
Fruit and vegetables:		
Apple	Local	¾
Orange	----do	¾
Banana	----do	½
Carrot	----do	3 med.
Peanuts	----do	1 doz
Chim Biscuits—2 oz	Kennel Food Supply Co., Fairfield, Conn.	2
Whole milk, reconstituted powdered.	Golden State Co.	1 pt
Dietary supplements added to milk:		
"Meritene"—protein supplement	Dietene Co., Minneapolis, Minn.	1½ tbsp [b]
"Vi-mix Drops"—Vit. A, B complex, D	Lilly	0.5 cc [c]
"Zymatinic Drops"—Vit. B complex, iron, liver	Upjohn	0.5 cc [c]

[a] Average amounts for 2- to 5-kg monkey, adjusted upward for larger animals.
[b] One-half human adult minimum daily protein requirement
[c] Approximately equal to human infant minimum daily requirement.

Appendix II.—Infant monkey diet

Item	*Source*	*Daily ration*
Formula:		
Whole cow's milk (16 oz), sucrose (3 tbsp)	Local	8 to 9 oz [a]
Fresh fruit (orange, apple, banana)	----do----	20 g
Dietary supplements:		
"Vi-daylin" (Multivitamin mixture)	Abbott	1 cc [b]
"Feosal" (Fe_2SO_4)	Squibb	1 cc [b]

[a] Formula constitution given for infants 2 months of age and older. A more dilute formula is used for the first 2 months.
[b] Approximately one-half human infant minimum daily requirement.

DISCUSSION

Patricia W. Durbin

Dr. Langham. I would like to ask, did I understand that you were or were not satisfied with whole body counting of Bremsstrahlung as a means of measuring strontium 90 in these animals?

Dr. Durbin. I think whole body Bremsstrahlung counting is a splendid idea, and I am sure that we would be much more satisfied with it if the equipment that we had to work with were a little bit more accurate and certainly a good deal more sensitive. At the present time our lower limit is a tenth of a microcurie in an animal that weighs about a pound and a half. I am sure with a liquid scintillator or larger crystals we could do a great deal better than that.

Dr. Langham. With the larger crystal you could. With the liquid scintillator, you could not get anything probably to amount to much, because it has about a 300-kilovolt cutoff in energy sensitivity. The bigger crystal might. The reason I was asking this, is that this is being proposed as a means of determining the strontium burden in the chronic dog experiments being planned in Davis, Calif.

PLUTONIUM CONTAMINATION FOUND OFF-SITE FOLLOWING ONE-POINT DETONATIONS

By M. W. Carter and O. R. Placak

U. S. Public Health Service, Las Vegas, Nevada

A series of four experiments were conducted during the winter of 1955 to determine if accidental detonation could occur and, if so, the potential spread of contamination resulting from accidental detonation of devices.

The off-site area includes all territory within an approximate 100-mile radius, but excluding the actual detonation area. A comprehensive report of these activities has been prepared and is available in the files of the Las Vegas Branch, Test Division, Albuquerque Operations Office, U. S. Atomic Energy Commission.

Estimation of alpha contamination over many square miles of desert is not an established routine undertaking. The following methods were used for monitoring purposes:

(1) Surface monitoring with portable proportional alpha survey instruments (Pee Wee).

(2) Fallout trays (80 square-inch sampling area) smeared with a relatively nondrying adhesive alkyd resin. These were placed in rings around the detonation area to distances of approximately 30 miles.

(3) Staplex air samplers using glass fibre filter papers and an effective filtering area of 63 square inches. Filter runs of 24 or 48 hours were accomplished without appreciable loss of flow rate. Air samplers were located in 11 populated communities surrounding the Nevada Test Site and at 12 locations on the site The maximum distance of air sampler location was 95 miles.

(4) A mobile air sampler consisting of a Staplex sampler shock mounted on a trailer unit towed by a Jeep was used to simulate work-party conditions in areas where ground contamination existed.

Pee Wee survey instruments are very useful in the field for locating contamination and for determining the order of magnitude of such contamination. Survey instrument readings should be considered as indicative of the minimum amount of alpha contamination present at a particular spot and not as a representative value for an extended area of desert. Results of alpha survey instrument monitoring indicate the extreme variability to be expected over a relatively small area on the same type of surface. For example, on a limited area of concrete pad, Pee Wee readings varied from 500 counts per minute to 1,400 counts per minute. There appears to be no strict correlation between Pee Wee ground surface readings and laboratory counts on fallout trays located at the same spot. In order to have strict correlation it would be necessary to have uniform distribution over the entire tray area in addition to the same amount of dust overlay acting to shield each uniformly distributed particle.

Fallout trays proved to be a simple convenient means of monitoring plutonium contamination. They are easy to monitor in the field and are easy to collect and transport to a central laboratory for more detailed analysis. They also serve to differentiate new fallout from residual alpha contamination which may be present in the same general area. Maximum contamination found on a fallout tray was 100,000 disintegrations per minute per square foot at a distance of approximately 5 miles.

Detectable contamination was noted on fallout trays located at distances of 50 miles

All air sampling stations, at some period during the tests, have indicated plutonium concentrations in the air. The highest single daily exposure within the Nevada Test Site occurred at Gate 385 and amounted to 154 disintegrations per minute per cubic meter. The highest single exposure beyond the confines of the Nevada Test Site limits occurred at Indian Springs, Nevada, and amounted to 5.3 disintegrations per minute per cubic meter. Detectable plutonium was found on air sample filters at distances of 100 miles and these results were confirmed by chemical analysis.

The pattern of contamination was the same for all air samples at all locations. Depending on the distance from the point of detonation, there was a sharp rise in alpha counts on air filters on shot day or the day following. This persisted for 3 to 4 days with decreasing intensity, with a return to background levels on the fourth or fifth day.

After an area has been contaminated, surface monitoring readings are inadequate to measure the hazard to work parties in this area. A mobile trailer mounted air sampler which could collect the dust stirred up by the towing vehicle was used to simulate working conditions. There is little correlation between these two types of readings. For example, the same air concentration of about 200 disintegrations per minute per cubic meter was obtained in areas where the Pee Wee readings were 1,000 counts per minute, 14 counts per minute and 7 counts per minute. The discrepancy between the two types of measurement increases with time. This is understandable when one considers that weathering due to rain and wind erosion tends to cover up the alpha contamination and to render it undetectable by survey instrument monitoring. There is continuous redeposition of plutonium due to wind action, but this appears to represent relatively minor concentrations, that is less than one disintegration per minute per cubic meter on air filters.

A workable method for decontamination of a relatively large area of the Nevada Test Site consisted of removal of topsoil in the areas of highest contamination and harrowing, wetting, compacting and stabilizing the balance of the area involved.

DISCUSSION

M. W. Carter and O. R. Placak

Dr. LANGHAM. Thank you Mr. Placak. This, of course, is a problem that has been rather dear to our hearts for some time now with regard to the possibilities of contamination from such detonations. Certainly the process of harrowing a piece of land and thereby mixing the plutonium with a greater amount of inert material is very comparable to the old, old trick of painting a laboratory surface with a coat of paint in order to remove plutonium contamination from the zone where it could become a potential health hazard. So these to me seem to be very sound practices with regard to the decontamination of the area All one has to do is to mix the plutonium at the surface with one centimeter of the upper earth's surface to produce a dilution factor of 1×10^5.

Do we have any other comments on this particular topic?

Dr. WYCKOFF (Bureau of Standards). May I suggest that this seems to be a test situation which offers a unique opportunity for making measurements of the fine structure of fallout patterns? Some measurements which were inquired about during the first day of this symposium and which it was indicated had never been made before. If there are particular buildings or structures in the area contaminated by plutonium, it would seem quite straightforward to make detailed measurements around these structures to indicate some measure of the variations in intensity of the fallout in close to large buildings, the snow fence effects, and things of this sort.

I wonder if any measurements of this type have been made by the health monitoring people?

Mr. PLACAK. Obviously we did not intend to put anything on the proving ground. It was supposed to go the other way. What did go into the proving ground went into an area that has only one real building, and that is the old 1953 civil defense house. We didn't make the measurements that you indicate. However, it may be very difficult to monitor significantly and determine the type of information you are asking for, because practically the only monitoring instruments we have are Peewees, or something similar. We found during this survey that if you take on the same surface an area of concrete about the size of that platform which was in the fallout pattern and presumably should have been uniformly contaminated—if we monitor that very carefully--we will find a wide range of monitoring results. They will go all the way from 500 counts per minutes to 1,500 counts per minute, depending, I suppose, on small nonuniformities in the surface, how much dust is on the top of the material or various other factors.

It is really difficult to monitor for this stuff. Unless we do it as we attempted to do it by establishing an artificial surface, a fallout tray covered with an alkyl resin, and then make a very desirable monitoring surface, I don't know how you can do it.

Have I answered your question at all?

Dr. LANGHAM. I think what this amounts to is that the short range of the alpha makes it so unusually difficult to detect that the methods that are easy for making such measurements are not sufficiently sensitive to give the detail you would like to have. This is the principal objection to it, I think.

448029 O—58——13

RETENTION OF SUB-MICRON AEROSOLS IN THE HUMAN RESPIRATORY TRACT[1]

By J. N. Stannard and P. E. Morrow
University of Rochester, Rochester, New York

Since the 1930's there has been a steady growth in our appreciation of the parameters which govern the deposition of dust in the respiratory tract of man and laboratory animals. Particle size, shape, density, and the anatomical and physiological characteristics of the respiratory system all play a part. Of cardinal importance in these is particle size.

A summary of our knowledge of the role of the particle size, or was until recently, virtually a summary of our knowledge of the deposition process. In Figure 1 is a graphic summary of some key studies on this subject. This is reproduced from a very timely and informative survey by Hultqvist [1].

As indicated in the legend, curves 1–5 are derived from theoretical considerations. They relate to ideal particles in model lungs, in postulated gravitational and centrifugal fields. Curves 6–12 are based on experimental data. Differences between the experimental curves are due in part to differences in the respiratory characteristics and methodology in the various experiments.

Note that the graphs refer to *total* retention in the respiratory tract, i. e., retention in the upper respiratory tract as well as the alveoli. If the percentage lower respiratory tract retention were plotted the relationship would be similar except a distinct maximum would appear at about 1–2 μ diameter. This occurs because particles above 5 μ seldom reach the lower respiratory tract.

The results show considerable variation in detail, but agree in showing two things:

(1) less percentage deposition at sizes between 2 μ and 0.2 μ than at either larger or smaller sizes. (This does not supply a minimum in total mass deposition.)

(2) almost complete lack of experimental information and no notable unanimity of theoretical opinion in the submicroscopic size range (i. e., <0.1 μ) where particles are relatively unaffected by gravity or the usual inertial forces.

From the standpoint of the hazard from inhaled fallout material, other things being equal, it will make a great deal of difference whether the radioactivity is:

(*a*) Predominantly on particles so large they will not be respired.

(*b*) Predominantly on particles in the size range which will deposit in the upper respiratory tract.

(*c*) Predominantly on small particles which will be retained largely in the deeper portions of the respiratory tree.

Data on the particle size distribution as related to the activity distribution are not generally available for fallout activity.

Obviously at early times the activity distribution will presumably involve a wide range of particle sizes. Later as settling or aggregation, etc., occur the bulk of airborne activity may be

[1] This paper was not originally intended as more than a 10-15 minute summary of some current experimental work being done by one of us (P. E. Morrow). However, in view of the rather extensive reviews presented on other phases of the fallout problem at this symposium, the paper was expanded somewhat to fit a more general consideration of possible inhalation hazards in a fallout field. The experimental work appeared as an abstract in the American Journal of Physiology, *187:* 618, 1956, and in a University of Rochester Atomic Energy Project report (UR-504). The complete manuscript has been accepted for publication in the A. M. A. Archives of Industrial Health. This being the case, the manuscript was revised somewhat in proof to omit experimental details which can now be found elsewhere.

448029 O—58——14

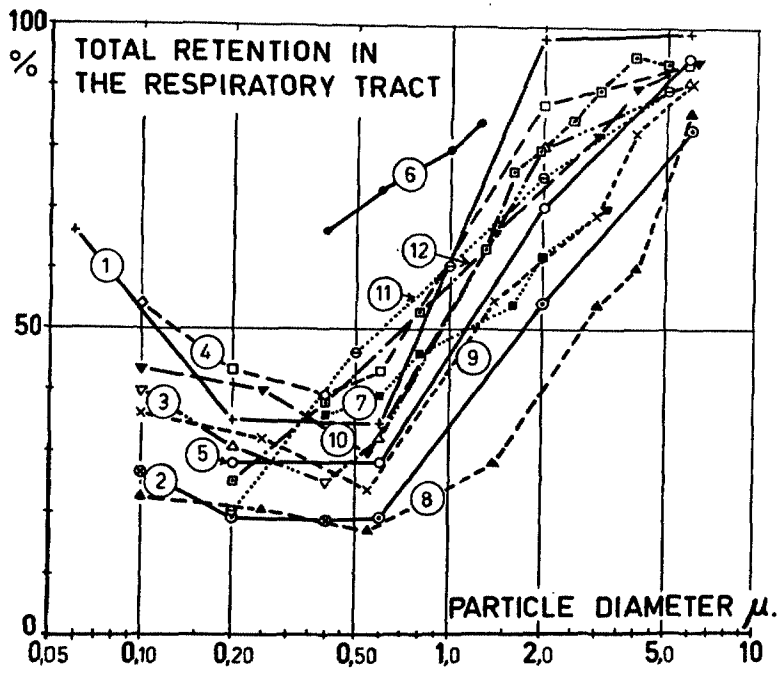

FIGURE 1.*

THEORETICAL DATA

Curve No. 1: Findeisen [2]: Postulated flow rate, 200 cm³/sec; 14 cycles per min.

Curve No. 2: Landahl [3] and Landahl *et al.* [4]: Flow rate, 300 cm³/sec; 15 cycles per min; tidal air volume, 450 cm³.

Curve No. 3: —— Flow rate, 300 cm³/sec; 7½ cycles per min; tidal air volume, 900 cm³.

Curve No. 4: —— Flow rate, 300 cm³/sec; 5 cycles per min; tidal air volume, 1350 cm³.

Curve No. 5: Landahl [3]: Flow rate 1000 cm³/sec; 15 cycles per min; tidal air volume, 1500 cm³.

EXPERIMENTAL DATA

Curve No. 6: Wilson and La Mer [5]: 5½ cycles per min.

Curve No. 7: —— 20 cycles per min.

Curve No. 8: Landahl *et al.* [4]: 15 cycles per min; tidal air volume, 450 cm³.

Curve No. 9: —— 7½ cycles per min; tidal air volume, 900 cm³.

Curve No. 10: —— 5 cycles per min; tidal air volume, 1350 cm³.

Curve No. 11: Brown *et al.* [6]: Each point represents the mean of many values.

Curve No. 12: Van Wijk and Patterson [7]: 19 cycles per min.

*Taken directly from reference 1.

on smaller particle sizes. In fact an interesting estimation of what it *might be* is found in the data of Wilkening [8] for distribution of *natural* radioactivity. A summary of his findings is presented in Table I.

TABLE I.—DISTRIBUTION OF NATURAL RADIOACTIVITY (WILKENING, 8) *

Particle diameter	*Percent of radioactivity*
>0.005	5
0.005–0.015	25
0.015–0.025	50
0.025–0.035	10
>0.035	10

*Wilkening used an electrostatic separator of special design, and as described by Mercer [9] the separation of sizes may not be very dependable. However, this does not negate the fact that most of the activity appears on small particles.

Thus virtually *all* activity appears to be on particles too small for detection in ordinary light microscopes.

In the light of these considerations experimental determination of the retention of *submicron* aerosols in the human respiratory tract has been of intense interest in our laboratory. A few results seemed worthy of presentation here since they represent one of the first extensions of experimental data into the "theoretical zone" seen in Figure 1.

The aerosol was composed of sodium chloride crystals, 99 percent of whose particles were less than 0.4 μ in diameter. Retention was measured by difference between the inhaled and exhaled concentrations.

The retention apparatus consists of two units: (1) an exposure unit, composed of an aerosol generator, mixing chamber, aerosol samplers and a cooperative respiratory valve (the latter item is a high-speed slide valve, controlled by minute pressure changes in the face-piece of the subject, which accomplishes the separation of *inspired* and *expired air*) and (2) a *control* system composed of the respiratory slide valve control unit, an electronic integrator for automatic tidal volume measurement, and a pneumotachograph. The apparatus and methods are described in detail in references 12, 13, and 14.

Thus, information obtained in an experiment includes percent mass deposition, particle size distribution of the inhaled air, and the various dynamic and volumetric characteristics of the respiratory physiology of the subject.

Particle size measurements were made by electron microscopy. A typical particle size distribution for the aerosol used is shown in Figure 2, where both the mass and the count diameters are plotted on a probit scale. The median diameter on a mass basis (MMD) is 0.43 μ, the median diameter by count (CMD) is 0.056 μ, with standard deviation σ g=2.3 in each case.

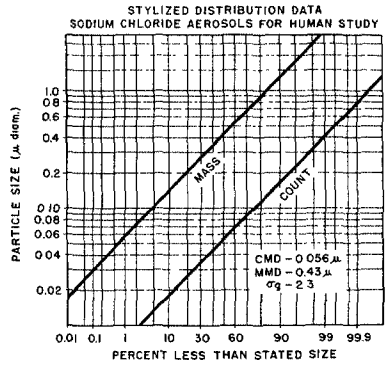

FIGURE 2.

The results of the 17 experiments done on 9 human subjects while breathing spontaneously provide several points of interest. First, on a mass basis the amount of the aerosol deposited in the respiratory tract was found to be somewhat greater than that predictable from commonly accepted particle size-deposition relations. The difference is not large but is significant statistically. Second, there appear to be several physiologic factors which affect the extent of mass deposition.

With regard to the first point, 63.4 percent of the inhaled aerosol mass was the mean deposition value. The 95 percent confidence limit is 57–69 percent for the mean. Generally ac-

cepted relations predict a percent mass deposition value for this particular aerosol distribution of not more than 55 percent. The hygroscopic nature of this aerosol is undoubtedly responsible for some of this increased value, but estimates of this contribution do not account for the difference seen.

The relation of deposition to respiratory characteristics is shown in Figures 3 and 4. In Figure 3 is seen a plot of the percent of the aerosol mass deposited as a function of the mean respiratory frequencies and the mean tidal volume from the 17 experiments. Each experiment provides a single point, and those on the same individual are interconnected. The heavy arrow is to denote the general trend, which is toward *increased* deposition with lower respiratory frequencies and/or higher tidal volumes.

This inverse relationship may be due to an interdependence of these two variables. In other words, the deposition may be a function of tidal volume and since an increased tidal volume is generally associated with a decreased respiratory frequency, such a relation would be expected. However, *unlike* previous reports (at larger particle sizes) wherein these were *invariably* related in an inverse manner, only in 60 percent of the experiments did the mean tidal volume increase as the mean respiratory frequency decreased. (It should be pointed out that these experiments were frequently weeks or months apart.) Consequently, it appears that the tidal volume is as relevant a factor as is the respiratory frequency. It is reasonable to explain this on the basis that as the tidal volume increases, the number of particles inhaled increases, and that a deeper, fuller respiratory tidal volume provides for a more intimate contact of aerosol particles with the vast mucosal surfaces of the lung. Both of these conditions tend to promote Brownian motion deposition. (Remember particle density is of no importance at these sizes.)

In Figure 4 is another set of parameters which appear interesting; the mean respiratory air flow *rates* as plotted against percent mass deposition. As seen in the figure, in the case of inspiration, an increase in the mean air flow generally resulted in an *increased* deposition whereas in the case of expiration, a *decrease* in the mean air flow rate was associated with an *increased* deposition. Again, there is a probable interdependence; one which may involve the tidal volume or respiratory frequency. For instance, it was found that as the respiratory frequency decreased, it was generally associated with an increase in the expiratory phase duration, more so than with the inspiratory phase duration; thereby tending to produce an increased mean inspiratory air flow rate particularly if the tidal volume increased. One may hypothesize that the deposition of the larger particles would be increased by higher inspiratory velocities by impaction and possibly the impaction process might be so efficient during inspiration that it would be relatively unimportant during the expiration. Even more possible is the idea that particle deposition would be improved by the increased turbulence induced by high air flows.

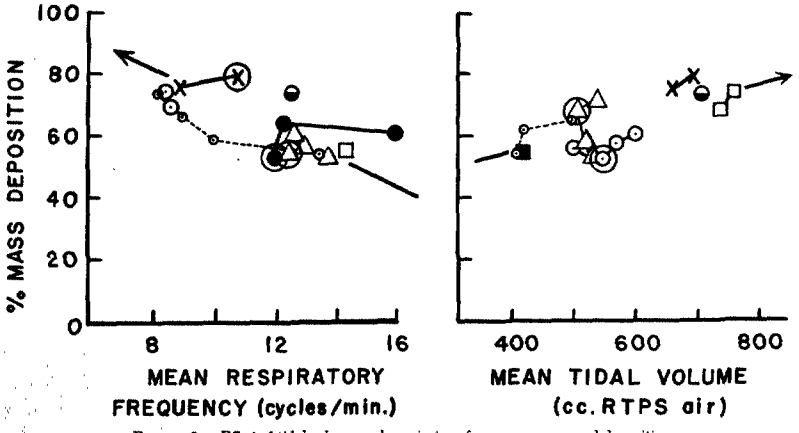

FIGURE 3.—*Effect of tidal volume and respiratory frequency on aerosol deposition.*

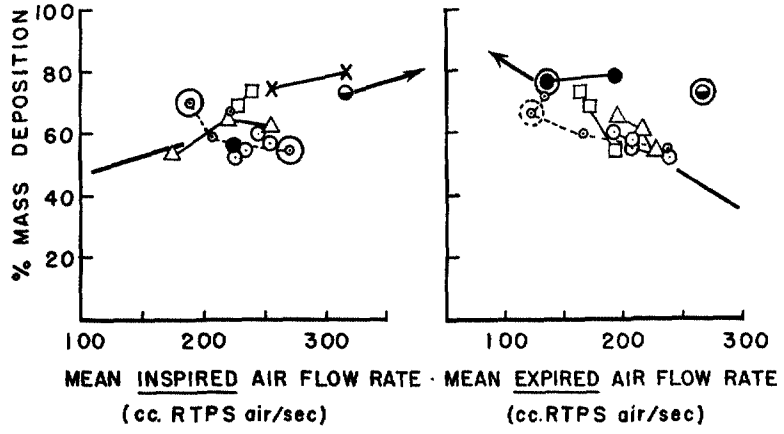

FIGURE 4.—*Effect of respiratory air flow rates on aerosol deposition.*

DISCUSSION

Some points in this experimental study relate to the fallout problem. One is best seen by returning to Figure 2. Observe the mass distribution of this aerosol: 99 percent of the particles are less than 0.4 μ diameter but only 50 percent of the aerosol mass is presumably due to these particles. In other words, a mass deposition value of 50 percent could be based on the nasal-pharyngeal deposition of a few thousand particles greater than 0.4 μ diameter or it could be due to millions of small particles depositing in the lung parenchyma. This point serves to illustrate, first, the need for *particle size* deposition data instead of, or along with, mass deposition data. It demonstrates that mass deposition measurements are based on the recognition of a relatively few particles which are generally believed to only rarely penetrate beyond the anatomical dead space. Such measurements ignore the contribution of the greater percentage of the particles which can presumably penetrate into the lung parenchyma.[2]

Thus, to return to the original considerations,

[2] There is increasing evidence that radioactive nuclei adsorb onto available dust as a function of their *total diameter* rather than total cross-section (Smoluchowski, 10). In our case, equivalent total diameters occur at about 0.09 μ size so that the first (smaller) 65 percent of the particles provide 50 percent of the total diameter but only a few percent of the mass (<3%).

the work of Wilkening and others have *indicated* that in nature, one does encounter a preponderance of radioactivity (>90%) on particles under 1,000 Å (0.1 μ) diameter. On the other hand, assay of fallout concentrations by sedimentation (with possibly a small amount of impaction and Brownian motion deposition) of airborne radioactivity onto adhesive surfaces is commonly employed. Presumably, this technique is about 63 percent "efficient" [11]. This implies that particles under 1,000 Å are primarily involved with the 37 percent remainder. The possibility that "fallout," as studied in the U. S. A., is in large measure tropospheric may be a basis for the apparent preponderance of radioactivity on "larger" particles. So one must presume that the persistent atmospheric radiation is in a colloidal state and that, provided not more than 10^3–10^4 of these particles occur/ml of air, they are likely to remain aloft subject only to radio decay and washout due to precipitation.

Animal experiments (particularly on rat, mouse, etc.) will probably underestimate the risk of these smaller particles due to the extraordinary surface to volume ratio of their nasal-pharynx. On the other hand, man's anatomy and physiology appear to predispose him to the deposition of these particles in amounts uniquely higher than in most experimental animals (with a few exceptions).

The above discussion should make it clear that while the role of particle size has been examined and discussed more than any other factor, many studies, including the present one, point out that the physiology of respiration can influence dust deposition as profoundly as particle size.

Direct application of the above experimental data to quantitative prediction of how the retention of fallout particles in the human lung will be related to particle size is obviously not possible nor intended. However, it is hoped that it will serve to emphasize some points not usually appreciated and aid in the planning of future field experiments.

CONCLUSIONS

(1) Particle size is a cardinal parameter in respiratory tract deposition.

(2) The distribution of radioactivity in a fallout field may or may not be the same as the distribution of particle sizes. More information on this point is needed from field tests. (Particularly number distribution vs. mass distribution.) However, there is a real possibility that much of the activity at later times may be resident on smaller particles, and relatively little experimental information is available on the retention of the smaller size particles by the human.

(3) Samples collected by settling techniques may or may not show the activity of greatest importance as an inhalation hazard.

(4) Preliminary experiments in humans with a sub-micron size sodium chloride aerosol show deposition (retention) to be somewhat higher than predicted by theory, and to be related to the breathing pattern of the individual. The former is important since much of the radioactivity may be on relatively small particle sizes. The latter indicates that, though the primary physiologic factors have not been isolated, there can be little doubt that the manner in which an individual respires may influence the deposition process quite significantly.

(5) While not immediately pertinent to the *short-term* effects of a fallout field because of the over-riding importance of external radiation hazards, and the relative radioresistance of the lung in an acute sense, these considerations can be of importance in the assay of the possible damage from particles which may be present in such a field—and possibly inhaled under conditions where the external hazard would be minimized. Obviously, they are pertinent to evaluation of the longer-term hazards.

ADDENDUM

Since the date of the fallout meeting, two reports pertinent to the distribution of radioactivity as a function of particle size have appeared. These are by Williamson (USNRDL TR 152, 1957) and by Farlow and Schell (USNRDL-TR 170, 1957). Also other indications that deposition of particles smaller than 0.1 μ may be higher than predicted by theory was published by Dautrebande, *et al.* (A. M. A. Arch. Ind. Health *16*, 179, 1957) and an indication of the same tendency was found in a paper by Verzár, *et al.* (Arch. ges. Physiol. *261*, 219, 1955).

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DISCUSSION

J. N. Stannard and P. E. Morrow

Dr. Langham. Thank you, Dr. Stannard. Because of the time we are not going to be able to discuss this. However, I would like very much, purely because the particle problem in this whole business has been that which is always thrown at you when you are trying to assess the hazards associated with inhalation of radioactive materials. Every time you make a statement there is always some fellow who brings up the idea that this all depends on particle size. When he says that it is supposed to stop you cold just like a doctor is supposed to stop you when he says you have a virus infection. So I think anything that can be done to get this particle size problem on a basis of where you can say what specifically does this mean to our problem, then I think we are getting somewhere.

THE SHORT TERM BIOLOGICAL FATE AND PERSISTENCE OF RADIOACTIVE FALLOUT AS MEASURED AT VARIOUS LOCATIONS WITHIN FALLOUT PATTERNS

By R. G. Lindberg and K. H. Larson

Radio-Ecology Division, Atomic Energy Project, University of California at Los Angeles

The problem of assessing the biological hazards of radioactive fallout may be arbitrarily divided into two parts; one, the acute or immediate hazard arising *primarily* from external radiation and *secondarily* from the metabolism of certain fission products; and two, the chronic or long term hazards arising *primarily* from the metabolized fission products, and *secondarily* from external radiation. The division of the problem is real. The exact duration of each phase is not.

The Alamagordo Section and the succeeding Radio-Ecology Division at the Atomic Energy Project, University of California at Los Angeles, has been engaged in part in studying the fate and persistence of radioactive fallout in areas adjacent to continental test sites since 1947. A reasonably continuous record is available of the fate of plutonium contamination near the New Mexico Test Site from 1947 to the present. A record of the fate of repeated fission product contamination in several areas adjacent to the Nevada Test Site from 1951 to the present is also available. Lest we stray from the "short term" objectives of the symposium, the data presented below will emphasize data collected during weapons testing programs and up to one year following fallout contamination of an environment.

During the course of these studies many kinds of environments have been sampled varying from the semiarid desert valleys, to juniper and piñion pine covered slopes, to relatively rich agricultural areas. By and large the sampling has emphasized the study of natural environments relatively unaffected by human exploitations. In these native, stable communities the occurrence of fission products originating from fallout have been documented as they occur in the various components of the environment. The particular components studied during weapons testing programs have been air, soil, plants, native rodents, and fallout. From these data, collected serially over a period of time, the cycling of bomb debris may be followed as the contamination passes from one component of the environment to another.

The kangaroo rat, genus *Dipodomys*, and the jack rabbit, genus *Lepus*, have been used as indicators of the biological availability of radioactive fallout to other mammals. These animals are abundant in most areas and are easily collected by trapping or shooting. The kangaroo rat is of particular interest because of its sedentary nature. In other words, the body burden of fission products in any particular rat is the result of that animal living its entire life within one or two hundred feet of the point of collection. Therefore, knowledge of the conditions of contamination within this area provides us with parameters for estimating the biological significance of any particular fallout condition.

The plant species and plant parts which go to make up the primary forage of the kangaroo rat and jack rabbit in any particular area are the ones chosen for documenting the occurrence of fallout materials in or on plants.

Experience has shown that radioactive fallout originating from continental weapons tests tends to remain in the surface inch or two of soil in

undisturbed areas for a period of years, at least within the distances studied thus far. The assessment of fallout on natural areas, therefore, is expressed in terms of activity per unit area. Fallout contamination of soil is determined from samples taken from either one square foot or one quarter square foot areas depending upon the objectives of the experiment. Soils data may seem out of place in a symposium on biological material. However, it will be shown that the biological fate of radioactive fallout is dependent to a large extent *not* upon total fallout deposited in an area but upon some fraction of the total. Soil sampling provides us with a method of characterizing the total fallout against which data we can compare the biologically significant fraction of bomb debris.

This latter point is exemplified in Figure 1 which compares the amount of fallout from a single detonation on soil as compared to the amount of fallout on plant material as a function of the distance of the sampling site from Ground Zero one day following contamination. It will be noted that activity per unit weight of dried plant material compares quite favorably with the activity contained in the less than 44-micron particle size fraction of soil as opposed to total soil contamination. Note also that the degree of plant contamination in this particular case appears to increase, or, conservatively, to stay the same over the major portion of the 80 miles distance studied. The significance of these observations are two-fold. First, animals grazing in these fallout areas and feeding upon these forage plants will not be ingesting gross fallout but rather a specific fraction (the less than 44-micron size group) which has been trapped by the plant acting as a selective fallout collector. Second, since the degree of plant contamination tends to remain the same over a very great distance the internal dose to animals grazing these areas will also be similar and the potential hazard similar over a great portion of the fallout pattern.

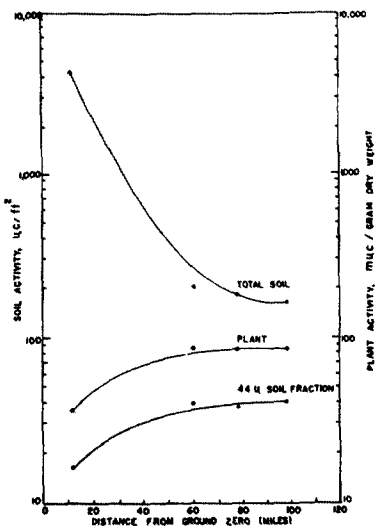

FIGURE 1.—*The relative degree of radioactive fallout contamination on soils and plants resulting from a single detonation expressed as a function of the distance of the sampling site from Ground Zero [1].*

Figure 2 shows a special preparation developed for the study of the characteristics of fallout material contaminating plants. In the field, plant leaves are carefully placed on gummed paper and backed with blotter material and dried. In the laboratory, an autoradiogram is made which serves as a map for the location of specific particles on the plant leaves. Detailed analysis of many of these preparations support the conclusion that the less than 44-micron diameter fallout particles are the principal source of radioactivity in forage material samples within a period of weeks following fallout contamination. It has not been possible to distinguish between external contamination and metabolized fission products in range plants. Experiments in which soil flats have been exposed to fallout and subsequently cultivated in the greenhouse do show that fission products (particularly Sr^{89}) are biologically available from fallout and will be accumulated in the plant parts [2].

Figure 3 is a photograph of the dried pelt of a

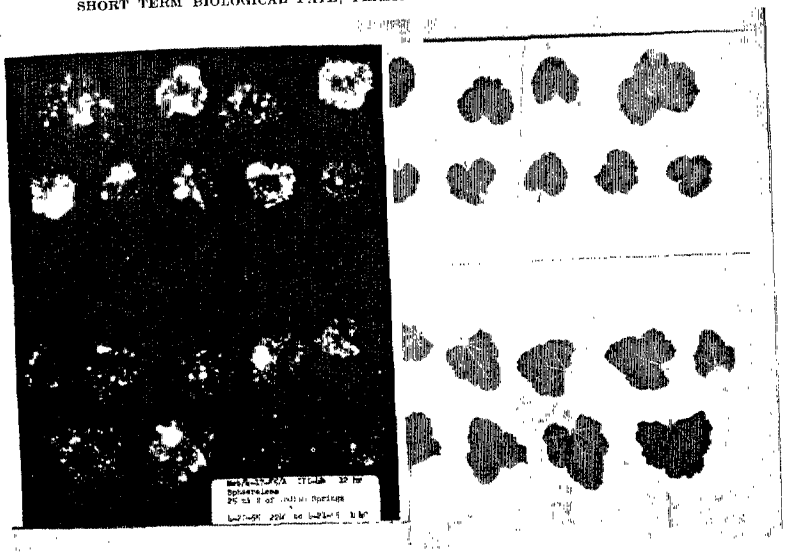

FIGURE 2.—*A preparation for the study of fallout particle retention by leaves showing the leaf preparation and the autoradiogram resulting from exposure of the preparation to X-ray film [1].*

kangaroo rat sampled from a fallout area and of the autoradiogram resulting from a one-half hour exposure of the pelt to X-ray film. This animal was collected approximately 12 miles from Ground Zero about 24 hours after fallout. Although the degree of fallout contamination is startling, it is interesting to note that we have found no indication of radiation burns or damage in the plants or rodents collected from these, or more distant, areas. Beta burns resulting from fallout particles have been verified, however, on livestock and deer grazing within 20 miles of Ground Zero.

In regard to metabolized fission products, it is indeed fortunate that, in terms of the physiological requirements of plants and animals, relatively few isotopes are of real biological significance. However, whether the biological hazards are interpreted in terms of radioiodine or radiostrontium or some other specific isotope, the fact remains that many other fission products are apparently present in tissues for a limited period as a result of fallout contamination. As long as the fundamental rule of radiation biology remains "that radiation is primarily a destructive force in living systems", then we are committed to learning more of this material that we must "learn to live with," whatever its half-life.

The relationship between the biological fate and persistence of radioactive fallout to time, the location of the biological material within the fallout pattern, and the behavior of the isotopic precursors of the particular fission products under concern are parameters which require special consideration and defy anything but an arbitrary division of the fallout phenomenon into short term and long term effects.

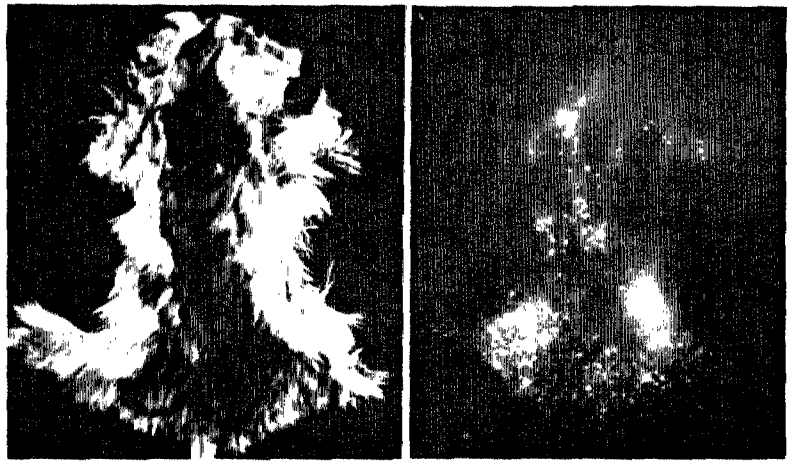

FIGURE 3.—*A dried pelt taken from a kangaroo rat exposed to fallout 16 miles from Ground Zero and the resulting autoradiogram showing the occurrence of radioactive fallout particles* [3].

Figure 4 summarizes the persistence of fission products in various tissues serially sampled from a natural population of kangaroo rats over a period of 90 days following a single fallout contamination in the spring of 1953. The decrease in tissue burden does not deviate markedly from the theoretical decay of mixed fission products based upon the $t^{-1.2}$ decay constant. This suggests that the tissue burdens are made up of mixed fission products in equilibrium with the concentration of fission products in the environment. During this time period there appears to be little evidence of biological concentration of fission products in terms of gross beta gamma activity. Figure 5, however, shows the gradual buildup of radioiodine in the thyroid of kangaroo rats and jack rabbits serially sampled from a fallout area located 12 miles from Ground Zero during the spring of 1955. Sampling was discontinued 15 days after fallout with the concentration of radioiodine still rising. It can be anticipated, however, that the accumulation of iodine was nearing its peak. This buildup of thyroid activity corresponds to similar phenomena described at Hanford Works and is considered to reflect the time necessary for the iodine in the thyroid and in the food supply to reach equilibrium. The problem is further complexed by the identification of I^{133} as the primary contributor to thyroid activity during the first day or two following fallout following which time I^{131} becomes dominant.

Figure 6 shows the influence of the location of the sampling site within the fallout pattern to the biological accumulation of fission products. In this case the accumulation of fission products is plotted against the distance of the sampling site from Ground Zero along the midline of fallout. As might be expected the tissue burdens generally appear to drop off with distance but not as sharply as does the total fallout. Note the striking deviation of the femur and kidney data from the other tissues. Figure 7 presents similar data from two separate events showing the increase in radioactive content of the thyroid as a function

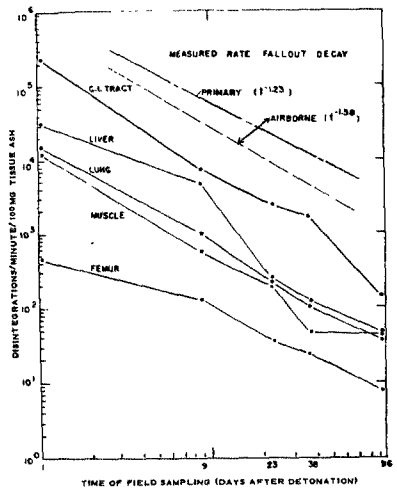

FIGURE 4.—*The occurrence of fission products in tissues sampled from a natural population of kangaroo rats living 19 miles from Ground Zero in a fallout contaminated environment expressed as a function of time after fallout* [3].

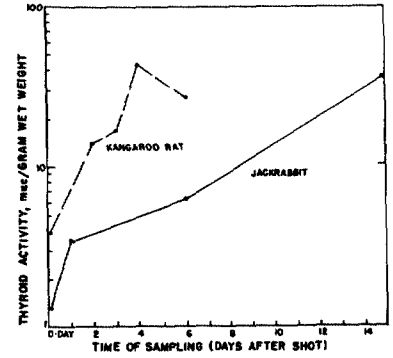

FIGURE 5.—*The occurrence of radioiodine in the thyroid of native animals serially sampled from a fallout contaminated area 12 miles from Ground Zero and on the approximate midline of fallout* [1].

of distance from the sampling site with a peak concentration at 60 miles from Ground Zero. Note that this distance is the same for both fallout patterns even though the conditions of detonation were very different. These data just presented are all from samples collected within 24 to 48 hours following fallout.

Figure 8 shows the interaction of time, and the position of the sampling site on the biological fate of fallout. Following the 1955 test series, two residual fallout patterns were defined and samples taken along the midline of contamination. The results from one pattern are shown in Figure 8 since these data are more complete and representative of both residual fallout patterns. The environmental contamination, a measure of gross residual fallout contamination, decreased sharply with distance. The gross beta gamma activity in jack rabbit bones sampled along the midline of residual fallout increased out to 130 miles and then decreased slightly and leveled off. The radiation levels above normal that occurred in the bone ash was accounted for by the presence of radiostrontium. The peaking of activity at 130 miles appeared more specifically to be attributable to the relatively heavy concentration of Sr^{90}.

This was not the first time that this phenomenon had been observed. In May 1954, *one year* following the 1953 Test Series, another residual fallout pattern was studied to a distance of 130 miles from Ground Zero, with the results that are summarized in Figure 9. Once again soil contamination was shown to fall off sharply while the burden of radiostrontium in jack rabbit bones increased to a maximum at 130 miles from Ground Zero.

Remember that with respect to the iodine data the maximum value occurred at 60 miles. The maximum in the strontium data occurs at 130 miles. Another parameter can be assumed as the time necessary for the parent fission products to decay into the daughter products

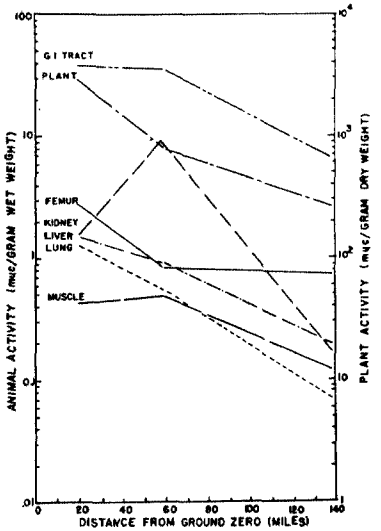

FIGURE 6.—*Fission product distribution in tissues from kangaroo rats sampled after two nights grazing (D, D+1), in a fallout area, expressed as a function of the distance of the sampling site from Ground Zero* [1].

which are measured in our samples. The radioactive life of the precursor and its chemical characteristics will determine how the daughter product is finally distributed as fallout material. The question as to the fate of other specific fission products such as cerium, cesium, ruthenium, and zirconium are under study.

In summary we can describe the biological availability of radioactive fallout as follows: First, it was found during participation in the weapons testing program that the predominant size of fallout particles greater than 100 microns in diameter decreased with distance from Ground Zero while the less than 100 micron material did not decrease but remained the same or increased with distance up to 200 miles from Ground Zero [6]. Furthermore, the smaller size material tended to be more soluble, and, therefore, potentially more available to the biological cycle [7]. Second, the majority of particles retained by foliage were below 44 microns in diameter having an average size of approximately 20 microns.

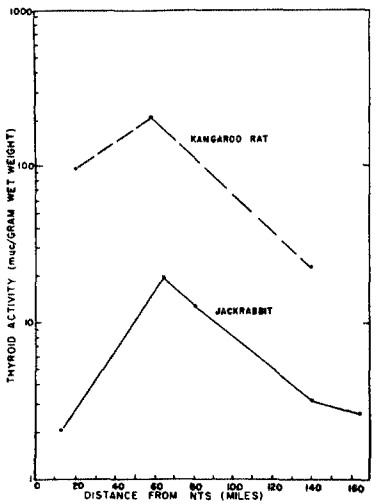

FIGURE 7.—*The relationship of distance to the occurrence of radioiodine in the thyroids of kangaroo rats contaminated by radioactive fallout, and to the occurrence of radioiodine in the thyroids of jackrabbits contaminated by a different fallout* [1].

A feasible explanation then is that the accumulation of radiostrontium, for instance, is related to particle size and that because the plant acts as a selective collector of very small fallout particles, the intake of radioactive debris by animals during grazing tends to be similar over a great distance and appears to be independent of the total fallout. The amount of any specific isotope present is dependent upon the physical and chemical behavior of its isotopic precursor during fallout particle formation. Therefore, the amount of any specific isotope at any particular location within the fallout pattern will be highly variable, and the occurrence of areas in which the biological accumulation of that isotope is high are to be anticipated.

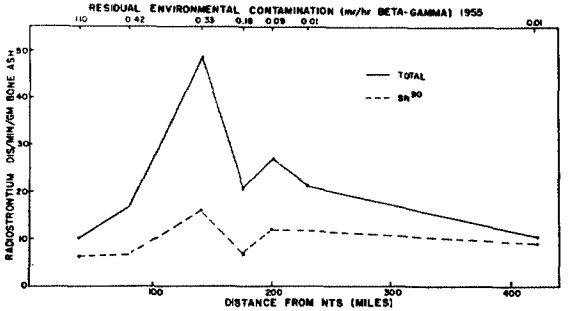

FIGURE 8.—*The occurrence of radiostrontium in the bones of jackrabbits sampled in fall, 1955 from the midline of residual fallout contamination as compared to distance of the sampling site from NTS, and the degree of residual environmental contamination* [4].

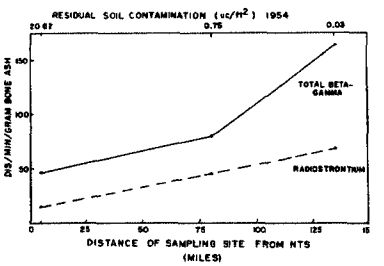

FIGURE 9.—*The occurrence of radiostrontium in the bones of jackrabbits sampled in spring 1954 from the midline of residual fallout contamination as compared to the distance of the sampling site from NTS, and the degree of residual soil contamination* [5].

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DISCUSSION

R. G. Lindberg and K. H. Larson

Dr. STANNARD (University of Rochester). One very simple question. Were these figures for particle size on a mass basis average or number basis?

Dr. LINDBERG. The technique has been to take a soil sample from a known area, and using standard soil methods break it down to the particle size in the soil. So the total activity is an expression of the activity in a particular size fraction and does not reflect the total number of particles involved nor any characteristics of that particle except size. The soil acts as a carrier for the separation.

RESIDUAL CONTAMINATION OF PLANTS, ANIMALS, SOIL, AND WATER OF THE MARSHALL ISLANDS TWO YEARS FOLLOWING OPERATION CASTLE FALLOUT

Presented by H. V. WEISS

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The object of this study was to determine the persistence and fate of radioactive material in the biological systems and in the physical environment of those Marshall Islands contaminated by fallout from the 1 March 1954 nuclear detonation. For this purpose a resurvey of the islands was conducted in February 1956 by a group of scientists from the Naval Radiological Defense Laboratory. Specimens of animals (land and marine) and birds, and samples of plants, soil and water were collected for analysis. Radio assays for gross beta and gamma activity were conducted and in addition radiochemical determination of individual fission products and induced activities were made.

A few weeks after the 1954 incident a survey was made of the contaminated atolls, [1] and soil, water, and biological specimens were collected from Rongelap and Utirik. These samples were analyzed and the results were given in the Operation CASTLE, Project 4.1 report [2]. Soil and water samples contained microcurie amounts of activity; barely detectable quantities were found in plants. Approximately 1 year following the nuclear detonation, a survey of the islands indicated that the activity was present in metabolic systems and was still in the environment at lower but significant levels [3]. The present study, conducted 2 years post-detonation, provides further data on the persistence and distribution of the fallout activity. From these data an evaluation can be made of the potential hazard from the ingestion of contaminated materials.

The gross beta activity of the plant specimens analyzed is recorded in Table 1 according to the island from which the sample was recovered. The data were corrected for the counting efficiency of Sr^{90} and presented as corrected counts per minute per kilogram of wet sample. Empirical corrections for self-absorption were not applied because the activity of most samples was so low as to prevent such evaluation with expediency. Furthermore since the nuclide composition varied among plants and even within different sections of the same plant, a blanket correction was impossible.

Portulaca was many times more active than other plant specimens recovered from the same island. Leaves of plants were generally more active than their fruit counterpart. The fact that surfaces of leaves were not decontaminated prior to analysis may account at least in part for this difference.

Three stages of coconuts—green, ripe, and sprouting nut—were analyzed. Both green and ripe pandanus keys were examined. No distinct differences between the stage of growth and activity were discernible.

Where possible the meat, milk, shell, and husk of coconuts were analyzed separately. Within the limits of the analysis, the activity appeared equally distributed among these fractions.

The order of plant activities relative to the island from which they were recovered was: Gejen > Eniwetak, Eniaetok > Rongelap > Sifo, Utirik > Likiep. These results agree well

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TABLE 1.—GROSS BETA ACTIVITY IN PLANT, WATER AND SOIL SAMPLES[a]

Plant	Part	PLANTS[b] (c/m/kg x 10^{-5})						
		Gejen	Eniwetak	Eniaetok	Rongelap	Sifo	Utirik	Likiep
Portulaca	Whole plant	87. 4	19. 2	3. 05	1. 26	.....	1. 71	1. 33
Arrowroot	Stems, leaves	11. 0	4. 5	. 32	. 25	0. 21	.....	. 03
	Tubers	2. 32	. 57	. 60	. 55	. 08	. 14	. 03
Pandanus	Air root	2. 87	. 17	1. 05	. 32	. 96	. 08	. 02
	Leaves	2. 64	1. 02	5. 26	. 38	. 15	. 21	. 03
	Green keys	1. 27	. 37	. 70	. 22	. 10	. 09	. 03
	Ripe keys	.....	.....	. 53	. 17	.....	. 07	. 02
Papaya	Ripe	.....	.....	.....	. 12	.....	. 11	.....
	Green	.....	.....	.....	. 25	.....	. 09	. 04
	Leaves, trunk	.....	.....	.....	. 09	.....	. 16	. 06
Ripe coconut	Milk	2. 87	.....	.....	. 54	. 63	. 12	. 57
	Meat	1. 90	. 36	1. 97	. 24	. 17	. 08	. 06
	Shell	4. 98	. 38	. 72	. 44	. 28	. 06	. 02
	Husk	1. 83	. 65	1. 57	1. 31	. 77	. 21	. 09
Green coconut	Whole	3. 1	.....	.....	.....	.....	.....	.....
	Milk	.....	. 29	. 11	. 05	. 13	.....	. 05
	Meat	.....	. 33	. 25	.....	. 08	. 07	. 02
	Shell	.....	.....	. 80	.....	. 37	. 08	. 09
	Husk	.....	.....	. 48	. 12	. 11	. 11	. 02
	Shell, husk	.....	. 11	.....	.....	.....	.....	.....
Sprouting coconut	Milk	.....	1. 61	. 76	. 79	. 71	. 11	. 09
	Meat	.....	. 38	. 40	. 12	. 30	. 07	. 06
	Shell	.....	. 29	. 41	. 35	. 18	. 04	. 02
	Husk	.....	. 73	1. 57	. 88	. 68	. 26	. 07
Coconut	Leaves	.....	15. 4	. 86	.....	. 84	4. 7	1. 66
	Frond	.....	. 94	. 51	.....	. 23	. 09	. 11
	Leaves, frond	1. 48	.....	.....	.....	.....	.....	.....
Banana	Fruit	.....	.....	.....	.....	.....	.....	. 06
	Bark	.....	.....	.....	.....	.....	.....	. 07
Taro	Leaves	.....	.....	.....	.....	.....	.....	. 18
	Leaves, stalks	.....	.....	.....	.....	.....	.....	. 06
	Tuber, roots with soil	.....	.....	.....	.....	.....	.....	. 19

[a] All counts were corrected for the counting efficiency of Sr^{90}-Y^{90}.
[b] Gross beta activity of plant samples was determined in April 1956 and that of soil and water in May 1956.

with the activities of the respective soils as shown in Table 2.

The gross beta activity of well, cistern, ocean, and lagoon water is shown in Table 2. The activities were either imperceptible or of a low order of magnitude.

To describe the downward movement of the activity, profile soil samples were obtained in increments to a depth of 56 inches. As shown in Table 2, the greater part of the beta activity appeared fixed to the upper surface of the soil; the remaining part diminished sharply and progressively at deeper levels. The bulk of the activity appeared to be firmly absorbed to the soil since it resisted the downward migration of the heavy rains to which these islands are subject.

Table 3 lists the gamma dose rates found on the island survey; levels observed 1 year before are included. The gamma activity was reduced over the 12-month period by 74±8 percent. Calculations based on the Hunter-Ballou curves for beta decay of mixed fission products [4] predict that 80 percent of the gamma activity

TABLE 2.—GROSS BETA ACTIVITY IN PLANT, WATER AND SOIL SAMPLES[a]

	Gejen	Eniwetak	Eniaetok	Rongelap	Sifo	Utirik	Likiep
SOURCE	WATER[b] (c/m/liter x 10^{-5})						
Cistern	.....	.....	.....	0. 008	.....	NDA[c]	.....
Well	.....	.....	NDA	.....	.....	0. 1 . 03 NDA	NDA
Ocean	NDA	NDA	0. 06	. 06	0. 09	NDA	0. 08
Lagoon	NDA	NDA	NDA	NDA	. 08	. 09	NDA
DEPTH (IN.)	SOIL[b] (c/m/kg x 10^{-5})						
0–1	3470	34. 8	6. 43	7. 00	4. 97	4. 43	NDA
12	.....	.....	.....	. 70	.....	.....	.....
18	0. 80	.....	NDA	.....	.....	.....	NDA
24	.....	NDA	.....	.....	. 04	. 51	.....
33	1. 33	.....	.....	NDA	.....	.....	NDA
36	.....	.....	.....	.....	.....	.....	.....
44–45	.....	.....	0. 07	.....	.....	.....	.....
48	.....	NDA	.....	.....	NDA	.....	.....
55–56	.....	.....	.....	.....	.....	. 70	.....

[a] All counts were corrected for the counting efficiency of Sr^{90}-Y^{90}.
[b] Gross beta activity of plant samples was determined in April 1956 and that of soil and water in May 1956.
[c] NDA indicates no detectable activity.

TABLE 3.—AVERAGE GAMMA DOSE RATES FROM PREVIOUS AND CURRENT SURVEYS

Island	11 months (mr/hr)	23 months (mr/hr)	Remaining activity (percent)
Likiep	0. 04	<0. 05	.....
Utirik	. 14	. 05	35
Eniwetak	. 7	. 16	23
Rongelap	. 7	. 09	13
Eniaetok	2. 4	. 28	12
Kabelle	4. 2	. 96	23
Gejen	5. 4	1. 5	28
Average	.....	.....	26

is lost by radioactive decay over this interval. This decay was obviously the significant factor in reduction of the gamma field rather than the leaching of nuclides to deeper layers and their eroding into the adjacent waters.

The long-lived isotopes of mixed fission products, which present the greatest internal radiation hazard to human inhabitants of a contaminated area, were analyzed in plant, soil, and water samples. These isotopes were the total rare earths, Sr^{90}, Cs^{137}, and Ru^{106} and comprised the total detectable fission product activity remaining 2 years after the nuclear detonation.

In Table 4 the relative contribution of the nuclides recovered from plant, soil and water are recorded. The primary contaminating isotope in coconuts, papaya fruit, pandanus keys and arrowroot tubers was Cs^{137}. Significant quantities of the rare earth components (16 to 18 percent) were recovered from papaya and

TABLE 4.—AVERAGE RELATIVE COMPOSITION OF NUCLIDES IN PLANTS, SOIL, AND WATER

Source		Number of samples averaged	Relative composition (percent)			
			Cs^{137}	Total rare earths	Sr^{90}	Ru^{106}
Plant:	Part	*Plants*				
Portulaca	Whole	1	48.9	39.2	11.8	
Papaya	Fruit	1	79.8	17.8	2.5	
Coconut	Husk	3	98.2	1.1	.7	
	Meat	2	98.9	.05	1.0	
	Shell	2	99.5	.4	.1	
	Milk	1	99.6	.2	.2	
Pandanus	Leaves	2	8.3	86.5	.4	5.1
	Keys	2	92.6	2.2	5.5	
	Leaves	2	72.7	13.3	5.1	8.9
	Air root	2	88.9	10.3	.8	
Arrow root	Tuber	1	75.4	16.8	1.0	6.8
	Leaves	1	11.7	83.9	3.0	1.4
	Soil					
Depth, 0–1 in.		2	.34	83.8	5.6	10.0
Source:	*Water*					
Cistern		2		64.4	35.6	
Well		2		100	0	
Lagoon		2		94.5	5.5	
Ocean		2		100	0	

arrowroot tubers and only a small fraction from coconuts and pandanus keys. The Sr^{90} concentration in these specimens was uniformly low.

The nuclide composition of the leafy structures in the coconut palm and the arrowroot plant differed markedly from the respective nut and tuber. These structures accumulated the rare earth isotopes in exceedingly greater concentration than Cs^{137}.

Table 4 shows further that plant leaves contained varying percentages of Ru^{106} and that the concentration of this isotope represented only a small fraction of the total activity.

In portulaca, a widely distributed plant, the nuclide composition was 49, 39 and 12 percent Cs^{137}, rare earths, and Sr^{90}, respectively.

Despite the inactivity of the water samples, rare earth and Sr^{90} determinations were performed since self-absorption as well as the size of aliquot used may have obscured the activity. Cs^{137} and Ru^{106} were not determined because self-absorption does not play an important role in the detection of these gamma-emitters. The results of these analyses are shown in Table 4. With the exception of a sample of cistern water which had a significant quantity of Sr^{90}, the observed activity was attributable to the rare earths.

With regard to soil, the average of two complete assays gave 84 percent rare earths, 10 percent Ru^{106}, 5 percent Sr^{90} and less than 1 percent Cs^{137}.

Where comparisons were available, the relationships among nuclides in the current survey in general agreed with those previously reported [3]. The only sharp difference was the higher percentage of Cs^{137} in the one papaya analyzed in the present study.

The sunshine units are recorded in Table 5 for the plant, water and soil samples analyzed.

Of the plant samples examined, portulaca had the highest sunshine units; values were 6,140 and 25,000 for the two specimens analyzed. In coconuts the activity of meat, shell and milk was not statistically significant, whereas the value for husks ranged from 1,200 to 4,000. Pandanus keys and pandanus air root values also fell within this range. Arrowroot leaves, stalks and tubers were significantly lower, ranging from 86 to 780 sunshine units.

The sunshine units in the 0 to 1 inch layer of soil on 5 islands were 17 to 92; the exception, Gejen, had a value of 7,000.

Strontium-90 was not detectable in most water samples; however 4 samples showed some activity with sunshine units between 150 to 200. A sample of cistern water from Rongelap, the notable exception, had a value of 10,000.

Noteworthy is the fact that the activity in portulaca, coconut husks, pandanus keys and air roots, as well as a sample of potable water, exceeded the maximum tolerance level of Sr^{90}.

TABLE 5—SUNSHINE UNITS OF PLANT, WATER AND SOIL SAMPLES

Sample	Island	SOILS		Sunshine Units (2.2 d/m Sr^{90}/g Ca)
		Calcium in kg of Soil (g)	Sr^{90} (d/m/kg)	
Depth (0–1 in.)	Rongelap	316	$3.3 \times 10^4 \pm 1.3 \times 10^3$	47±2
	Gejen	341	$5.26 \times 10^6 \pm 5.2 \times 10^3$	$7 \times 10^3 \pm 70$
	Eniaetok	352	$2.1 \times 10^4 \pm 2.2 \times 10^3$	28±3
	Sifo	350	$1.3 \times 10^4 \pm 1.0 \times 10^3$	17±1
	Eniwetak	360	$5.8 \times 10^4 \pm 2.3 \times 10^3$	73±3
	Utirik	268	$4.8 \times 10^4 \pm 3.0 \times 10^3$	92±6
		WATER		
		Calcium in Liter (mg)	Sr^{90} (d/m/liter)	
Cistern	Rongelap	48	1180±10	$1.1 \times 10^4 \pm 230$
	Utirik	61	20±14	147±104
Well	Utirik	88	39±10	201±54
	Utirik	80	NDA	0
	Eniaetok	2300	NDA	0
Ocean	Rongelap	352	NDA	0
	Utirik	408	NDA	0
	Eniwetak	402	NDA	0
Lagoon	Rongelap	456	190±68	188±68
	Eniwetak	137	NDA	0
	Utirik	441	204±150	208±150

NDA indicates no detectable activity.

TABLE 5.—SUNSHINE UNITS OF PLANT, WATER AND SOIL SAMPLES - Continued

Sample	Island	PLANTS			
		Sample weight (g)	Calcium content (mg)	Sr^{90} (d/m/sample)	Sunshine units (2.2 d/m Sr^{90}/g Ca)
Portulaca	Eniaetok	223	178	10000±100	2.58 x 10^4±250
	Gejen	23	398	5380±106	6140±120
Papaya	Rongelap	240	338	240±33	322±44
Coconut Husk	Rongelap	200	162	340±28	950±76
	Eniaetok	23	58	150±24	1200±190
	Gejen	360	47	420±24	4060±240
Coconut Meat	Rongelap	450	28	110±60	1801±960
	Eniaetok	160	40	18±29	200±320
	Gejen	190	20	28±23	635±520
Coconut Shell	Eniaetok	90	16	25±18	706±500
	Eniaetok	120	8	NDA	0
	Gejen	85	23	NDA	0
Coconut Milk	Gejen	140	20	41±21	955±500
Coconut Leaves	Eniwetak	35	69	197±37	1300±250
	Utirik	36	163	NDA	0
Coconut, Whole	Gejen	170	195	157±22	3600±520
Arrowroot Tuber	Eniaetok	305	1140	250±26	103±10
	Sifo	280	383	73±16	86±19
	Gejen	103	114	196±35	780±140
Arrowroot Leaves and Stalks	Gejen	15	385	290±44	340±50
Pandanus Keys	Eniaetok	180	86	1060±50	5600±280
	Eniaetok	215	134	420±44	1400±150
Pandanus Leaves	Eniaetok	10	65	460±41	3200±300
	Gejen	32	43	NDA	0
Pandanus Air Root	Eniaetok	46	23	20±33	390±650
	Gejen	30	14	105±27	3360±840

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DISCUSSION

H. V. Weiss

(Please see discussion on pages 217 to 218.)

PERSISTENCE OF RADIOACTIVE CONTAMINATION IN ANIMALS OF MARSHALL ISLANDS TWO YEARS AFTER OPERATION CASTLE

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An unique opportunity for study of the internal radiation hazard associated with the contamination of a large land mass was afforded when several of the Marshall Islands were contaminated by fallout from the nuclear detonation of March 1, 1954. Within a month of the accident, numerous land animals, birds and marine specimens, as well as samples of plants, soil and water were collected for analysis of the concentration and distribution of radioactive material. On the basis of these findings the initial hazard to human beings from exposure to internal radiation resulting from the ingestion and inhalation of radioactive fallout was estimated.

In order to ascertain the degree of hazard associated with the residual contamination, and thus to assess the habitability of the contaminated areas, resurveys of the Islands were conducted at 1 and 2 years. Data on the physical availability of the contaminant in the environment and the biological availability in plant foods has been presented by Dr. Weiss. In addition, however, knowledge of the biological transport of these radionuclides, especially Sr^{90}, through the food chain is required. It is readily apparent that one cannot deduce, from data on the physical environment alone, what will be the ultimate deposition in the skeleton of animals living in this area.

Readily detectable levels of radioactivity in land and marine animals of the Marshall Islands contaminated by the 1954 nuclear detonation were measured in February 1956. A summary of the residual radioactive contamination at 2 years in the tissues of 85 fish and marine invertebrates from the various island lagoons, expressed in terms of gross beta and gamma activity is presented in Table 1.

Considerable variation was observed in the concentration of activity per unit weight of individual fish and marine invertebrates from the same area as well as from different geographic locations. Part of this variation may be due to differences in feeding habits, but no correlation between the level of radioactivity and the eating habits of the fish (carnivorous, herbivorous, omnivorous) could be ascertained. Other factors such as currents and localized concentrations of radionuclides may also play a role in determining concentrations of residual activity in the lagoon fish.

Fish and marine invertebrates caught in the northern section of the Rongelap Lagoon had the same level of beta activity but twice the gamma activity of fish from the southern section of the lagoon. This ratio of activity in marine invertebrates between the north and south ends of the lagoon was considerably lower than that observed 1 year following the detonation. This finding suggests a redistribution of activity from the higher concentration originally existing in the northern end of the lagoon. The pattern of the 1954 fallout was such that the activity on the northernmost islands was tenfold higher than on Rongelap Island, at the southern end of the atoll.

The internally deposited activity in the

TABLE 1.—SUMMARY OF BETA AND GAMMA ACTIVITY IN FISH AND MARINE INVERTEBRATES

Island	Fish			Crabs			Clams			Snails		
	Number of samples	Activity (d/m/kg x 10^{-4})		Number of samples	Activity (d/m/kg x 10^{-4})		Number of samples	Activity (d/m/kg x 10^{-4})		Number of samples	Activity (d/m/kg x 10^{-4})	
		β	γ		β	γ		β	γ		β	γ
RONGELAP ATOLL:												
North: Gejen	8	24.5	78.8	2	28	87				4	648	613
Kabelle	10	14.9	55.4							1	17.7	43.9
Central: Eniaetok	5	19.3	45.1	1	4.5	14.1	1	4.5	5.8			
South: Rongelap	5	17.7	32	6	23.4	24.5	2	23	56	2	31	51
RONGERIK ATOLL:												
Enjwetak	8	2.2	7.8	1	2.8	18.3						
ARLINGANE ATOLL:												
Sifo	6	4.5	22.7	3	21.9	14.5	1	6.4	15.0			
UTIRIK ATOLL:												
Utirik	8	1.6	2.1							3	.006	2.8
LIKIEP ATOLL:												
Likiep	8	2.6	1.3									

lagoon fish was only very roughly proportional to the external radiation dose over the adjoining island.

Crabs and clams were found to have a residual concentration of beta-emitting radionuclides of about the same level as fish from the corresponding locality. This is in contrast to the larger differences noted between crabs and clams as compared to fish at 1 year post detonation.

Snails from Gejen had considerably higher concentrations of activity than fish from the same locality, as was noted in the 1-year resurvey. The higher level of activity of the snails may be related to their habit of feeding on the bottom of the lagoon where higher concentrations of radionuclides were found.

The internal distribution of radioactivity in the tissues of fish (primarily carnivores) collected in the various lagoons indicated that an average of 20 percent of the total beta and gamma activity was found in the skeleton (Table 2). The head contained an average of 30 percent of the total beta and 21 percent of the gamma activity. Muscle contained approximately 14 percent of the total beta and gamma activity. The activity of the viscera and contents varied considerably, but contained on the average about 33 percent of the total beta activity and 16 percent of the total gamma activity. The remainder of the activity was found on the skin and gills. The internal distribution of activity, particularly the muscle activity concentration, was very similar to that found in the fish collected and analyzed at one year post detonation.

The results of the radiochemical analyses for specific radionuclides are presented in Table 3. The most important finding is the very high percentage of the total activity in fish which is contributed by Zn^{65}. The manner in which this induced activity is concentrated has not been determined. The Zn^{65} in fish is distributed fairly evenly among the various tissues. The Zn^{65} was not found in clams, crabs, or snails, with the exception of one helmet snail from Kabelle Island.

The rare earth group of fission products constituted a small percentage of the total beta activity in clams and fish. The rare earth elements as a group do not appear to be selectively localized. The rare earth activity of the crabs was high, an average of 20 percent of the total beta activity. Snails concentrated the largest amounts of rare earth elements.

The Sr^{90} concentration was very low, contributing generally a fraction of 1 percent of the total beta activity. The Sr^{90} content is of particular importance, since it is the radionuclide of greatest potential hazard. The Sr^{90} hazard derives principally from its long

TABLE 2.—DISTRIBUTION OF GROSS BETA AND GAMMA ACTIVITY IN TISSUES OF FISH

Island / Fish	Wet wt (g)	Radioactivity (d/m/tissue x 10^{-3})																	
		Total		Skin		Head		Muscle		Bone		Gill		Viscera					
		β	γ	β	γ	β	γ	β	γ	β	γ	β	γ	β	γ				
RONGELAP ATOLL, SOUTH:																			
Rongelap: Goat	218	8.8	15.5	0.2	2.4	0.45	3.3	1.1	2.1	1.5	2.7	0.6	2.2	4.9	2.8				
Rongelap: Grouper	452	5.2	5.7	.4	.3	.8	.7	.4	.3	1.4	2.6	.3	.3	1.9	1.4				
Average		7.0	10.6	.3	1.3	.63	2.0	.8	1.3	1.5	2.7	.5	1.3	3.4	2.1				
Percent of total activity		100	100	4.2	12.1	8.8	18.7	11.2	12.1	21.0	25.2	7.0	12.1	47.7	19.6				
RONGELAP ATOLL, NORTH:																			
Gejen	1,154	26.3	87.0	1.0	11.8	6.6	24.7	3.4	16.9	5.5	13.7	1.7	2.1	6.1	15.9				
Kabelle	735	12.3	16.5	1.0	11.2	4.5	1.9	1.0	.7	2.4	4.4	.3	1.1	2.9	6.3				
Kabelle	1,957	24.8	71.3	1.1	8.9	8.5	20.9	2.4	6.6	7.0	26.4	.8	2.7	5.0	8.8				
Average		21.1	58.9	1.0	10.6	6.5	15.8	2.9	8.0	5.0	14.5	1.0	2.0	4.7	10.3				
Percent of total activity		100	100	4.8	17.3	30.8	25.9	13.7	13.1	23.7	23.7	4.8	3.3	22.3	16.9				
ARLINGANE ATOLL:																			
Sifo	640	3.2	38.9	.3	5.9	.7	9.9	.6	6.2	.5	10.6	.1	2.7	.9	3.6				
Percent of total activity		100	100	9.7	15.2	22.5	25.4	19.3	15.9	16.1	27.2	3.2	7.0	29.0	9.4				
RONGERIK ATOLL:																			
Enjwetak	387	.41	2.0	.02	.35	.23	.55	.04	.27	.06	.39	.02	.03	.04	.4				
Percent of total activity		100	100	4.9	17.3	55	27.2	9.8	13.4	14.6	19.3	4.9	1.0	9.8	18.8				
UTIRIK ATOLL:																			
Utirik	925	.95	.87	0	.24	0	.09	.15	.22	.13	.13	0	.04	.98	.2				
Percent of total activity		100	100	0	27.6	0	10.3	22.7	25.3	19.7	15.0	0	4.6	57.5	17.2				
LIKIEP ATOLL:																			
Likiep	453	1.1	2.2	0	0	0	.02	.1	.2	0	0	0	0	.1	.2				
Percent of total activity		100	100	0	0	0	.9	9	9	0	0	0	0	91	90				

TABLE 3.—RADIOCHEMICAL ANALYSIS OF BIOLOGICAL SPECIMENS FROM RONGELAP ATOLL

Sample No.	Sample	Tissue	Wet wt. (g)	Ca (mg)	Beta activity (d/m sample x10⁻³)	Gamma activity (d/m sample x10⁻⁴)	Nuclide	Nuclide activity (d/m sample x10⁻³)	Percent of total activity	Sunshine units [a]
RONGELAP ISLAND										
1502C	Goat Fish	Bone	29	860	1.5	217	R. E.[b]	NDA [c]	0	
							Sr^{90}	11±1.7	7.3	587±90
							Zn^{65}	240	89	
		Viscera	10	37.5	4.9	2.8	R. E	0.68	.14	
							Sr^{90}	NDA	0	0
							Zn^{65}	250	89.3	
		Skin	28	337	.2	2.4	R. E	2.5	12.5	
							Sr^{90}	0.34±0.26	1.7	45±34
							Zn^{65}	230	95.8	
		Muscle	87	111	1.1	2.1	R. E	NDA	0	
							Sr^{90}	0.46±0.76	.4	189±313
							Zn^{65}	190	90.6	
1509	Killer Clam	Soft Tissue	1800	743	20	33	R. E	NDA	0	
							Sr^{90}	2.4±0.69	12	146±42
							Co^{60}	2090	63.4	
1513	Killer Clam	Soft Tissue	862	1565	31	83	R. E	77	2.5	
							Sr^{90}	83.8±0.90	2.7	2436±31
							Co^{60}	7370	89	
1520A	Langousta Crab	Soft Tissue	79	330	1.3	2.1	R. E	26	20	
							Sr^{90}	NDA	0	0
1520C	Red Eye Crab	Soft Tissue	57	2343	.75	3.8	R. E	37	49	
							Sr^{90}	0.13±0.07	.2	3±1
1520D	Red Spotted Crab	Soft Tissue	73	2900	.75	.43	R. E	15	20	
							Sr^{90}	1.28±0.18	1.7	26±3
1520B	Coconut Crab	Soft Tissue	114		3.5	3.1	Cs^{137}	25	7.4	
							R. E	0.58	16.5	
KABELLE ISLAND										
1538	Snapper Fish	Muscle	281	85	0.95	0.69	R. E	4.1	4.2	
							Sr^{90}	NDA	0	0
							Zn^{65}	58	84.2	
		Skin	89	987	1	4.1	R. E	2.4	2.4	
							Sr^{90}	0.53±0.76	.5	24±34
							Zn^{65}	380	92.7	

[a] Sunshine Unit = 0.001 pc Sr^{90}/kg Ca.
[b] R. E. = Rare Earth Group
[c] NDA = No Detectable Activity.

radioactive half-life (28 yr.) and also from its high fission yield and its availability to biological organisms. Sr^{90} levels and Sr^{90}/Ca (sunshine units) are reported for a number of samples in Table 3.

The skeletons of fish concentrated and retained the largest amounts of Sr^{90}, as would be expected from the similarity of strontium metabolism to calcium metabolism. The skeleton of a fish from Rongelap had 587 sunshine units, the highest observed in any fish. The highest number of sunshine units in any of the samples analyzed appeared in a clam from Rongelap (2.43 x 10^3 units).

In general, snails had a high number of sunshine units (276 to 502). A relatively high level of Ru^{106} (19.2 percent of beta activity) was also found in a snail from Gejen. A high level of Cs^{137} (with a 37-year half-life) was found in a coconut crab. In the analyses from previous island resurveys, Cs^{137} was the major radionuclide found in land food plants and also in the tissues of land animals. The coconuts, which had high levels of Cs^{137}, were

undoubtedly the source of the Cs^{137} activity found in the coconut crab.

The presence of Co^{60} in two samples of clams was noted for the first time in the 2-year period since the detonation. The Co^{60} accounted for the major fraction of the total activity in these samples. The Co^{60} was detected by gamma spectral analysis, and confirmed by chemical separation and absorption measurements. The ability of clams to concentrate Co^{60} selectively was verified in laboratory experiments using clams obtained locally.

Comparison of the fish and marine specimens collected immediately after detonation and 1 year later with those studied in the present report (2 years after detonation) indicate a drop in activity. The fish from the Rongelap lagoon had approximately one-fourth the activity of those analyzed 1 year postdetonation.

A rooster caught on Rongelap Island had a beta activity of 6.1 x 10^5 d/m and a gamma activity of 1.2 x 10^6 d/m (Table 4). The level of beta activity of this rooster was 40 percent of that of a rooster from the same locality analyzed at 1 year postdetonation. About 86 percent of the total activity in the body was concentrated in the skeleton.

The pattern of distribution of residual activity within the skeleton is shown in the autoradiograph of the rooster tibia (fig. 1). The activity is diffusely spread throughout the diaphysis. The concentration of activity in the diaphysis and its absence in the ends of the bone indicates that the primary deposition occurred soon after the detonation while the chickens were young and growing. The radiation dose to the skeleton from internal emitters is obviously considerably higher at this time than that to any other tissue. The muscle contained 8 percent of the beta activity, and the liver, 4 percent. The gastrointestinal tract had 1.3 percent of the beta activity, and about one-fourth of this was found in the respiratory tract.

The average activity for individual tissues of 4 rats collected on Rongelap are also presented in Table 4. The rats had a beta activity of

TABLE 4.—SUMMARY OF GROSS BETA AND GAMMA ACTIVITY IN RONGELAP ISLAND ANIMALS

Sample	Number of samples	Average weight (g)	Radioactivity			
			Beta		Gamma	
			(d/m/sample x 10^{-6})	(d/m/kg x 10^{-4})	(d/m/sample x 10^{-4})	(d/m/kg x 10^{-4})
ROOSTER	1	2250				
Skeleton		560	52	93	101	181
Muscle		1050	5.1	4.9	6.9	6.6
Gastrointestinal tract		185	.8	4.3	1.6	8.7
Liver		192	2.4	12.5	9.4	49.0
Respiratory tract		32	.2	8.7	.4	17.4
Total activity			60.5		119.3	
RATS	4	62.9				
Skeleton		4.1	.73	179	.15	35.5
Head		5.4	.15	36	.1	18
Muscle		39	.03	7.5	.04	10.2
Gastrointestinal tract		10	32	32.0	.27	27
Liver		3.6	.08	21.7	.06	15.6
Respiratory tract		.5	.03	62.0	.02	36.0
Total activity			1.34		.64	

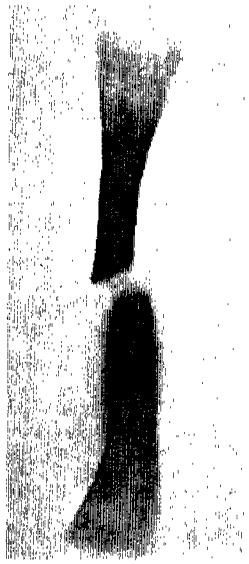

FIGURE 1.—*Autoradiograph of tibia of Rongelap rooster.*

0.095 μc/kg body weight which was approximately the level of activity in the rooster, 0.12 μc/kg body weight. The distribution of residual activity in the rat skeleton is illustrated in the autoradiograph of the femurs of the 4 rats, Figure 2. The activity is diffusely spread throughout the bone which suggests that these animals were born after the detonation. This diffuse activity represents the incorporation of low levels of activity over a long period of time. The one exception is rat No. 4, which shows a heavy line in the epiphyseal region suggesting that the animal was a young adult at the time of exposure.

As these rats lived for a period of years on Rongelap they serve as an indicator of the internal radiation hazard in human beings inhabiting this area. The Sr^{90}/Ca ratios for the tissue of these rats are presented in Table 5.

The carcass contained 470 to 545 sunshine units while 2 bones of the rooster analyzed contained 105 and 272 sunshine units.

For comparison, an autograph of a tibia of a kitten that was exposed to the initial fallout and collected 8 days after the detonation is shown in Figure 3. This animal died from natural causes at 1 year following exposure. The pattern of deposition of the fission products was similar to that observed in the rooster with dense concentration in the shaft of the bone. The light regions at the ends of the bone reflect the region of growth after the animal had been removed from the contaminated area. There was less translocation of deposited activity than seen in the rooster. Detectible amounts of activity, however, are seen in the ends of the bone.

In general, the internal radiation hazard from fallout depends on two parameters—availability of the various fission products and the biological effects produced when these fission products are deposited internally.

Evaluation of the biological effects produced by internally deposited fallout can be expressed in terms of limiting pathological processes.

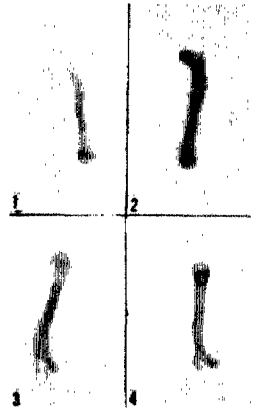

FIGURE 2.—*Autoradiograph of femurs of Rongelap rats.*

TABLE 5. STRONTIUM ^{90} LEVELS IN ANIMALS LIVING ON RONGELAP ISLAND

		Wet wt.	d/m Sr^{90}/sample	Ca/sample (grn)	S. U.[1]
Rats:					
1515	Carcass[2]	44.7	642 ± 23	0.533	545 ± 19
1516C	do	62.5	315 ± 62	.315	453 ± 90
1517C	do	32.3	367 ± 21	.353	470 ± 27
Rooster:					
1510	Femur	[3] 26.0	1210 ± 39	5.19	105 ± 3
1510	Tibia	41.0	5702 ± 119	9.50	272 ± 5

[1] S. U. = $\frac{2.2 \text{ d/m } Sr^{90}}{\text{gm Ca.}}$

[2] Does not include head, femurs, tibiae and viscera.

[3] Dry weight of 2 femur halves.

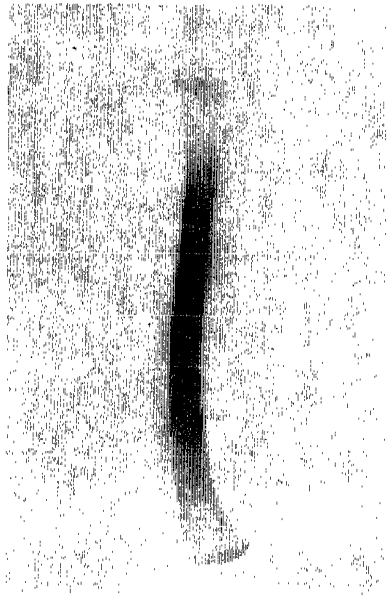

FIGURE 3.—*Autoradiograph of tibia of kitten collected on Rongelap, March 9, 1954. The kitten died of natural causes at 1 year post detonation.*

At relatively high dose levels these are damage to bone marrow, bone and to the G.I. tract. At very low levels as observed in this study, the limiting process is probably carcinogenic.

The long term effects can be described in terms of relatively few radionuclides. By the end of the 2nd year after detonation, the hazard may be characterized in terms of the levels of Sr^{90}. This is the critical element responsible for practically all the long term effects and in terms of which the habitability of a contaminated area may be assessed.

The relationship between environmental availability of the contaminant and the biological retention has not as yet been clearly delineated. Further laboratory and field studies are required to provide data on this relationship to allow for an estimation of internal radiation hazard to human beings from the physical availability data alone.

DISCUSSION

H. V. Weiss and S. H. Cohn

Dr. CHADWICK (PHS). I didn't happen to see on Dr. Weiss' figures anything about the breadfruit, and I was wondering, did you have any figures on breadfruit or not? I know in reading the calcium levels for breadfruit, it seemed that they ran higher than those for

cocoanuts. I was thinking that possibly strontium might follow that. Do you have figures?

Dr. Weiss. Just one sample of breadfruit was recovered, and it was not sufficiently active to warrant complete analysis.

Dr. Chadwick. I did sort of a limited dietary survey on some of the natives out there and found that breadfruit was one of the principal articles of their diet, and a very important one. To reassure Dr. Cohn, about the clams as nearly as I could understand it, the only seafood the natives ate to any extent at all was the fish. They didn't seem to eat the clams or crabs or langousta, the type of lobster they have out there.

Dr. Conard (BNL). It might be of interest that last May the one death that we had in the Rongelap people, a man 45 years old, who died of hypertensive heart disease, we obtained an autopsy and the bone specimens were examined at the New York Operations Office. The levels were very, very low in activity. There was a slight amount of strontium, at the same level we find in the autopsies on the American bones.

The urine of the Rongelap people at 2 years post exposure showed very low levels of activity. I think cerium, praseodymium about 6 disintegrations in 24 hours, and a slight amount of strontium 90; and a very small amount of cesium.

I would also like to add that these people during the first 2 days lived under extremely bad environmental contamination. I think it is of interest that after 2 years they have such low levels of body burden.

Dr. Lindberg (UCLA). As you gather, we are pretty well convinced that this distance factor is pretty real in regard to the distribution of the fission product. One is tempted to experiment with the fallout patterns with those in the Pacific as compared to those in the continental States. These are much more extensive. The figures I have seen seem to suggest that the islands that are being sampled in the Pacific would correspond to areas very close to ground zero on the continent.

Do you have any experiences in the Pacific that would let you comment whether the strontium or iodine samples might be much higher if you could sample effectively 1,000 or 500 miles out. I don't care to direct this question to anyone in particular. It is just pure speculation.

Dr. Cohn. No, we don't have any specific information on that. A number of islands were studied. They varied by distances of several hundred miles. Again the concentrations were roughly proportional to the distance and depend on the fallout pattern and so forth. Does this answer your question?

Dr. Lindberg. Yes.

PUBLIC HEALTH IMPLICATIONS OF SHORT TERM HAZARDS

By J. G. Terrill, Jr.

U. S. Public Health Service, Washington, D. C.

Dr. Terrill. Mr. Chairman and members of the symposium, it gives me a great deal of pleasure to be able to talk to you and to exchange views with you at a meeting of this type. It is a real opportunity for the Public Health Service and I think we should all thank you for inviting us to this meeting, and for giving us an opportunity to learn of your research and investigations in these various fields.

If we trace back in history a bit, we find that this pattern of cooperation between the Public Health Service and the military departments and the AEC has quite a historical background. Some very specific things were done during the Spanish American War, World War I, and World War II. The Public Health Service had a few officers assigned to the Manhattan Engineer District. The Department of Defense has helped in our training activities. Other more recent examples are our cooperative projects with the Atomic Energy Commission in Nevada and with Joint Task Force Seven in the Pacific.

In addition to these specifics, of course, there is a constant interchange of information through various scientific meetings.

From the public health viewpoint, one of the principles that we must bear in mind is the concept of total dose. From our standpoint it really matters little whether the population as a whole receives their limiting dose in a series of acute exposures or in very small amounts on a more continuous basis. At least that is what all of the authorities in this field generally seem to agree upon, even though they might not all agree on the specific limits.

In arriving at the standards that we talk about in technical meetings, and that are published in the newspapers, we feel that there is a great lack of human data, and that all of the standards leave much to be desired from the standpoint of explaining differences of opinion to the public in terms of human data rather than animal data, extrapolations and calculations. This is something that we all have to live with, but we have to recognize it as a real need. We hope that with the aid of such groups, as are represented here, and by other means, to obtain better information in this area.

Now, in terms of weapons tests, which are with us all the time, or more or less all the time, as contrasted with actual nuclear warfare, which we hope will never be with us, there are a series of public health phases that I would like to outline, and explain to you, with reference to the prevention of radiation exposure.

The first phases are actually in the hands of the AEC and the Department of Defense. This is clear to many of you, but all of you may not realize what an important public health job the planning groups in AEC and in the Department of Defense do in this regard.

One of the things they do is to select weapons or devices to minimize fallout. Others are the selection of the method of detonation, timing, place, and overall weather conditions in such a way that the total radiation load on the population is reduced. In these areas the first steps of preventive work rest with people who are represented at this meeting.

The next phase also is largely a matter for the test organizations to carry out. It is a matter of operational measurement. Scientifically these are based on research and special projects that you carry on at the test sites. However, they

are also related to clinical observation and evaluation. A consensus of this information and available instrumentation determines the operational techniques. Operational plans are then based upon techniques, objectives, and location. The summarized data are then available for emergency action, future detonation planning, and public health evaluation.

Another activity that test organizations provide for and which is very beneficial in reducing the total radiation load of the population is the matter of providing emergency measures in case the unexpected takes place. This has been done, I know, both in Nevada and in the Pacific, and I think it is an important public health service.

As we move from that point, we find that the responsibility and work load begins to become more diffuse. It is necessary to think about public relations and pseudo as well as real injuries to people. It is expected that most of the complaints and most of the reported injuries in the area around the test site in Nevada and in the Pacific will not be actually due to radiation. However, in this country those who can best help you explain what has actually occurred in a community or in individual cases are those agencies which we refer to as the local medical service agencies and the public health agencies. Thus the public health services enter the weapons test picture.

As a test period terminates, the radiation persists. Other factors affecting the test organizations and the detonations persist with the radiation. Some of these are manifestations of radioactivity that have both a public health and economic import.

Typical are questions related to milk supplies. People are concerned about the radioactivity in their milk. They are particularly concerned about the strontium in their milk. The photographic industry is concerned about particles on photographic paper. They are not so much concerned about total activity associated with particles.

Another affected group is the nuclear industry. Generally it must meet maximum permissible concentration standards for discharge of radioactivity into the environment. As the activity from our weapons tests and from foreign tests continues to increase, the radiological latitude which they have diminishes.

Another broad area of importance is the question of long term effects of radioactivity. Here we enter an area which is not clearly defined. We know qualitatively that radiation can increase the cancer rates under certain conditions. We know that it may cause genetic effects and we know it could change our aging pattern, and might change such things as thyroid function. However, as efforts are made to assess these quantitatively in population groups, normality must first be determined. This is difficult. Is the cancer incidence in any form actually increasing? If it is, what is the cause? Is radiation the cause? Is hypothyroidism increasing or isn't it, and how would we expect this to manifest itself in terms of population groups? If it does, what are the normal levels for these particular clinical manifestations that seem to affect our population?

If we have determined in a given situation that radiation has caused some increase in an observable way, then the sources become more important. However, their determination for a specific injury or group of injuries may be difficult. These are some of the things that make the problem of radiological public health particularly difficult. It is not as simple as making measurements and having data in a physical sense. It is a matter of being able to assess these data in terms of effects on people.

If a person is injured due to some radiation exposure complex involving the concept of total dose, who is actually responsible, and what systems are available for that person to seek help or to receive some financial reimbursement for his difficulties?

I will name some of these, and I think you will see that the problem is complex, and no one group or no one individual bears this total responsibility. In most cases, an injured person first looks to his own resources. He tries to determine whether this is a relatively small matter to be handled between himself and his physician, or whether it is beyond the scope of his personal resources. In a few instances where only one acute exposure has occurred and weapons tests are a factor, he might seek some type of compensation directly from the Atomic Energy Commission. If he is an officer or employee of the Department of Defense, he has available a relatively complete system of hospital and medical care services both before and after discharge. Survivors' benefits for active duty officer personnel are also available. However, the person not associated with the AEC or the military organizations is in a much weaker position to take care of himself in case of an unfortunate incident. He has his individual resources, and he has public hospitalization. He has such organizations as the Social Security Administration which provides benefits for him and his family in many instances. In other cases, the State compensation laws are applicable. But in the broad picture, the individual has no one place to go and no specific resource to fall back on. He falls back on the health and welfare resources of the communities as they exist today.

Thus there is a very broad area of potential responsibility in case these acute effects that you folks have discussed here today, create directly or synergistically public health effects of a measureable type.

Since my time is up, I should like to remind some of you, and tell others who are not familiar with the activities of the Public Health Service, that in addition to following through these administrative relationships which are very important to all of us, the Service does carry on a broad system of training, research, and support of public health organizations and medical care facilities which can help solve many public health problems which may be created either through military operations or through the increased exposure to radiation of an occupational or medical type in the future.

DISCUSSION

J. G. Terrill

Dr. Henshaw. Some of the health problems are international in character. Waste disposal into the sea is just one. I would like to ask whether any developments are under way for cooperation at the international level, say at the World Health Organization?

Dr. Terrill. Yes, there are developments under way within the Public Health Service and within the World Health Organization. I would have mentioned those except for the nature of this meeting. But briefly I will outline these for your information.

The Public Health Service is the WHO representative for the United States. About a year and a half ago after conference with Dr. Dunham of the AEC and Lauriston Taylor of the International Committee on Radiation Protection, it was decided to make every effort to integrate the international activities that Dr. Taylor had undertaken over the past years into the WHO organization. I should say organization system. This has been undertaken, and I understand it has been approved by both groups. This group in turn has set up a committee that has studied the matter of waste disposal among other things, and also another major concern has been the matter of training. Our Division of International Health in the Public Health Service is cooperating with both WHO and to some degree with the International Division of AEC and the Division of Biology and Medicine, in an effort to acquaint people throughout the world with our knowledge in waste disposal areas in particular, and in a broad training sense generally. Does that answer your question?

DISCUSSION ON TOPIC V

Internal Emitters

Col. TRUM. I have material similar to that presented by Dr. Lindberg. I would like to show a slide on which the results of an I-131 survey on cattle and humans are summarized.

The data contained in Figures 1 and 2 were taken from values of I-131 measured since 1954 to present. The survey began shortly after Van Middlesworth made his initial report. The cattle samples were collected by veterinarians of the Armed Forces throughout the world. They are averaged in presentation. This survey was done in conjunction with Comar's group at the Oak Ridge Institute of Nuclear Studies, where the survey of human thyroidal I-131 was made from samples submitted from various points in the United States.

In July 1955, a limited symposium on this subject was held at the Medical Division, Oak Ridge Institute of Nuclear Studies. It was pointed out at that time that there was a significant difference observed in the I-131 content of thyroids from pastured and stabled cattle. However, for the purpose of these data, only beef fed on the range or grown on the range and stabled for a short time furnished the samples.

Dots, which are indistinguishable to me from this distance, represent nuclear detonations. If we were able to make the distinction you would note that some are labeled Russian, English, and United States shots. Contrary to the British, who have told me they can see a USA flag in every radionuclide they find, we find little difference. There is a peaking following each test. There is a delay in peaking which we would not expect with a short-lived nuclide such as I-131.

I wish Dr. Comar were here to explain this more definitively. However, in my estimation, if radioiodine is to be critical in fallout, it will not be in this type of pickup but in a type which I had hoped would be discussed at this symposium, and that is the pickup of the shorter-lived isotopes. In my experience these may change the picture somewhat. I had hoped that there would be a program some place in which attention had been centered on these nuclides, where I think the relation between ingestion and inhalation or other factors may give us more variation.

I should like to point out, unless Col. Rust who is present would like to speak on this, that it does not take a lethal dose of irradiation to vary iodine pickup in the animal thyroid.

Col. RUST. You go ahead.

Col. TRUM. The first of the Col. Rust's slides is a micropathology section of the normal animal (Figure 3). Figure 4 is the thyroid of the acutely irradiated animal. Note the microfollicular changes that distinguish the normal from the irradiated thyroid. Figure 5 shows the results of iodine pickup in irradiated animals. The scatter of values reflects the physiological changes demonstrated in the histopathology of the previous figures. Although dose dependent, the variations are great. These variations are not due to techniques, but are reflections of the physiological condition of the animals. This phenomenon has been verified at the Radiobiology Division of the Army Medical Research Laboratory at Fort Knox. They have stated that under 900 r of whole body irradiation this phenomenon is a fairly good indication of dose.

I point out these things because we happen to have these data. If anybody has more of such material we would like to know of it. Thank you.

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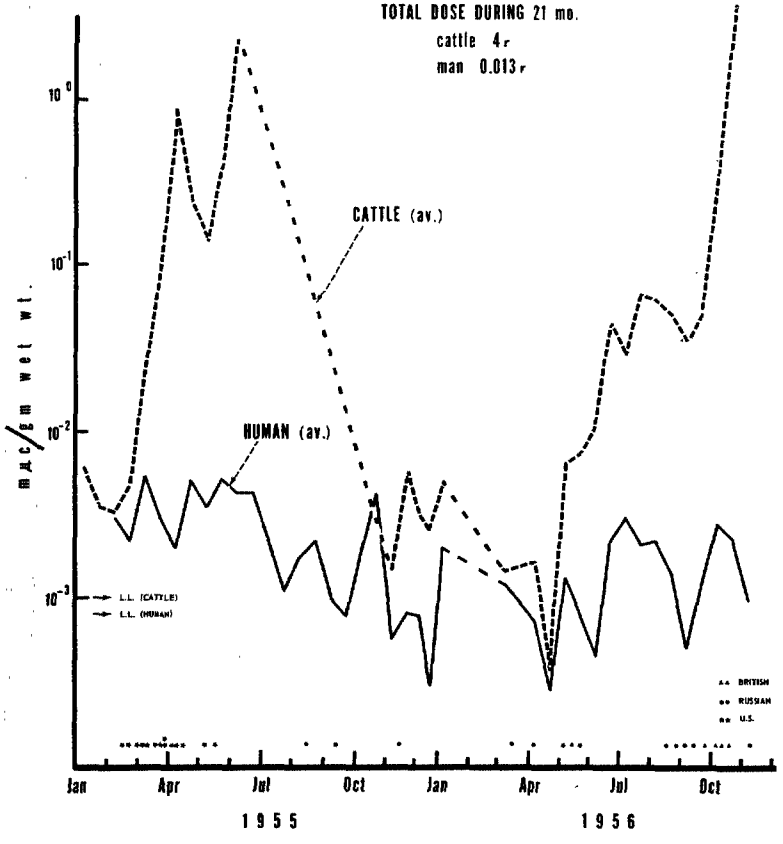

FIGURE 1.—*Total dose I-131 during 21 months. (1955–1956).*

Dr. LANGHAM. To summarize the status of the problem of internal emitters in one short sentence, it is quite a mess. The problem is, as always, lack of adequate data, and especially does this apply to human data.

I would like to more or less summarize the status of internal emitters by really pointing out where our lack of information might lie in hopes that it will stimulate the experimental radiobiologists, primarily, to increase their efforts, and perhaps especially their efforts to get more data on long-lived animals, primates, and especially man.

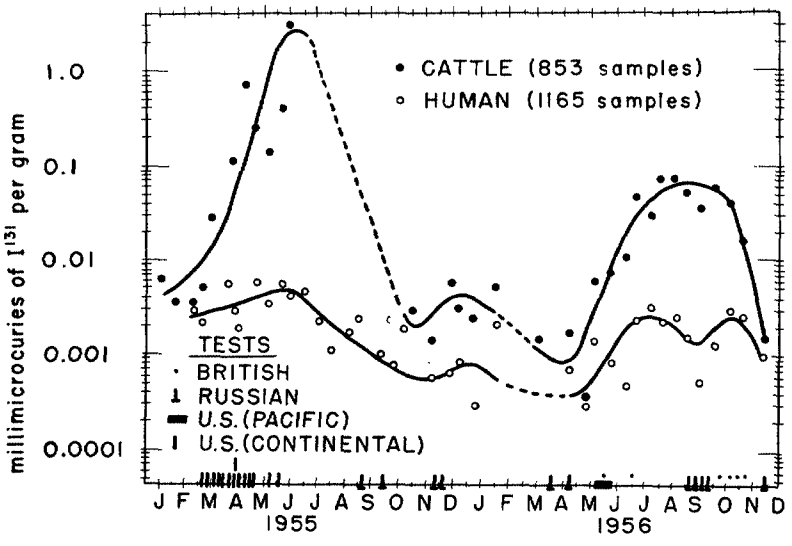

FIGURE 2.—*Cattle (853 samples), and human (1,165 samples) thyroid levels of I-131.*

I would like to put on the board the basic formulae from which Dr. Morgan has worked so diligently the last few years to get us tables and tables of numbers. From these formulae one can see the very inadequacies in our data which, if supplied, would allow us to put the whole subject of internal hazard on a somewhat more sound basis.

$$q=\frac{0.1(0.99)}{f_2}\times\frac{162}{\Sigma E(\overline{RBE})\overline{N}}$$

$$=\frac{16}{f_2\Sigma E(\overline{RBE})\overline{N}}$$

$$q=\frac{100mW}{\left[\begin{array}{l}3.7\times10^{4}\times1.6\times10^{-6}\times\\6.05\times10^{5}f_2\Sigma E(RBE)N\end{array}\right]}$$

$$q=\frac{2.8\times10^{-3}mW}{f_2\Sigma\overline{E}(\overline{RBE})\overline{N}}=\frac{8.4\times10^{-4}m}{f_2\Sigma E(\overline{RBE})\overline{N}}$$

$$(MCP)_a=\frac{3.5\times10^{-8}qf_2}{\tau f_a(1-e^{-0.693t/\tau})}$$

$$(MPC)_w=\frac{3.1\times10^{-4}qf_2}{\tau f_w(1-e^{-0.693t/\tau})}$$

q = maximum permissible body burden
W = 0.3 rem/week
m = mass of critical organ (g)
f_2 = fraction in critical organ of total in body
$\Sigma E(RBE)N$ = weighted energy absorption term given by equation C_5
$(MPC)_a$ = maximum permissible concentration in air (μc/c.c.)

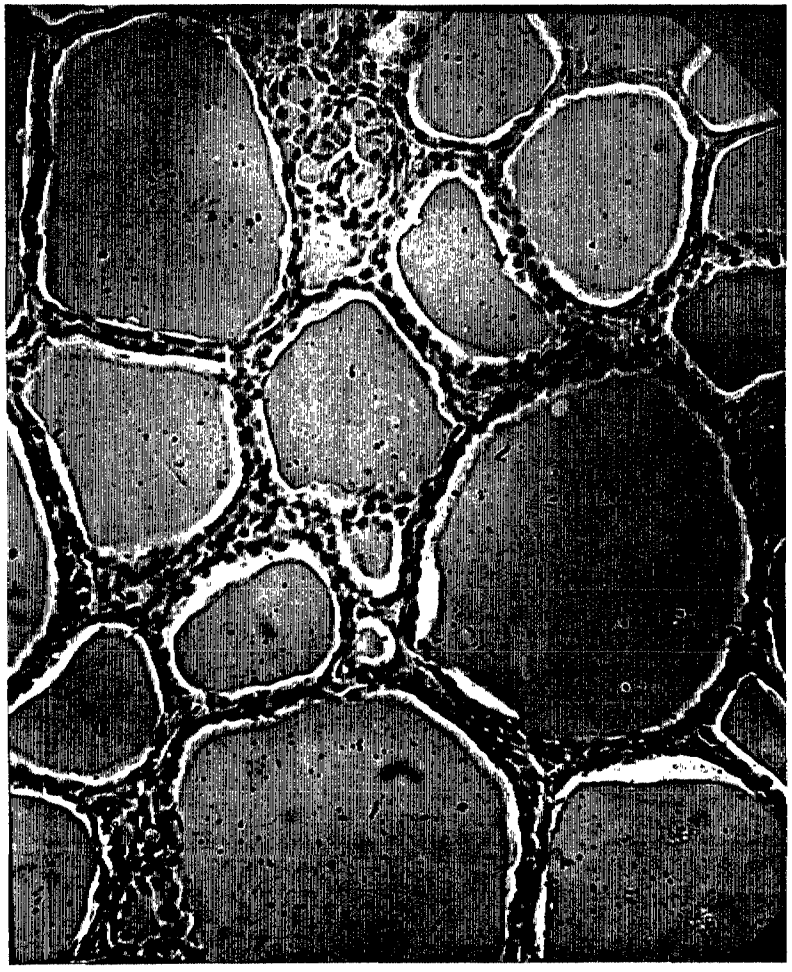

FIGURE 3.—*Normal thyroid burro.*

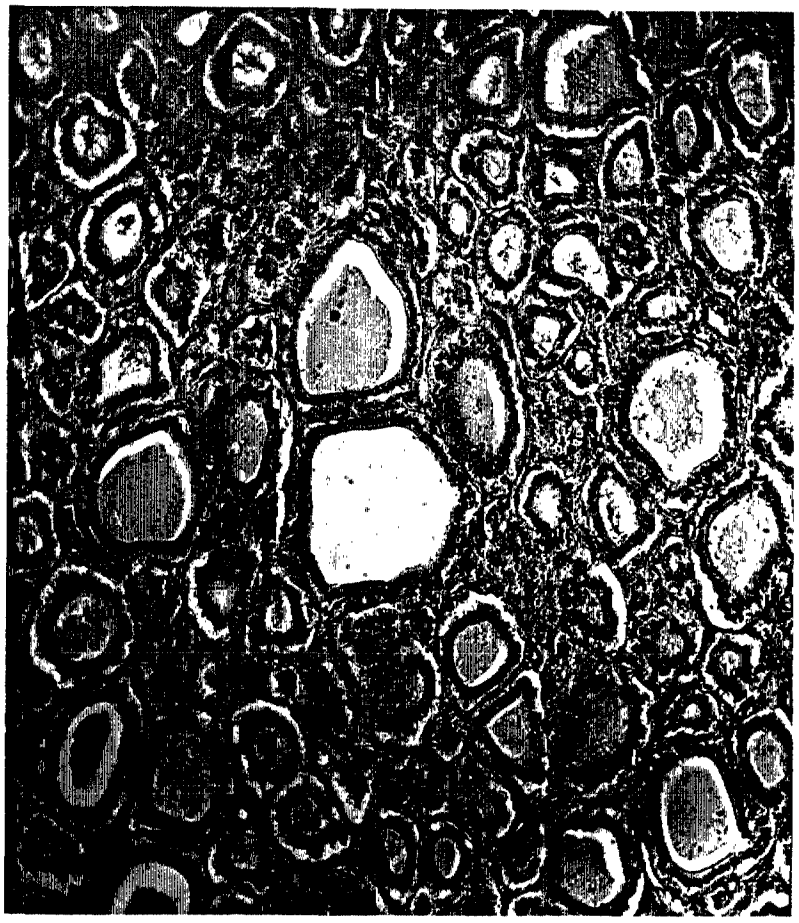

FIGURE 4.—*Thyroid of burro post irradiation.*

	Burro Number									
r	332	313	339	308	334	314	333	319	344	328
0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1 0	1.0	1.0
100		1.3		1.3		1.6		1.3		1 3
500	1.5		1.4		0.9		1.2		1.4	
1000		1.0		1.0		1.4		1.0		0.8
1500	7.3		10 0		5.7		8.5		9.1	
2000		0.7		0.0		0 5		0.3		0.2

FIGURE 5.—*Thyroid uptake of I-131 as influenced by irradiation.*

$(MPC)_w$ = maximum permissible concentration in water (μc/c.c.)
r = effective half-life (days)
f_a = fraction inhaled that arrives in critical body organ
f_w = fraction ingested that arrives in critical body organ
t = period of exposure (days)

The basis of our present calculations for the maximum permissible burden of internal emitters goes back to either of two concepts that are themselves based on human experience. We have had enough experience with X-rays and gamma rays to feel that 0.3 roentgen per week will not do appreciable damage to a person if taken throughout a working lifetime.

On the basis of 0.3 roentgen per week, then, the Subcommittees on Internal Tolerance or Internal Maximum Permissible Levels have chosen to relate the dose of the internal emitter to that amount of the internal emitter which will deliver the equivalent of 0.3 of a roentgen per week to a critical organ, usually the organ which shows the highest concentration of the material. We find that q, the maximum permissible amount in microcuries, is equal to a constant times the mass of the critical organ times the 0.3 rem per week. The biologist cannot even tell Dr. Morgan with certainty what the mass of the critical organ is. Obviously this is the fault of biological variability and not the biologists. This factor is divided by the fraction of the material in the total body that concentrates in the critical organ, that is, f_2, Dr. Morgan gets these f_2 values from any animal experimentation that he can or anywhere he can find them.

Many of us do only service to him by calling an occasional number to his attention. But for that matter, no one is really certain in every case of every isotope what the fraction of that in the total body is concentrated in the critical organ. Especially is this true of humans. This then is multiplied by the sum of all the energies - this of course can be gotten from physical data—weighted for some or all the energies for each disintegration, weighted for the relative biological effectiveness of each.

Now we really have him in the land of uncertainty. RBE is supposedly that effect of the radiation when compared to a similar effect of X-ray on an energy to energy basis. In other words, it is surprising to find that we do not agree to this day whether or not 100 ergs of energy delivered from an alpha particle is 1 or 20 times as effective as 100 ergs of energy delivered from X- or gamma ray. So obviously, RBE is an area of uncertainty, and one which will probably remain uncertain for a great length of time, because it seems now that RBE may be specific or may be different for every biological effect and every biological system that one wishes to test.

Then the factor N, which is the distribution factor, and in some cases is called the ignorance factor. It is into N that we can lump all of those uncertainties, including the uncertainty as regards the homogeneous distribution of the material in the critical organ. There, then, we can see that there is plenty of room for improvement in the various numbers that go into the basic formula of calculating maximum permissible levels.

Taking advantage of another human experience, it is customary to relate the maximum permissible level of internal emitters of bone seekers to 0.1 microcurie of radium, and the first formula I gave previously expresses this relation. In other words, q now the microcuries of the unknown substance which is the bone seeker

is equal to 16 divided again by the f_2, the fraction of that in the total body which is concentrated in the critical organ, times the summation of the energies of all the disintegrations, each one weighted for its RBE and for its distribution and energy deposition in the critical organ. We can see for that matter that there is absolutely an area of uncertainty in whether or not a tenth of a microgram of radium is a true base line in humans on which to base these data. But until better data are available, this then is the best we can do. When we calculate maximum permissible concentrations of air and water we find that the MPC equal to a constant times q, the q which was derived in some manner as specified earlier, times the fraction of the material in the total body that is in the critical organ, divided by another biological uncertainty, and that is the fraction of that which is inhaled which ends up in the critical organ, times the effective half time, and the effective half time in this case is the radiological half time times the biological half time divided by the sum of the two, all of that times one minus e raised to the 0.693 power times t, the time of exposure that we intend to let the individual receive in order to come to equilibrium, divided again by the effective half time.

We see here, then, many places where the data could be improved by experimental data on humans or primates or for that matter even better animal data. For example, the biological half time of many of these substances has never been determined in animals. The fraction of that which is inhaled, which goes to the critical tissue, is very closely tied in with the problem Dr. Stannard was discussing this morning. Obviously this depends on particle size. Particle size is only 1 of 17 different factors reported in the literature, which are supposed to affect lung retention.

After we get the particle problem taken care of we still have 16 others to go. We can then see a great degree of uncertainty that may exist where more information or more nuclides in humans especially, and primates, are sorely needed, even such a simple thing as a biological turnover time. In regard to that particular factor, I would like to mention our own work which involves whole body human counting in which by means of the whole human body techniques (the liquid scintillator at our place, and the crystal spectrometer at the Argonne), we find it is possible to give as little as 1/100th of the rem maximum permissible burden to a human and get biological turnover times by merely counting the individual at intervals. We have started through the periodic table using every good gamma emitter to try to correlate biological turnover time in mice, rats, dogs, monkeys and man. Here we inject the material into the animal and merely count him in the whole body counter periodically to get the retention curves.

Going through the periodic table is a slow process. In fact, as I said at the Health Physics Society meeting, if we report on one sub-family each year, then we are assured of getting to attend the meeting for the next 18 years. We find that we are not quite keeping up with the schedule. You cannot get through one subfamily of the periodic table in that length of time.

Such interesting relationships are already starting to come out as trying to correlate the weight of the animal with the biological turnover time. We find that for sodium and potassium, if one plots the log of the weight of the animal species versus the biological half time one gets a nice straight line. When one gets to rubidium and cesium, up through the dog and monkey, one seems to get a straight line. When one gets to man, man no longer fits on the curve. The biological half time determined by whole body counting of cesium is of the order of 110 days in man; for rubidium it is of the order of 85 days. We hope by going through the periodic table and picking gamma emitters and giving them to human volunteers we can eventually get more data on biological retention times.

Another point I would like to mention, of course, is this very idea of retention of particulate matter in the lung. If one looks in the International Commission handbook, and I am sure in the new version of the National hand-

book, one will find a very arbitrary decision made on the disposition of radioactive particles in the lung, made primarily to enable us to go ahead and calculate the maximum permissible air concentrations based on lung retention. One will find that we arbitrarily say that 25 percent of that which is inhaled or of those particles which are inhaled come right back out without setting down on the mucous linings of the respiratory tract. We say 50 percent deposit in the upper bronchial tree. Twenty-five percent of these particles get on down into the alveolar sacs. This might be true for a specific particle size distribution. To think it would be true for all 17 of these conditions that I mentioned on which lung retention is dependent is asking for quite a bit. But anyway, in order to have a basis for calculation, we must assume this 25 percent turns around and comes right back out, 50 percent deposits in the upper bronchial tree, 25 percent gets in the alveolar sacs. Of that which deposits in the upper bronchial tree we say it is all essentially removed with a biological half time that is strictly a guess. We say of that which goes into the alveolar sacs, if the particle is insoluble, twelve and one-half percent of it turns around and comes up in the bronchial tree, and eventually ends up in the gut.

I think Dr. Morgan said this morning that 61 percent ends up in the gut. I hope he meant 62.5 percent, because his pencil can pick up those differences. The biologists cannot tell the difference plus or minus a factor of 10.

Of that 12½ percent which remains in the alveolar spaces, all is assumed to be eventually absorbed and contributed to the body burden. That is a model on which our present concepts are based. To test this model in animals of various types and especially in the human is certainly one of the great needs and I think one of those things for which the biologists will eventually collect the data if they live long enough, and receive enough support from the various agencies.

Lastly, something that has dominated this meeting entirely is this concern for the fallout problem. Even though this meeting was supposed to be on immediate fallout, it was obvious the thinking was on long-term chronic fallout, such as may be involved in worldwide contamination. Here our ignorance becomes even greater, though reading as much of the information from Operation Sunshine that one can, I can't feel that we are in any serious trouble. I think it is true that we in all probability may have the strontium content in children by 1970 up to maybe 1/100th or maybe 2/100ths of the maximum permissible body burden for large populations, that being set at 0.1 microcurie for strontium. As far as I can see all of the excitement that we have just had over this problem, is hardly justified. There is hardly any doubt that we are dropping radioactivity on people, and we have, in keeping with the urgency of the Public Health Service, been pursuing this as a problem in order that we will know what the status of it is, and what to do with it before it ever becomes a problem, we hope. Let us merely question this 0.1 microcurie for worldwide populations.

Long term chronic studies are needed to really determine whether 0.1 microcurie of strontium is a maximum permissible level in the human subject, one that we can live with and feel confident of. I would say that it is probably a conservative one. If one calculates the radiation delivered to the bone from natural sources over a 70 year period under normal radium content soils and building materials, he comes up with the idea that the bone may receive about 8.5 rem per 70-year lifetime. In high radium areas, it may be as much as 3 times that, or 4 times, which would be up to around 30 or 36 rem per 70-year lifetime. If one takes one-tenth microcurie of strontium and assumes that this remains in the bone throughout a 70-year lifetime one comes up with about 18.5 rem per 70-year lifetime.

This is taking the pessimistic view, because we know that a major part of this strontium is laid down by age 20, and that in all probability maybe equilibrium will be maintained by exchange. Maybe it won't. If one considers a factor of decay from age 20 on, then one would say that a tenth of a microgram of strontium deposited up to age 20 and maintained at the normal radioactive decay rate to age 70 would deliver a dose to the bone that is about equal to normal background from cosmic rays and radium.

This is cutting the number pretty fine when we are considering that for somatic changes; since strontium does not concentrate in the gonads, we are considering that we can only double the natural background. But until we do know where we are going, it is obviously wise to walk with caution.

I might say still more fundamental and troublesome in this whole problem of internal emitters is the formula that we gave earlier and especially the second one. This formula is based on the concept that it is equally bad to give to an organ 0.3 of a rem per week as it is to give 0.3 of a rem to the whole body.

Is the radiation partially delivered to an organ worse than to the whole body? I think the importance of this can be seen when you see Dr. Morgan now calculating maximum permissible levels on the basis of the radiation of a small amount of the large bowel. Is to irradiate the large bowel with 0.3 rem a week as bad as radiating the whole body? It may be that irradiating the large bowel is just as important because the large bowel may be a very sensitive organ.

This I have said in summary of the symposium here because it is my understanding that the Chairman has the privilege of getting up and making broad sweeping statements and closing the meeting, which does not allow his sweeping statements to be a subject of discussion.

CLOSING REMARKS

EUGENE P. CRONKITE

Dr. CRONKITE. Dr. Dunning, Col. Maxwell, I certainly must admit that I accepted this rather reluctantly, not being by training and experience really qualified to interpret all of the different diverse disciplines and talents that have been discussed here. However, I did accept it, and if you will bear with me for a few moments, I will go over some of the things which I primarily looked at perhaps as a physician.

The objectives were to try to get some idea of what should be done, some idea of what is actually well known, and where to go from here. I will try to take each section rather briefly.

The first topic on decay constants, weathering and shielding, produced some rather interesting facts that I had personally not appreciated. Dr. Nagler of the Weather Bureau outlined the input data for their model for the prediction of fallout which embraces the necessary physical parameters that must be put into Stokes law. However, I detected a rather simple statement that he made as he went over this, that in reality they took past experience and fed past experience into their machines, and then predicted the fallout, rather than used the actual mathematical model. This seems to indicate that in this area, not only for the rather diffuse planar distribution of fallout material, but particularly to get practical information on drift, turbulence, piling up and inhomogeneities that must certainly exist in areas, particularly in urban areas, if fallout should occur, is really an urgent field for further mathematical and practical study.

Dr. Graveson presented encouraging data on the effectiveness of shielding by a building that is comparable in its dimensions to the average American home. It appeared that these measurements gave very significant protection. However, the diminution in the intensities actually measured inside this aluminum building seemed to be somewhat in conflict with the concepts that were later presented by Dr. Borg and Dr. Bond.

It appears that many more empiric studies of this sort are indicated to try to bring together experiment and theory. Dr. Breslin pointed out the great effectiveness of simple types of washdown provided the conditions of wetting and adequate volume flow are maintained.

The data presented by Dr. Zobel on the emission of fission products very early after fission confirms the calculations of Borg and gives much further useful information that can be fed into the experimental models.

Dr. Mather's contribution was a most practical point. The spectrometry readings varied considerably with angle from the surface of the ground, and pointed out the practical problems of shielding, and that shielding is most effective against the horizon.

Dr. Borg pointed out that the Spencer-Fano equations for gamma radiation can be used most effectively to define the spectrum at any point in space from a monoenergetic or polyenergetic source when the necessary factors are fed into the model. The mean or effective energy of a polyenergetic source is useless. The source must be treated as separate, discrete fragments, to study the behavior of each with distance using the appropriate buildup factors to describe the condition in space in which one is biologically interested.

He pointed out that the actual measurements in the field were initial radiation, and those predicted by theory are very close, indeed.

Dr. Borg further pointed out that a similar method could be well applied to the analysis of the spectrum from a fallout field, and in fact, preliminary calculations have indicated its

feasibility. It appears that this is a very important area in which further analyses are necessary.

Dr. Bond exhaustively demonstrated the facets that determined depth-dose patterns in phantoms. From the data of Chamber, Imirie and Sharp, he conclusively demonstrated that the patterns observed in the fallout field and with initial radiation are approximately what theory indicates they should be when the source is treated as multiple separate discrete sources, and using the appropriate buildup versus distance considerations. The inadequacies of air dose to express biological effect was proved, and the dependence of the biological effect on depth-dose pattern was evident. It can only be hoped that the approaches and conclusions of Drs. Borg and Bond will be used by the hazard-evaluation people, and by those people performing further empirical field studies.

In addition the apparent acute hazard of neutrons to man was dealt a rather severe blow when depth-dose considerations were dropped by use of what one might call the engineering RBE from a maximum of two to a mouse to less than 0.1 for a large animal, such as man. This is in respect to the acute effect.*

In the section on biological repair, Dr. Henshaw has courageously proposed a work capacity versus dose and time graph. This will be accepted gratefully by those who have to estimate hazards. However, it can only be hoped that they will use it in the manner that was proposed, and with all the reservations that Dr. Henshaw presented. I can't help but feel that the rather flat depth-dose effect response that Dr. Henshaw presented for man might be much more steep if all of the air doses that went into it were appropriately converted to tissue dose.

Dr. Storer, Sacher, Blair and Jones were fortunately assembled all in the same room at the same time. The result was certainly from my standpoint most educational and interesting. As a basis for all approaches are some very strong assumptions that injury processes are linear. If these basic assumptions are proved wrong, it is quite evident their theories will predict inaccurately. What appears more important is that as further analyses are made, one realizes the death function both acutely and chronically is exceedingly complex. Repair processes proceed at different rates in different tissues. Death can be reached by a multiplicity of mechanism and causes, and it appears that much more experimentation with all the permutations and combinations of radiation techniques, of varying dose rate, area of body irradiated, fractionation, etc., will be necessary to finally resolve the relationship between total dose, dose rate, fractionation and life shortening.

The areas of agreement seem to have broadened considerably. Although Dr. Blair doubts half times for the recovery of injury processes can be correlated with any measureable physiologic parameter, it appears that this would be a desirable area to investigate.

From the practical standpoint, a correlation of recovery in peripheral blood with half time seems desirable for here is a point to use in extrapolation to man, since long term hematologic data is becoming available in the Marshallese. It is quite evident that this is one area in which the direct clinical research is not acceptable.

It was of interest that Dr. Trum's data on the hematologic recovery in burros looked very much like the Marshallese data to date.

In the section on beta burns, Col. Brennan, making certain assumptions on energy and uniform distribution of fission products, calculated the contributions of dose at a point in a planar field as a function of radius and height above surface. This approach coupled with the Spencer-Fano equation could describe the dose at this point from polyenergetic fission field more adequately. This dose should represent the maximum hazard since drift, directionality and shielding would all effectively diminish the effect as previously considered by Drs. Bond and Borg. I personally do not share the feeling that beta bath is a real hazard as implied by Col. Brennan. The contact beta burn is a reality. The beta bath effect is diminished by movement, by clothing, by a foxhole. If one were prone or supine, immobile and nude, suspended 5 centimeters from the ground, the effect would be great indeed at a beta-gamma ratio of 50 to 1.

For situations that I can see with dose rates that are probable, one would have to be precisely prone, immobile, and nude and probably dead. Not meaning to introduce levity, but this is a difference of opinion, and certainly more study is needed to resolve these differences.

In respect to the biological effect of beta irradiation, the obvious question is, do animal studies apply to man? In part, I think the answer is yes. However, I have been assured by many veterinarians that the skin of cattle and of swine is particularly more reactive and prone to produce hyperkeratosis and acanthotic lesions as observed. There can be no question about a qualitative similarity, but I somewhat question whether one can say there will be quantitative similarity between the animal studies and man. I personally think that the cosmetic future of the Marshallese is rather good. Certainly Dr. Conard in his continuing studies of the Marshallese throughout their lifetime or his will find out the answers.

Particular importance in assessing the beta hazard, I believe, are the attentuation curves that Dr. Conard presented.

In Dr. Morgan's presentation it was certainly welcome news to know that the National Bureau of Standards Handbook 52 will be revised and have a broader base and include new nuclides and both single and chronic hazard estimates. I do not see how he and his group can possibly do all this work that is involved in these revisions and we certainly owe them all a debt of gratitude.

Dr. Jones' studies on iodine-131 uptake in the thyroids of cattle and of man certainly were most encouraging and show quite conclusively that the dose is small. The observations of Trum concurred with this indeed.

I was most interested in the studies of Dr. Durbin on the kinetics of strontium-90 uptake, retention and excretion. It goes without saying that much more studies of this type, as has been so ably demonstrated by Dr. Langham a few moments ago, are urgently needed in a wide spectrum of animals and over the wide entire spectrum of the radio nuclides.

Dr. Placak's observation on plutonium-239 and its distribution in the Nevada test site and areas remote from there quite conclusively demonstrated that though there is apparently no hazard here again is another subject that must be closely watched and a continuing study is essential.

Dr. Stannard listed the physical and physiological parameters necessary to evaluate the pulmonary hazard from particle inhalation. However, the problem was not put to rest. It looks as though a start has only been made, and a tremendous amount of work yet is to be done to try to evaluate a single nuclide, let alone the sphere of size and substances from fission products.

In Major Woodward's absence, Dr. Schrodt presented the problems that were closely allied to the previous observations of Dr. Jones and Col. Trum on urinary excretion of iodine-131. It seems that there is one minor or possibly important difference here. It seems inconceivable that man could be taking the iodine in other than by inhalation. The cattle intake was from feed, predominantly grass.

The studies of Dr. Lindberg and Dr. Larson brought out what struck me as two rather important considerations. First, the fractionation of fission products by the size of the particles between plants on which animals graze, and the underlying ground, and the fractionation of iodine-131 and strontium-90 with distance from the site of detonation. It appears that all of these factors must have to be fed into the ultimate models for assessing both acute and long term fallout hazards.

I was quite impressed with the mass of data that Drs. Weiss and Cohn presented. However, as a physician, I find myself completely unable to interpret the importance. It appears

*Subsequent work has shown that the relative effectiveness factor may lie in the vicinity of 0.5.

that a tremendous amount of kinetic data on the relationship of not only strontium-90, but all of the substances that are in fallout in respect to the availability, uptake, retention and circulation in all of the biological cycles that eventually lead into the food chain are essential before one can have an adequate model to evaluate hazards.

I need only comment on Dr. Terrill's talk that the gist of his statement is the sort of thing that I personally feel should be disseminated widely in the appropriate form to the public.

In concluding the summary, and although instrumentation was not a part of this symposium—it was deliberately not a part of this symposium—I can't but have the feeling that instrumentation development, manufacture and use is going ahead without, at this time, sufficient delineation of the real biomedical problems that need to be known. Perhaps further study of the instrument side should be gone into and further evaluation of what does one really need to know from an instrument before another instrumentation development program with its tremendous expense of time and money is entered into.

In concluding, I would like to say that this symposium has been most valuable and educational to me, and on behalf of all of you, I would like to thank Dr. Dunning and Col. Maxwell for organizing it. [Applause.]

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