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Report to the Scientific Director

OPERATION CASTLE - FINAL REPORT PROJECT 41

**Study of Response of Human Beings
Accidentally Exposed to
Significant Fallout Radiation**

41402

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October 1954

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and

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ABSTRACT

Following the detonation of Shot 1 on Bikini Atoll on 1 March 1954, 28 Americans and 239 Marshallese were exposed to fallout. One hundred fifty-seven of the Marshallese were on Utrik Atoll, 64 were on Rongelap Atoll, and 18 were on the neighboring atoll of Ailinginae. The 28 Americans were on Rongerik Atoll. The presence of significant fallout on these atolls was first determined by a recording dosimeter, located on Rongerik, when this device went off scale at 100 mr/hr shortly after the detonation. Emergency surveys detected radiation on the inhabited atolls, and evacuation of inhabitants to the Naval Station at Kwajalein was promptly carried out. The dose of radiation to which the individuals were exposed was calculated from the intensities found on the islands and the decay exponent of the fallout material. The individuals on Rongelap received approximately 175 r, those on Ailinginae received approximately 69 r, and the Americans on Rongerik received an average calculated dose of 78 r. The Marshallese on Utrik received approximately 14 r. The fallout on Rongelap, Ailinginae, and to a lesser extent on Rongerik was distinctly visible. No fallout was observed on Utrik. A significant number of individuals on Rongelap suffered from mild nausea and one or two individuals vomited on the day of the exposure. With the exception of nausea in one Ailinginae individual, there were no other definite gastrointestinal symptoms in the other Marshallese or the Americans. The Marshallese on Rongelap and Ailinginae, and the Americans experienced to a varying degree burning of the eyes and itching of the skin from 1 to 3 days. Later, signs of radiation injury included definite epilation in the Rongelap and Ailinginae groups, and the development of spotty, superficial, hyperpigmented skin lesions that desquamated from the center of the lesions outwards. In some cases the skin damage was sufficient to result in raw, weeping lesions. There was no full thickness necrosis of the skin. The Americans developed only minor skin lesions without ulceration. There were no skin lesions in the Utrik natives. All lesions healed rapidly with no further breakdown of the skin noted during the period of observation. Microscopic examination of biopsies of the lesions showed changes usually associated with radiation injury. Fully clothed individuals and those remaining inside of buildings or huts were protected to varying degrees from development of lesions. Hematologic changes were definite in the Rongelap, Ailinginae, and the American groups. Lymphopenia appeared promptly and was persistent for a prolonged period of time. Neutropenia occurred in all of the individuals with initial minimum values occurring around the 11th day followed by an increase in the counts and a secondary minimum around the 40th to 45th day. The most consistent hematologic change was the depression in the platelet counts. Platelets were below normal when first counted on the 10th day of post-exposure and progressively decreased attaining a minimum between the 25th and 30th day. Although recovery commenced following this minimum, the platelet counts had not returned to normal by the completion of the initial study on the 76th post-exposure day. The incidence of various respiratory and cutaneous infections was identical in all exposure groups and bore no relationship to the hematologic changes.

Urinary excretion of radioisotopes was studied. Small amounts of radioactive material were found. Estimates of total body burden indicate that there is no long term hazard and that ingestion and inhalation of isotopes did not contribute significantly to the initial radiation exposure.

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FOREWORD

This report is one of the reports presenting the results of the 34 projects participating in the Military Effects Program of Operation CASTLE. For readers interested in other pertinent test information, reference is made to WT-934, Report of the Commander, Task Unit 13, Military Effects Program. This summary report includes the following information of possible general interest.

(a) An over-all description of each detonation, including yield, height of burst, ground zero location, time of detonation, ambient atmospheric conditions at detonation time, etc., for the operation.

(b) Discussion of all project results.

(c) A summary of each project, including objectives and results.

(d) A complete listing of all reports covering the Military Effects Test Program.

Detailed reports on dosimetry and internal radioactive contamination will be published as supplements to this report.

Much of the medical terminology in this report is explained in parentheses for the benefit of the layman.

PREFACE

Operation CASTLE did not include a biomedical program. The participants in Project 4.1 were drawn from various laboratories and were totally unprepared for a field program when the joint DOD AEC Medical Research Team was organized after the operation had begun.

Dr. John C. Bugher, Director, Division of Biology and Medicine, AEC, and Lt Col L. E. Browning, MC, USA, Surgeon of the Armed Forces Special Weapons Project, selected the project officer and requested that the selection of technical and professional personnel be commenced immediately. Rear Admiral Clarence Brown, Deputy Surgeon General, Medical Department, USN, gave immediate and complete support of all naval medical research activities and assigned responsibility to CAPT Van Tipton, MC, USN, and CDR Harry Etter, MC, USN, of the Atomic Defense Division, Bureau of Medicine and Surgery, USN, for the implementation of the project.

Since little detailed information was available about dose and initial symptomatology, the research team was organized to include the following talents for the constitution of the emergency medical team: internal medicine, hematology, radiation technology and radiobiology. In the selection of personnel the emphasis was placed on past experience in biomedical research in the field with atomic weapons. In addition, provisions were made for a second echelon of specialized personnel in case they were needed. Accordingly, a preventive medicine unit of the Commander in-Chief, Pacific Fleet, was alerted for possible bacteriologic studies; blood bank personnel, and additional clinicians and nurses were notified in case conditions justified their services in the Kwajalein area. Rear Admiral Bartholomew Hogan, MC, USN, Pacific Fleet Medical Officer, immediately made any needed medical facilities of the Pacific Fleet available.

Personnel were obtained within the Continental limits of the U. S. for the research team as follows:

Naval Medical Research Institute (NMRI)

4 medical officers (E. P. Cronkite, project officer, R. A. Conard, N. R. Shulman, and R. S. Farr)

2 medical service corps officers (W. H. Chapman and R. Sharp)

6 enlisted men (C. R. Sipe, P. K. Schork, C. P. A. Strome, W. C. Clutter, R. E. Hansell, J. S. Hamby)

U. S. Naval Radiological Defense Laboratory (NRDL)

1 civilian M.D. (V. P. Bond)

1 medical service corps officer (L. J. Smith)

4 enlisted men (W. H. Gibbs, J. C. Hendrie, W. S. Argonza, J. Flanagan)

Division of Biology and Medicine, AEC

2 civilian M.D.'s (C. L. Dunham and G. V. LeRoy)

Armed Forces Special Weapons Project (AFSWP)

1 Army Medical Officer (L. E. Browning)

Preliminary studies had been made by CDR W. S. Hall, MC, USN, Station Medical Officer and his staff, and decontamination of the individuals was well underway when Project 4.1 person-

nel arrived in the field. Preliminary hematologic studies indicated that the individuals probably had not received acutely fatal doses of radiation. The primary responsibilities within the project group were delegated as follows:

- Clinical observations and care LT N. R. Shulman, MC, USN
- Organization and operation of laboratory LT R. S. Farr, MC, USN
- Compilation and daily analysis of all data Dr. V. P. Bond
- Decontamination and radiation measurement LT (jg) R. Sharp, MSC, USN
- Senior Petty Officer in charge of laboratory P. K. Schork, HMC, USN

As the clinical picture developed, a further breakdown in responsibility was necessary. Commander R. A. Conard, MC, USN, and Lt Col L. E. Browning, MC, USA, were made responsible for a daily survey of skin lesions, and Dr. S. H. Cohn was made responsible for studying the problems concerned with the excretion of radioisotopes and the estimates of body burdens in the exposed individuals.

The project officer commends all of the professional and technical members of the group for their excellent motivation, initiative, and voluntary long hours of extra work that were essential for the accomplishment of the clinical and research objectives, and for the rapid collection of the preliminary data in the field. It is emphasized that the work was a cooperative endeavor in which all were mutually dependent upon each other. The willing efforts of all concerned constituted a remarkable example of team-work and sacrifice of personal ambitions and desires for the good of the project at large.

The authors wish to express their gratitude and indebtedness to Dr. John C. Bugher, CAPT Van Tipton, and CDR Harry Etter; CAPT W. E. Kellum, MC, USN, and CAPT T. L. Willmon, Commanding and Executive Officers, respectively, NMRI; CAPT R. A. Hanners, USN, Director, NRDL, and CAPT A. R. Behnke, MC, USN, Associate Director, NRDL; all of whom gave unlimited support and reduced administrative procedures to a bare minimum, thus making it possible for the unit to be assembled and underway in a matter of hours.

On arrival at Kwajalein, RADM R. S. Clarke, USN, Commanding Officer, U. S. Naval Station, Kwajalein, supported Project 4.1 with all the facilities at his disposal. As a result, a laboratory and clinic was established and operating within 24 hours after arrival of the project personnel.

Project personnel also wish to acknowledge the outstanding contributions of Col C. S. Maupin, MC, USA, Field Command, AFSWP, CAPT H. H. Haight, MC, USN, Division of Military Application, AEC; Col K. Houghton, MC, USAF, Special Weapons Center; CAPT Donald Dement, MC, USN, CINCPAC Fleet; Drs. T. L. Shipman, T. White, and P. Harris of Los Alamos Scientific Laboratory; Dr. Gordon Dunning, Division of Biology and Medicine, AEC, and Dr. G. V. LeRoy, University of Chicago. During all phases of the early care of the exposed individuals, the foregoing participated as much as their other primary duties would permit. In addition the authors wish to thank them for the extensive and complete data which they collected in the atolls or their home laboratories and kindly furnished to the project personnel.

The continuous help and cooperation of Trust Territory representatives and their aid in obtaining necessary control data on native Marshallese at Majuro is hereby acknowledged. The authors are particularly indebted to Mr. John Tobin. His help as an interpreter and his extensive knowledge of the Marshallese language and habits were invaluable. Lieutenant J. S. Thompson, MC, USN, furnished his records on the exposed individuals decontaminated by the radiation group of the VP-29 squadron stationed at Kwajalein.

The authors wish especially to express their admiration for the excellent job done by the medical personnel of the U. S. Naval Dispensary, Kwajalein, in completing the extensive laboratory examinations that were required to obtain a prompt initial evaluation of the severity of the radiation injury.

The authors are deeply grateful to Dr. David A. Wood of the University of California Hospital, San Francisco, and Dr. Edward L. Alpen and Miss Pat Roan of NRDL, for invaluable aid in carrying out the histopathological evaluation of skin lesions. The extensive contributions of Mr. H. H. Hechter in the statistical analyses of the data, and of Mr. C. A. Sondhaus of the NRDL in dosage calculations are gratefully acknowledged.

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CHAPTER I

INTRODUCTION

1.1 OBJECTIVES

Project 4.1 was organized with the following specific objectives:

- (1) To evaluate the severity of the radiation injury in the human beings exposed to the fall-out radiation.
- (2) To provide for all necessary medical care for these individuals.
- (3) To conduct a scientific study of radiation injury in human beings.

1.2 GENERAL DESCRIPTION OF THE EXPOSED GROUPS

Shot 1 of Operation CASTLE was detonated on 1 March 1954. Following the detonation significant amounts of radioactive materials fell on the following populated neighboring atolls: (1) Rongelap, (2) Ailinginae, (3) Rongerik, and (4) Utirik. Exposure groups are identified according to their geographical location at the time of exposure. The numbers of individuals involved, their location, the distance of the atoll on which they were located from the site of the detonation on Bikini, the calculated dose of radiation, the probable time of beginning of the fall-out and its duration are tabulated in Table 1.1. The Rongelap group received the highest calculated dose. These individuals were living under relatively primitive conditions in lightly constructed palm houses (Fig. 1.1). The Ailinginae people were a part of the Rongelap group who were on their Ailinginae farms from the time the fallout began to the time of evacuation. Their calculated dose was smaller than that of the other members of their group that had remained on Rongelap. The third and largest group of Marshallese, inhabitants of the atoll of Utirik, received the smallest dose of radiation.

The American military personnel exposed on Rongerik were aware of the significance of fallout and promptly put on additional clothing to protect the skin. As far as duties would permit, they remained inside of Butler-type buildings. In contrast, most of the native Marshallese remained out of doors and thus were more heavily contaminated by the material falling on the atolls. Some of the Marshallese, however, went swimming during the fallout and many of the children waded in the water; thus washing a considerable amount of the material from their skins.

1.3 EVACUATION AND EARLY CARE OF THE EXPOSED GROUPS

The American military personnel were evacuated to Kwajalein via air in two groups on 2 March. The native Marshallese were evacuated by a combination of air and surface trans-

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portation. Since a survey of all individuals showed that there was significant contamination of skin and clothes, prompt decontamination was instituted. Clothes were removed and laundered. Their skin and hair were washed repeatedly with fresh water and soap. In many individuals, particularly in the Marshallese, it was difficult to wash the radioactive material from the hair because of the heavy coconut oil hair dressing used by these people.

Table 1.1 EXPOSED AND CONTROL UNEXPOSED GROUPS

Group Designation	Total Number in Group	Distance from Bikini (Naut. Miles)	Approximate Effective Time of Fallout	Time of Evacuation	Instrument Readings Used in Dose Calculations	Best Estimate of Total Gamma Dose in Air(r)*
Americans, Exposed Rongerik	28	135	H + 6.8 hr	H + 28.5 hr (8 men) H + 34 hr (20 men)	280 mr/hr H + 9 days	78
Americans, Control Kwaj American	105					
Marshallese, Exposed Rongelap	64	105	H + 4 hr	H + 50 hr (16 people) H + 51 hr (48 people)	375 mr/hr H + 7 days	175
Ailinginae	18	83	H + 4 hr	H + 58 hr	100 mr/hr H + 9 days	69
Ulirik	157	270	H + 22 hr	Started at H + 55 hr Completed at H + 78 hr	40 mr/hr H + 8 days	14
Marshallese, Control Majuro Group	117					
Total Exposed - 267						
Total Controls - 222						

* See Section 1.4.3.

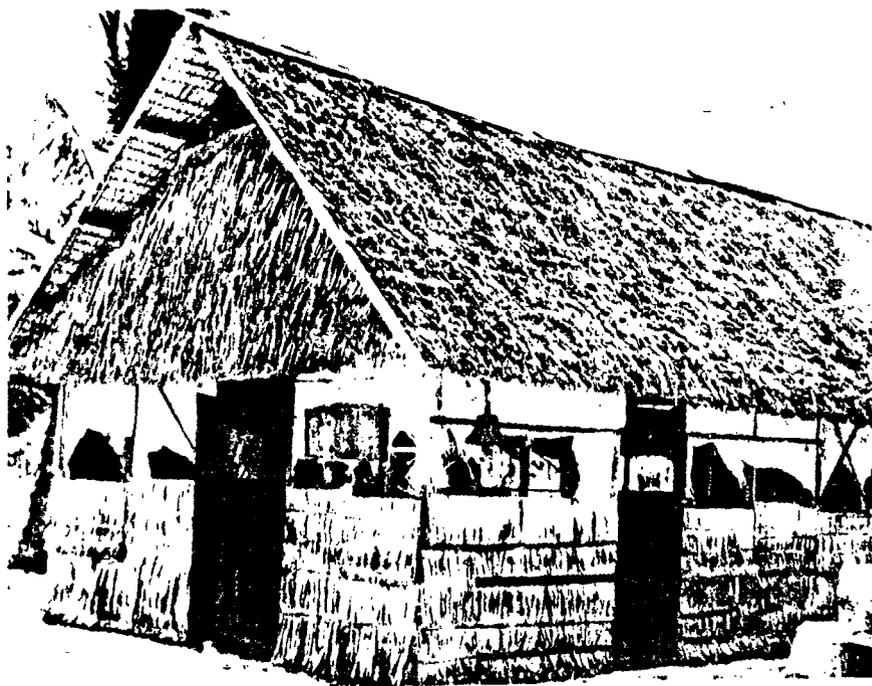
1.4 ESTIMATION OF WHOLE BODY EXTERNAL DOSE

The estimated values of external dose given in Table 1.1 were based on readings of AN PDR-39 field instruments. Averages of a number of dose rate measurements on each island at a given time were used. The readings were taken in air, approximately 3 feet above ground, several days after the inhabitants were evacuated. Before this time, adequate surveys with well calibrated instruments had not been possible.

Several variables which influence the results are indicated below. These will be discussed in greater detail in an addendum report on external dose, which will contain the data, methods and calculations by which the external dose analysis was made.

1.4.1 Energy Distribution of the Fallout Gamma Radiation, Its Variation With Time, the Response of the Meter in Each Energy Region and Its Correction Factor for the Total Dose Spectrum

Fallout deposited as an effectively infinite plane source resulted in the dose-energy histogram shown in Fig. 1.2. Its energy distribution was the result of degradation of the original



F.g. 1.1 The Living Area on Rongelap Island, Indicating the Light, Open-type of Construction

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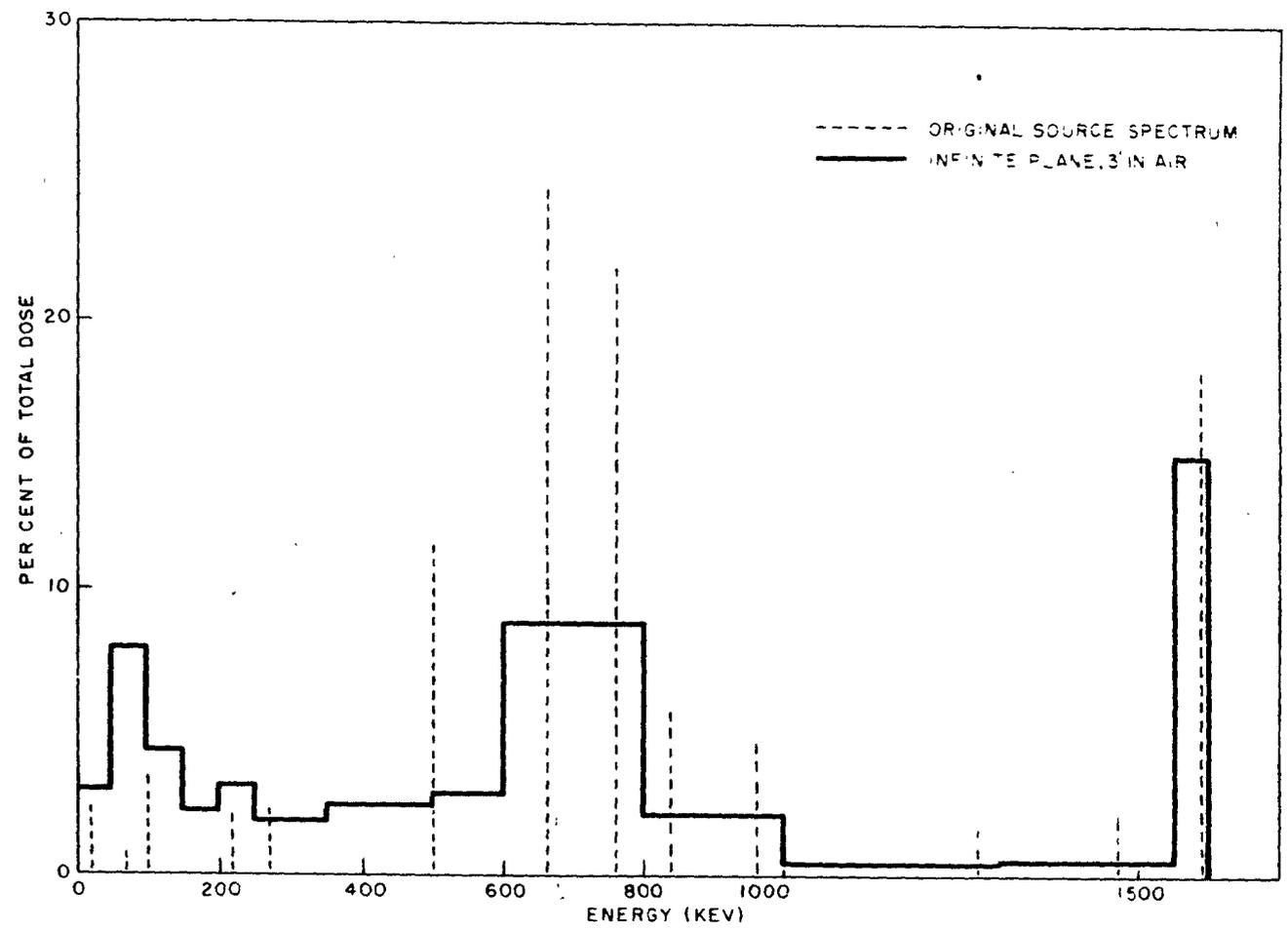


Fig. 1.2 Histogram of Proportion of Total Dose Contributed by Various Energy Regions from the Fallout Radiation

source energies, mainly by Compton scattering in air. The dose from each of the resulting energy intervals was calculated and plotted as a fraction of the total dose. This was seen to group roughly into three regions, with maxima at 100, 700, and 1500 kev. An exposure to such a source was thus the resultant effect of partial doses from each region, making the exposure energy conditions quite different from those of the clinic or laboratory.

Figure 1.2 illustrates the dose spectrum of 4-day-old fallout from a cloud sample. In the absence of other data to the contrary, this had to be taken as representative of the fallout on all of the islands. At this time the proportion of low energy component was at its maximum. During the several days before and after this time, the general shape of the spectrum apparently did not vary grossly from that illustrated here, since the observed flux decay rates closely followed that of the observed gamma dose rate. For the period between fallout and surveys, therefore, a knowledge of instrument response to each energy region allowed a total correction factor to be calculated. The instruments used were calibrated just prior to the surveys, and their readings have been corrected for the spectrum shape here illustrated.

1.4.2 Rate of Decay of the Fallout Mixture

Decay rates of fallout samples were measured in the field and in the laboratory, where a fairly consistent pattern was observed among various locations and samples. In addition, theoretical considerations based on the radiochemical composition of the fallout mixture permitted decay rates to be calculated for different intervals between the times of initial exposure and later survey readings. These agreed well with the experimental data, and were used both in the dose calculations during the exposure intervals and in extrapolating the later survey readings to earlier times.

1.4.3 Time of Arrival of the Radioactive Cloud, Duration of the Fallout, and Time of Evacuation for Each Case

Only the time of evacuation is known accurately for all the islands. On Rongerik, however, the time of arrival of the radioactive cloud was determined precisely by the continuously recording dose rate monitor at the weather station. The fallout became visible at the time the instrument first indicated the presence of a radiation field above background. The material had the appearance of snow. The times of beginning of fallout on Rongelap and Ailinginae were estimated from similar visual observations, combined with knowledge of the relative distances of these atolls from Bikini and the wind velocities in the area. Fallout was not observed on Utirik, hence the estimate of arrival time there was made on the basis of the Rongerik fallout time, wind, and distance factors.

Two extreme possibilities exist relative to the duration of the fallout: the first, that the fallout occurred entirely within a short time; the second, that it was gradual and extended over a period of many hours. The monitoring instrument on Rongerik went off-scale at 100 mr/hr, $\frac{1}{2}$ hour after the dose rate began to rise above background. If this rate of increase is extrapolated to a point for which subsequent decay would reduce the dose rate to the values found at later times, a long fallout is implied. This was taken as one limiting case, and corresponding doses were calculated. However, the possibility does not seem great that this actually occurred. Existing data are inconclusive, but several indications tending to favor the short time hypothesis are summarized below.

First: a long fallout probably would not be uniformly heavy throughout, the first portion being the most intense and the balance tailing off. The total phenomenon thus tends toward the effect of a shorter fallout. This is supported by monitor data from other nuclear events.

Second: the estimated durations of fallout, of about 18 hours, which result from the above extrapolation for Rongerik and Rongelap, appear too long to have occurred at the distances of these atolls from Bikini, since the wind velocity in the area was high enough for the cloud to pass over the islands in a considerably shorter time.

Third: the accounts of the visibility of the fallout, although conflicting, do not seem to indicate such late cessation.

Fourth: doses calculated on the long fallout hypothesis are lower than those due to a short fallout, since a short fallout quickly deposits a large amount of activity. On Rongerik, a set of film badge readings covered the range listed below. Several badges worn both outdoors and inside buildings on the island read 50-65 r, and one badge which remained outdoors over the 28.5 hr period read 98 r. Another group kept indoors inside a refrigerator read 38 r. These dose values represent a variety of conditions, but considering the shielding and attenuation factors, are consistent with the assumption that the dose reached the calculated upper limit outside, again favoring the shorter fallout hypothesis. The upper limit of 98 r will result if it is assumed that the fallout lasted one hour during which the intensities rose from zero to the maximum dose rate which then decayed to values observed later. A long fallout will not produce such a high dose of radiation.

Fifth: on Utirik, only a short fallout time is consistent with the later dose rates observed, provided the fallout began as late as was estimated from wind and distance factors. A one hour duration* of fallout appears likely. On the other islands the actual fallout time is known to have exceeded one hour; however, since the approximate dose discussed above was seen to fit the film data on Rongerik, it was used for the other islands as listed in the calculations in Table 1.1. The hour limit is thus "an effective value."

If the long fallout case is also considered, a lower limit for the dose may also be estimated, though the upper limit is taken as most probable. The ranges are then as follows: Rongerik 50 r - 104 r; Rongelap 102 r - 175 r; Ailinginae 53 r - 69 r; and Utirik - 14 r.

The dose value for Rongerik given in Table 1.1 is 75 per cent of the short fallout case value, averaged for 28.5 and 34 hour exposures. This best expresses the average air dose received by personnel who spent roughly half their time inside structures where the dose rate was later found to be roughly half that outdoors. On the other islands no such shielding was present.

Figure 1.3, for the Rongelap atoll, illustrates the cumulative dose as a function of time after the detonation. It can be seen that the rate of delivery of the dose varied continuously, the major portion being received at the higher dose rate prevailing in the early portion of the exposure period. By the time that 90 per cent of the dose had been received, for example, the dose rate had fallen to less than 30 per cent of its initial value. Thus the dose rate of exposure differed markedly from that usually encountered using x-ray units.

1.4.4 Geometry of the Exposures

A third difference between the type of exposure encountered here and other external exposures lay in the geometry of the source. These doses were delivered from a plane source, so that the radiation field did not follow the narrow beam geometry usually employed experimentally. In such a diffuse 360° field, the decrease of dose with depth in tissue is less pronounced than that resulting from a unilateral or bilateral exposure to an X-ray beam, so that for a given energy, the dose at the center of the abdomen is approximately 50 per cent higher than a given air dose would imply for the narrow beam case. Figure 1.4 illustrates an experimental simulation of the field geometry using a spherically oriented group of Co⁶⁰ sources with a phantom placed at their center, compared with a conventional depth dose curve obtained with a single source. It would appear under the circumstances that the midline dose, rather than dose measured in air, would be the better parameter in terms predicting biological effects. On this basis, the air dose values stated in Table 1.1 should be multiplied by approximately 1.5 in order to compare their effects to those of an exposure using a narrow beam geometry. If this is done, assuming a fast fallout of one hour, the following doses in terms of an air dose under laboratory conditions result: Rongelap 260 r; Ailinginae 100 r; Rongerik 120 r; and Utirik 21 r.

* While it is obvious that the fallout lasted longer than one hour, calculations of dose are based on an assumed one hour fallout as explained in the text.

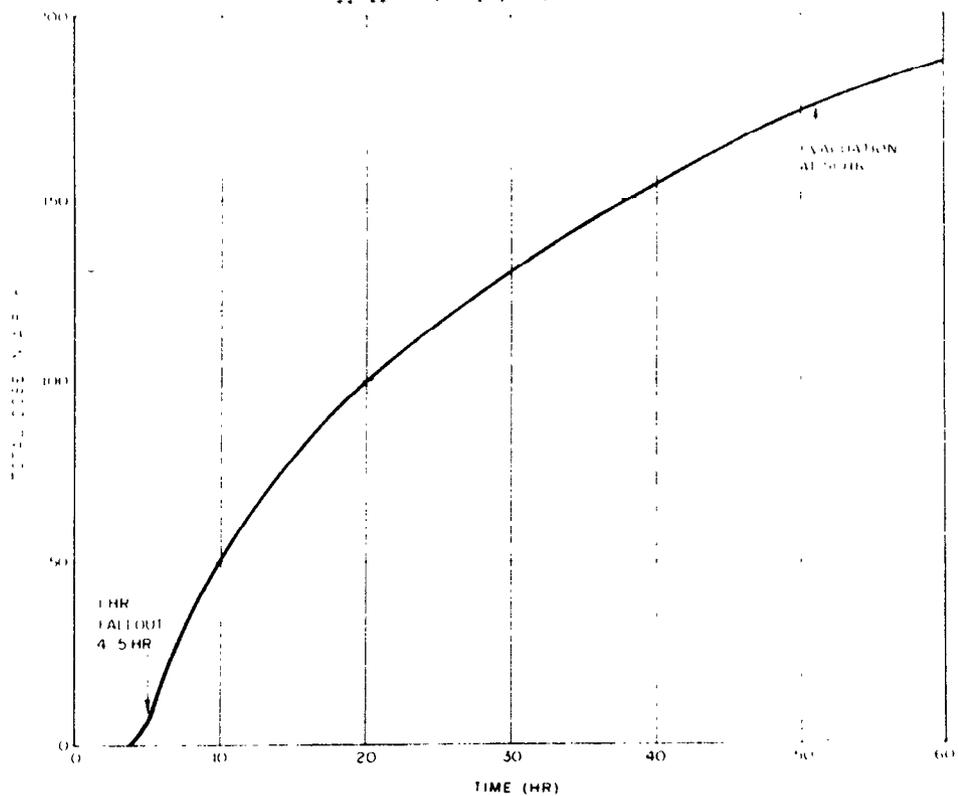


Fig. 1.3 Cumulative Dose as a Function of Time After the Detonation

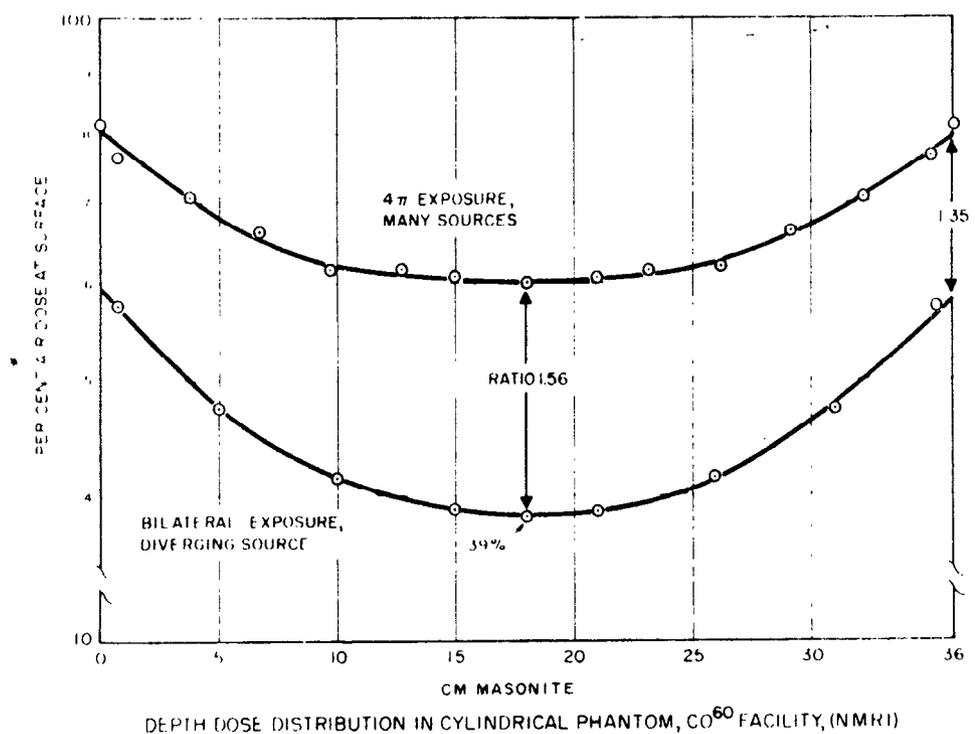


Fig. 1.4 Comparative Depth Doses for Bilateral Exposure from a Small Source and Multiple Source 4π Exposure with Cobalt-60

1.5 ESTIMATE OF MAXIMAL SKIN DOSE FROM THE GROUND

In addition to the total body gamma dose, the very soft gamma and higher energy beta radiation from the plane source contributed to the skin dose. Further skin irradiation resulted from local deposits of fallout material on the body surface itself. The latter is impossible to estimate, but the former may be roughly attempted as follows.

The beta dose rate in air at a height of 3 feet above the surface of an infinite plane contaminated with mixed 24-hour-old fission products is estimated to be about three times the

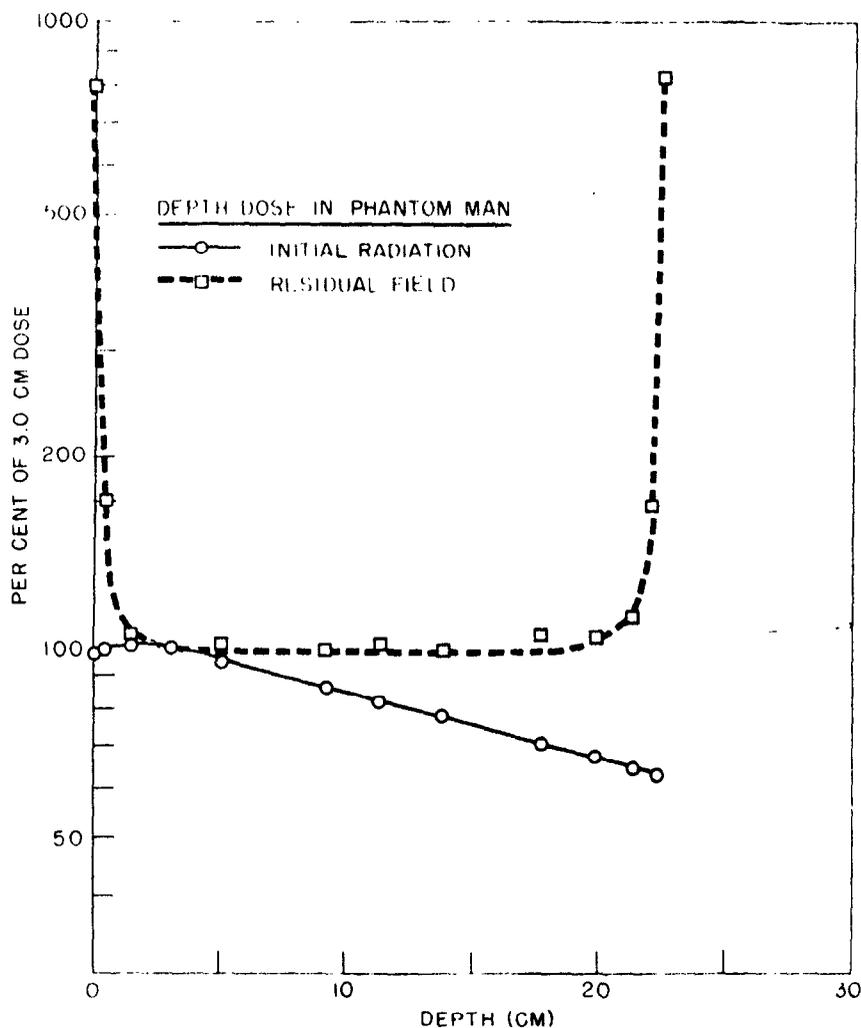


Fig. 1.5 Comparative Depth Doses in a Phantom Man of Initial Atomic Bomb Radiation and Radiation from a Field of Fission Products

gamma dose. The midline gamma dose is approximately 60 per cent of the portion of the air gamma dose due to 80 kv radiation or above. This portion in turn is estimated to be 90 per cent of the gamma dose measured in air by the instrument. Thus the dose at the surface of a phantom exposed to mixed fission product radiation from an external plane source might be expected to be $3/(0.6)(0.9)$ or about six times the midline dose, if both occur at 3 feet off the ground. Such a depth dose measurement has in fact been made experimentally at a previous

field test,* using a phantom man exposed to both the initial and residual radiation. The depth doses for each situation are shown in Fig. 1.5, with all data as per cent of the 3 centimeter dose. With the diverging initial radiation from the point of explosion, the exit dose was seen to be 63 per cent of the 3 cm dose, but with the diffuse residual field of fission product radiation, a surface dose some eight times greater than the 3 cm and deeper dose from the harder gamma components was observed. This is seen to be of the same order of magnitude as that estimated above. At heights above and below the 3 foot level this surface dose would become lower and higher respectively, but probably would not exceed 50 times the 3 foot air gamma dose or 80 times the midline dose, even in contact with the ground. An estimate of skin dose due to ground contamination for the Rongelap case would result, for example, in a figure of about 2000 rep to the dorsum of the foot, 600 rep at the hip level, and 300 rep at the head if continuous exposure with no shielding occurred. Some reduction in dose undoubtedly resulted from shielding and movement, and it seems probable that the external beta dose from local skin contamination far outweighed that from the ground in importance. This is emphasized by the fact that clothing probably reduced the beta dose from the ground by 10 to 20 per cent.

* F. W. Chambers, Project 2.2b, Residual Gamma Depth Dose Measurements in Unit-Density Material, AFSWP, WT-719, Operation UPHOT-KNOTHOLE.

CHAPTER 2

CLINICAL OBSERVATIONS AND THERAPY

2.1 INTRODUCTION

It was known immediately that the exposed groups had received a significant amount of penetrating radiation to the entire body, extensive contamination of the skin, and possible internal deposition of radioactive materials. It was therefore decided that clinical observations would be as extensive and frequent as facilities and personnel permitted in order to recognize and care for radiation effects as early as possible. Accordingly complete initial histories and physical examinations with numerous follow-up examinations were carried out. Surveys of the skin were conducted at frequent intervals and the detailed skin findings are reported in chapter 3. Extensive hematological studies were conducted, the detailed results of which are presented in chapter 4. Results of examinations for urinary excretion of radioisotopes are reported in chapter 5.

In addition to periodic examinations, routine sick call was held twice daily. Medical care was available at all times and hospital facilities were available at the Kwajalein Naval Dispensary.

In view of the widespread conflicting opinions in regard to the value of various prophylactic and therapeutic measures in treatment of radiation effects, it was decided in advance that therapy would not be given arbitrarily but would be instituted as indicated clinically for specific conditions on an individual basis. However, if severe granulocytopenia developed (below 1000 cells/cm) the prophylactic use of antibiotics was to be considered. Whole blood transfusions were likewise to be used only in case of development of serious anemia.

2.2 SYMPTOMS AND SIGNS RELATED TO RADIATION INJURY

Several symptoms that developed during the first day or two after exposure probably were attributable to radiation. Itching and burning of the skin and eyes during this period occurred in over one quarter of the Rongelap population, to a lesser extent in the Ailinginae and to a very slight extent in the Americans. The skin symptomatology* might have been due in part to the marked alkalinity of the fallout material (calcium oxide). About two thirds of the Rongelap group reported nausea during this early period and one tenth of the group reported vomiting and diarrhea. Only one Ailinginae individual reported nausea. The people of Utirik and the Americans developed no signs or symptoms that might be related to radiation.

*The symptomatology is based on questionings through an interpreter by several observers. Despite the repeated interrogations and the inevitable suggestion of the interrogators, the stories remained remarkably consistent.

With the exception of the development of skin lesions and epilation, physical examinations at no time revealed findings in any group that could be attributed with certainty to radiation. The various clinical conditions encountered in the most severely exposed groups were not remarkably different from those seen in the least exposed Utrik group. The skin lesions and epilation appeared about the 12th to 14th post-exposure days.

2.3 CLINICAL OBSERVATION AND THERAPY AS RELATED TO HEMATOLOGICAL FINDINGS

Although detailed hematological findings are presented in chapter 4, certain considerations of the relation of clinical to hematological findings are discussed here.

2.3.1 Leukocyte Counts

Between the 33rd and 43rd post-exposure days, 10 per cent of the Rongelap individuals reached an absolute granulocyte level of 1000 per cubic millimeter or below. The lowest count observed during this period was 700 granulocytes. During this interval the advisability of giving prophylactic antibiotic therapy to such individuals was carefully reconsidered. However, prophylactic antibiotics were not instituted because of the following considerations:

(1) All individuals were under continuous medical observation so that infection would be discovered in its earliest stages.

(2) Since some individuals might require antibiotics for long periods of time if infection occurred, the premature administration of such drugs would not only possibly obscure medical indication of treatment, but might potentially lead to the development of drug resistant organisms in an individual with an already lowered resistance to infection.

(3) There is no accurate knowledge of the number of granulocytes required by man to prevent the types of infection seen in agranulocytosis.

(4) The observed situation was not strictly comparable to agranulocytosis with an aplastic marrow due to potentially lethal doses of radiation. In the latter instance, granulocytes fall rapidly and there are practically none in the circulation when infection occurs. In the present group of individuals exposed to radiation, although most counts were approximately one-fourth of normal, the fall to that level was gradual and there was some evidence of granulocyte regeneration.

White counts were repeated at three or four day intervals on all of the exposed individuals and more frequently on those with the lowest counts. Those with symptoms or elevated temperatures were treated only after an attempt to establish a diagnosis was made, even if a period of observation was necessary. During the observation period, the patients were examined at frequent intervals and the temperature checked every few hours.

Twenty-seven individuals had total leukocyte counts of 4000 or below or absolute neutrophil counts of 2500 or less at some time during the period of observation. Of these 27, 13 had symptoms of disease that required evaluation for possible antibiotic therapy. Eleven of the 13 had severe upper respiratory infections, one individual had abdominal pains and fever, and one had urticaria (hives) with fever. The incidence and type of symptoms in the group with low leukocyte counts did not differ materially from those in the remainder of the population and it appeared that the occurrence of disease and the presence of leukopenia was coincidental. The 13 instances in which it was necessary to consider the use of antibiotic therapy in neutropenic individuals are summarized below:

Eleven of the 13 individuals had severe upper respiratory infections. Eight of these had malaise, sore throat, nasal discharge, and temperatures that varied between 99 and 101.4°F and then fell to normal within a 12 hour period. Since the response of this group to upper respiratory infection appeared identical with that of individuals without neutropenia, no therapy was given. Two of the 11 had marked malaise, headache, abdominal pain, nausea, diarrhea, and high fever. Both were children, one age seven, the other age two. In both instances, the symptoms were out of proportion to the physical findings, which were negative except for head colds and mild pharyngeal injection. The seven-year old child had a temperature of 102.6 when first seen

and 4 hours later, 104. The two-year old child had an initial temperature of 101.8 degrees which rose to 103.5 degrees in 4 hours. Both were given intramuscular procaine penicillin when the sharp rise in temperature occurred, and both were without symptoms and fever the following day. A second injection of penicillin was given at this time, and therapy was discontinued. In spite of the fact that the neutrophils remained depressed in both cases long after the fever had passed, both individuals recovered and had no further illness. In Figure 2.1 the blood counts and platelets of the two-year old patient and the time of the occurrence of the illness with fever are illustrated. A one-year old boy had symptoms of upper respiratory infection for several days before being brought to the clinic. When he was seen, his temperature was 100.8 degrees. In addition to the head colds there was pharyngeal injection and numerous coarse rhonchi (wheezing) throughout the chest. A diagnosis of upper respiratory infection with associated bronchitis was made and the child was given a single intramuscular injection of procaine penicillin. On the following day no rales or rhonchi were heard, the temperature was 99°F, and he remained asymptomatic without further treatment.

One of the 13 individuals was a 50-year old man who reported to the clinic with weakness, nervousness, mild abdominal pain, and shooting pain in the upper anterior chest, bilaterally. He appeared moderately ill, his temperature was 99.6°F, and the only positive physical finding was moderate tenderness in the right upper quadrant of the abdomen. Since his granulocyte count remained low on repeated examination, he was seen at frequent intervals. Within a 10 hour period the temperature rose to 101.6°F, following which it fell gradually to normal. The abdominal tenderness continued for 24 hours and then gradually disappeared during the subsequent two days. A diagnosis of cholecystitis (inflammation of the gall bladder) was made. No specific therapy was given. In Figure 2.2 his white blood count and platelet counts in relation to the appearance of symptoms are shown.

A female, 38, developed generalized urticaria, fever, and headache. Urticaria and fever subsided within 8 hours without any therapy.

2.3.2 Platelet Counts

All individuals with a platelet count of 100,000 or less were examined daily for evidence of hemorrhage into the skin, mucous membranes, and retinae. Urine was examined for red cells and albumin, and women were questioned concerning excessive menstruation. There was no evidence of hemorrhage into tissues even though 11 individuals reached platelet levels between 35,000 and 65,000. Two women menstruated when their platelet counts were 150,000 and 130,000 respectively. Both menstruated several extra days and thought that the bleeding was excessive but not sufficient to cause concern.

2.3.3 Hematocrit Changes

Nineteen individuals in the Rongelap group had hematocrits of 35 per cent or below; however, none of these were below 31 per cent. Nine of the 19 were children, aged one to five years who would be expected to have a lower hematocrit than normal adults; four were over 70 years of age, in which age group a decreased hematocrit is frequently present without obvious cause. Two of the 19 had had menorrhagia (profuse menstruation) prior to the determination, two were three to four months pregnant and had not received supplementary iron, and two were young women. The low hematocrits that were observed could be explained on the basis of normal physiological variations rather than to the effects of irradiation on hematopoiesis (blood formation). At no time were whole blood transfusions considered since hematocrits remained within levels consistent with well being and normal activity.

2.4 THE EFFECT OF AN EPIDEMIC OF UPPER RESPIRATORY INFECTION ON AN IRRADIATED POPULATION

Between the 27th and 42nd post-exposure days an epidemic of upper respiratory diseases (URI) occurred. Fifty-eight per cent of the Rongelap group and 56 per cent of the Ailinginae group were involved. Seventy per cent of the affected individuals developed symptoms between

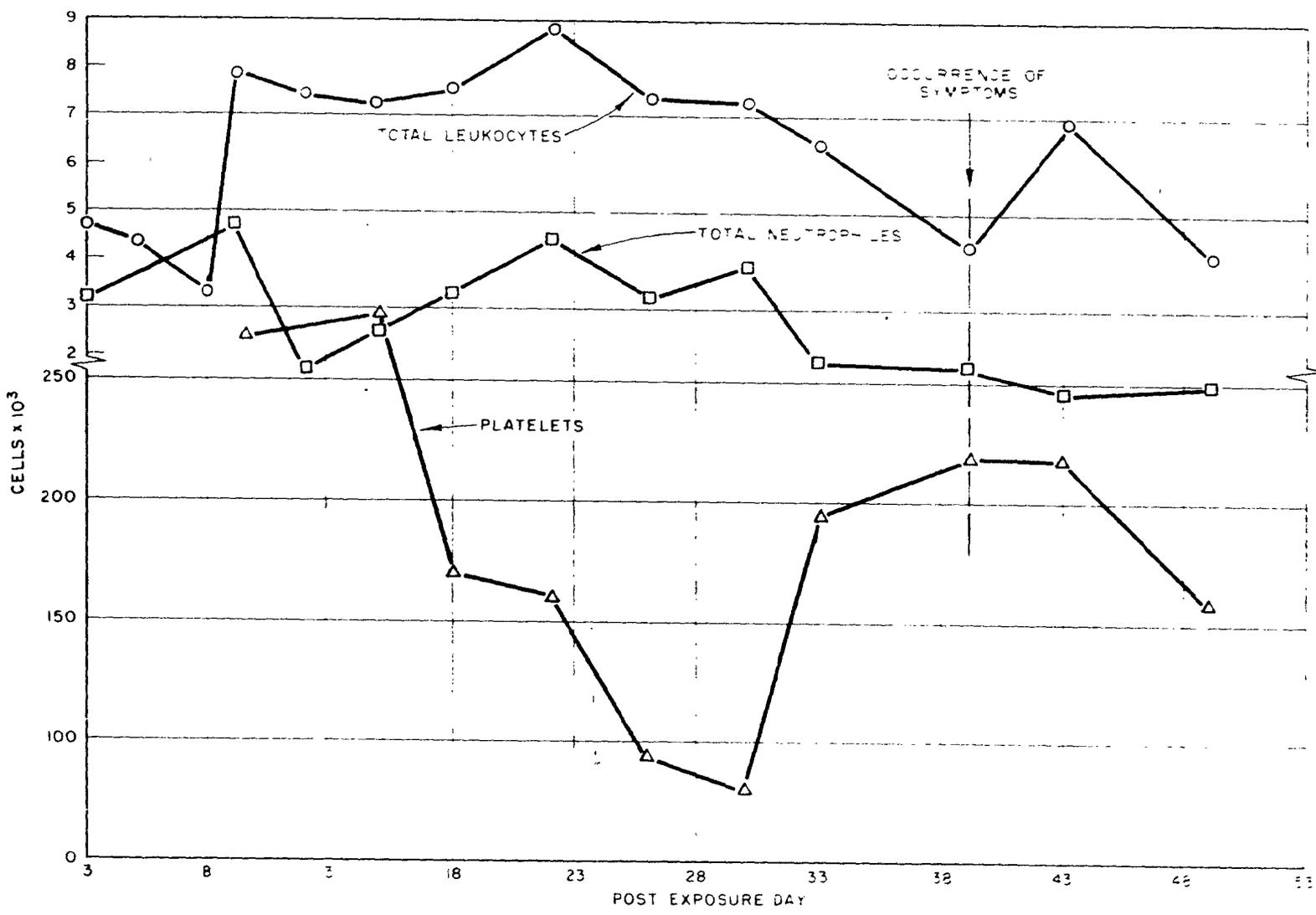


Fig. 2.1 The Blood Count Changes in a Two Year Old Child with Fever

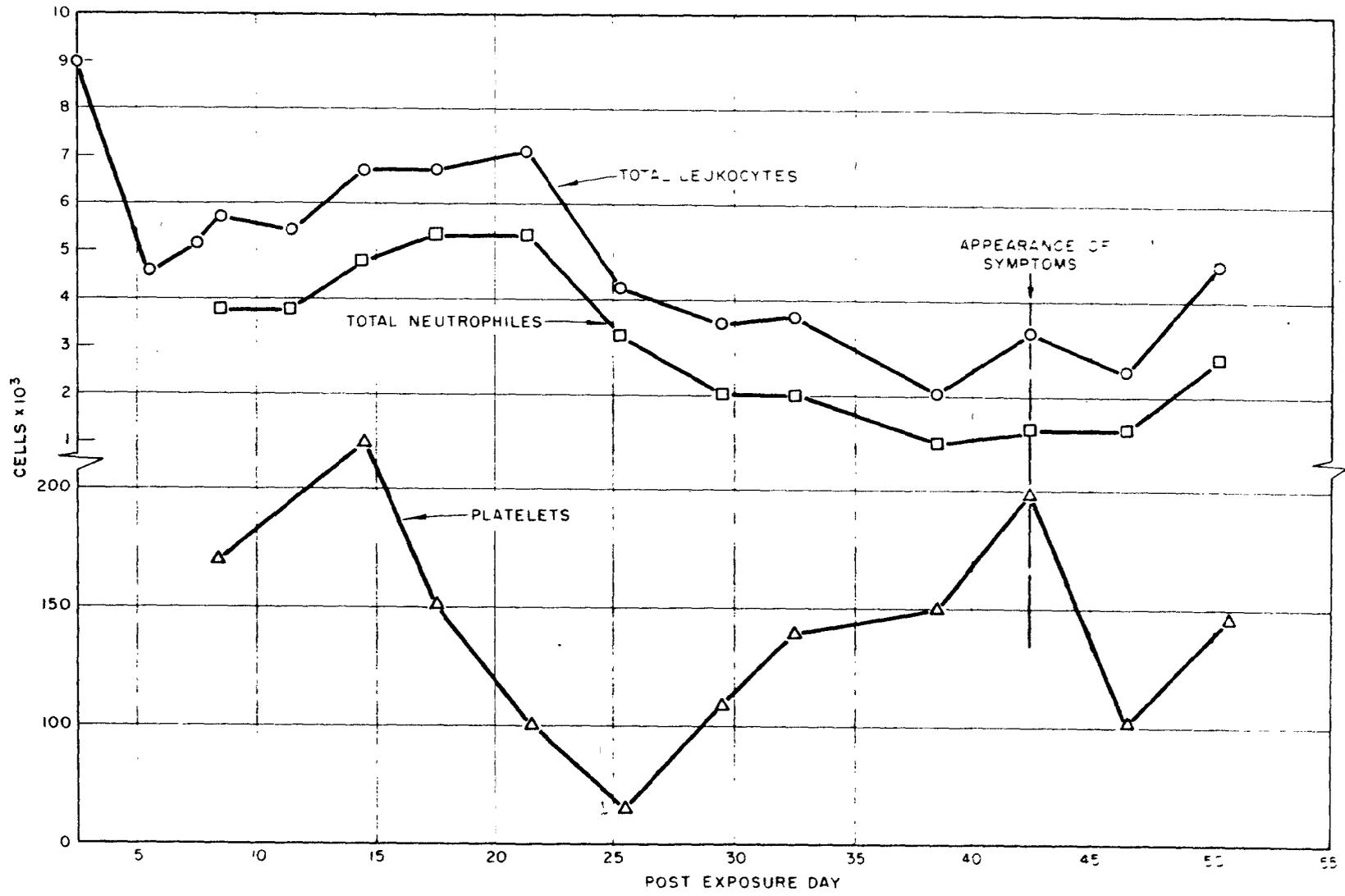


Fig. 2.2 The Blood Count Changes in a 50 Year Old Man with Fever

the 27th and 32nd post-exposure days. During the period in which individuals were developing symptoms of the respiratory infection, the leukocyte count of some diverged from the population trend. Fifty-two per cent of the leukocyte counts were observed to increase to higher levels, the increase being due primarily to granulocytes (see chapter 4). Since the increase in the mean granulocyte count of the entire population occurred about the time the epidemic of URI developed, it seemed pertinent to determine whether in individual instances the increase was spontaneous or was in some way related to the presence of respiratory infection. The relationship between the observed leukocyte increase and the presence or absence of upper respiratory symptoms in the individuals exposed to radiation suggested that the two effects were unrelated.

Table 2.1 DISEASES THAT APPEARED DURING OBSERVATION OF THE RONGELAP AND AILINGINAE GROUPS

Disease	Number of Individuals	Disease	Number of Individuals
Furuncle (boils)	2	Bronchitis	1
Gum Abscess	1	Appetous ulcer of tongue (canker sore)	1
Cholecystitis (inflammation of gall bladder)	1	Spondylolisthesis (malformation of vertebra)	1
Tinea (ringworm)	1	Impetigo	5
Mittelschmerz (intermenstrual pain)	1	Tooth extractions	2
Generalized urticaria (hives)	1	Gastroenteritis (inflammation of stomach and intestines)	10
Erythema multiforme (red rash)	1	Upper respiratory infections	47
Migraine headache	1	Follicular tonsillitis	1

Of the 64 individuals from Rongelap, 27 had no respiratory infection and of these 13 (48 per cent) without URI showed a rise in leukocytes; 37 had the infection, and of these, 24 (53 per cent) showed a rise; 7 of these 24 showed a rise three or more days before symptoms appeared. Of the 18 from Ailinginae, 8 had no respiratory infection and 3 (37 per cent) of these showed a rise; 10 had the infection, of these, 3 (33 per cent) showed a rise in count.

It is also of interest that not only the irradiated individuals developed the respiratory infection but in addition the medical personnel involved in their care and study also developed equally severe respiratory infections. The respiratory infections consisted of moderate malaise, sore throat with prominent lymphoid follicles, pharyngitis, moderate fever on the first day, and a purulent (pus) nasal and tracheal discharge for 10 days.

2.5 COMPARISON WITH THE UTIRIK GROUP OF DISEASES SEEN IN THE RONGELAP AND AILINGINAE GROUPS DURING THE PERIOD OF OBSERVATION

The diseases that were seen in the Rongelap and Ailinginae groups during the period of observation are listed in Table 2.1. None of these appeared to be related to the effects of irradiation, either directly or as a result of the hematologic disturbances. For comparison, the diseases that were seen in the Utirik group during the period of observation are listed in Table 2.2. The high incidence of gastroenteritis (inflammation of the stomach and intestines) in both groups was probably due to the Marshallese keeping perishable foods unrefrigerated for long periods, and was not seen after this practice was stopped. It would appear that a higher percentage of the Rongelap-Ailinginae group developed upper respiratory infections compared to the Utirik group. However, all of the Rongelap-Ailinginae group were questioned concerning even mild symptoms of URI, whereas only those of the Utirik group with severe symptoms of

Table 2.2 DISEASES THAT APPEARED DURING OBSERVATION
OF THE UTIRIK GROUP

Disease	Number of Cases
Osteoarthritis (joint disease)	4
Epithelioma of ankle, with necrotic degeneration (cancer)	1
Chronic bronchitis	1
Furuncle of calf	1
Chronic bronchitis and bronchiectasis	1
Abscess of sole of foot	1
Carbuncle on right thigh	1
Tooth extraction	1
Fungus infection of gums and palate	1
Confusion, traumatic	2
Gastroenteritis	30
Upper respiratory infections	15
Arteriosclerotic heart disease, decompensated	1
Pyelonephritis (inflammation of kidney)	1
Insect bite, with marked palpebral edema	1
Chorioretinitis, unknown etiology (retinal inflammation)	1
Thrombophlebitis, antecubital vein (inflammation of vein)	1
Impetigo	3
Dysmenorrhea (painful menstruation)	1
Exfoliative dermatophytosis (scaling fungus infection of skin)	1
Ectropion, right eye (malformed eyelid)	1
Asthma	1
Benign hypertension with headache (high blood pressure)	1
Hypermetropia with headache (far sightedness)	1
Myopia with headache (near sightedness)	1
Fungus infection, auditory canal	1
Trichomonas with cystitis (inflammation of bladder)	1
Tinea (ringworm)	1
Simple headache	3
Bronchitis	1
Possible ruptured intervertebral disc	1
Fevers of unknown origin	1
Mongolian idiot	1
Pregnancy	6

URI came to the clinic for treatment. Similarly, the per cent of the Rongelap-Ailinginae group that developed purulent infections appears higher than the Utirik group. However, the diseases are tabulated for the period of observation of each group, and the Utirik group was observed for only half as long since they were moved to another island when it was evident that further study was unnecessary.

2.6 CHANGES IN WEIGHT AS AN INDICATION OF DISTURBANCE IN THE OVERALL METABOLISM

The body weight of individuals in the Rongelap-Ailinginae group was followed routinely. Since they had an unrestricted diet and all ate well, their change in weight might be taken as an



indication of any disturbance in their over-all metabolism. The weight changes are summarized in Table 2.3. It would be expected that within a period of six weeks, individuals below 16 years and particularly those below 8 years would gain some weight. The fact that most of them lost weight may indicate that they received a dose of radiation sufficient to interfere with normal metabolism. In spite of their relatively inactive life and hearty appetites, most of the adults also lost weight which may indicate some interference with their normal metabolism. There is little difference in observed weights between the Rongelap and Ailinginae groups. It appears that the difference in doses received by the two groups did not differentially affect their body weight. Whether the failure to gain weight was connected with radiation or changes in environ-

Table 2.3 WEIGHT CHANGES, RONGELAP AND AILINGINAE GROUPS

	Age Categories		
	Below 7 years	Below 16 years	Above 16 years
	Rongelap Group		
Number observed	17	24	36
Number that gained weight	4	5	14
Average gain (lb)	5	3	3.5
Spread of gain (lb)	0.5-10.0	0.5-10.0	1-11.5
Number that lost weight	13	19	21
Average loss (lb)	2	2	4
Spread of loss (lb)	0.5-5.5	0.5-5.5	0.5-8
Per cent of group that lost weight	77	80	58
	Ailinginae Group		
Number observed	7		9
Number that gained weight	0		3
Average gain (lb)			2.7
Spread of gain (lb)			2-4
Number that lost weight	6		6
Average loss (lb)	2		2
Spread of loss (lb)	0.5-3		0.5-4.0
Per cent of group that lost weight	88		67

ment is open to question. Unfortunately, the weight of the individuals from Utirik was not systematically followed and no satisfactory control exists to aid in interpreting the loss in weight of the Rongelap-Ailinginae group.

2.7 THE EFFECTS ON PREGNANCY

Four women in the Rongelap group were pregnant when brought to Kwajalein. Two were in the first trimester, one in the second trimester, and one in the third trimester. None of these women had abnormal symptoms referable to pregnancy, and as far as can be determined pregnancy continued in a normal fashion. In the Ailinginae group, one woman was in the second trimester. No abnormality was detected. Fetal movements were unaffected in the individual in the third trimester. The hematologic changes of the pregnant women are listed in Table 2.4. Two individuals in the first trimester had marked depression of platelets but at no time was

Table 2.4 BLOOD COUNTS ON PREGNANT INDIVIDUALS
RONGELAP AND AILINGINAE GROUPS

Trimester of Pregnancy	Lowest Platelet Count	Lowest WBC	Lowest Neutrophile Count
Rongelap Group			
First	35,000	4,500	3,000
First	50,000	5,000	2,500
Second	150,000	4,000	3,000
Third	120,000	10,000	7,000
Ailinginae Group			
Second	170,000	7,000	3,200

Table 2.5 PATIENTS TREATED WITH ANTIBIOTICS

Patient No.	Condition	No. of days treated	Antibiotic used
1	Deep extensive slough of epidermis of foot	2	Penicillin
2	Tooth extraction	1	Penicillin
3	Tooth extraction	1	Penicillin
4	Inflamed tonsils with high temperature and URI	2	Penicillin
5	URI and bronchitis with high temperature	1	Penicillin
6	URI, severe, with pharyngitis and high temperature	2	Penicillin
7	Rapid progressing undermining impetigo	2	Penicillin
8	Traumatic gangrene of foot	7	Penicillin
9	Cystitis	5	Gantrisin
10	Furuncle on buttock	2	Penicillin
11	Furuncle on forehead	1	Penicillin

there any vaginal bleeding. However, all of the pregnant women were blood typed as a precautionary measure. So far, the exposure to radiation has not had a deleterious effect on pregnancy. Since the departure of the medical group from Kwajalein, one apparently normal baby has been born.

2.8 DISCUSSION ON THE USE OF ANTIBIOTICS IN THIS GROUP AND IN RADIATION INJURY

There were few indications for the use of antibiotics when the principles of treatment previously mentioned were used. Individuals in the Rongelap and Ailinginae groups that received antibiotics are listed in Table 2.5. Of the individuals treated with the antibiotics, the first three received it prophylactically and it would have been indicated had they not been irradiated. The indication for its use in the next three was questionable. There was a definite indication for its use only in the next three individuals and again it would have been given for similar conditions in unirradiated individuals. The last two individuals with furuncles (boils) would probably have responded equally well to surgical treatment alone, since the infection was well localized and easily drained.

In general, it can be concluded that human beings with the degree of depression of hematopoiesis noted did not need antibiotics prophylactically. With severer degrees of radiation injury resulting in a greater depression of hematopoiesis, prophylactic antibiotic therapy may be indicated in selected cases. If the number of individuals is small enough so that changes in the blood count, temperature, and clinical evidence of infection can be followed closely, it would appear that antibiotics should not be started until there is a clear-cut clinical indication for the use of these agents.

CHAPTER 3

SKIN LESIONS, EPILATION, AND NAIL PIGMENTATION

3.1 INTRODUCTION: EARLY SYMPTOMATOLOGY REFERABLE TO THE SKIN

The Marshallese on Rongelap saw a visible fallout of powdery material that began approximately five hours after the initial flash was seen. The powder whitened the hair and adhered to their skin as a salt-like film. The Marshallese on Ailinginae reported a similar but less striking fallout. The Americans on Rongerik also saw a fallout and described it as "mist-like." The Marshallese on Utrik did not see a fallout. The early symptoms were limited to the Rongelap, Ailinginae, and to a lesser extent the Americans on Rongerik. The early symptoms consisted of a generalized itching and burning of the skin, limited almost exclusively to the exposed parts of the body. A less consistent symptom was burning of the eyes accompanied by tears. The symptoms began the night of the fallout and continued into the next day. A few individuals had symptoms lasting as long as two or three days. Decontamination of the skin of the exposed individuals was initiated either aboard the destroyers while they were being evacuated or upon their arrival at the naval base on Kwajalein. The classical initial erythema of radiation injury to the skin was not noticed by the observers who examined these individuals during the first 10 days. If an initial erythema developed in the native Marshallese, it was masked by their dark skin. An erythema was not seen in the white skinned Americans exposed on Rongerik.

3.2 SKIN LESIONS

3.2.1 General Description

Skin lesions first appeared in the Rongelap group after 12 to 15 days and in the Ailinginae and Rongerik groups after 20 days. There were no skin lesions in the Utrik group. There was considerable difference in the length of time necessary for the development of the various lesions. However, it was found that there was a consistent pattern in the sequential development of lesions on various exposed parts of the body. The principal lesions occurred roughly in the following sequential order: scalp (with epilation); neck, axillary region, antecubital fossae, feet, arms, legs, and trunk. Lesions on the flexor surfaces tended to precede those on the extensor surfaces (see Table 3.1 for the time of appearance of various lesions).

A clear cut primary erythema was not seen, nor was a late erythema. In a few cases, there was considerable scratching of the skin due to intense itching prior to development of gross lesions. In these cases, an erythema of questionable etiology was observed. This erythema may well have been due to the scratching. Erythema was likewise not observed preceding development of lesions in the white and Negro Americans of the Rongerik group.

The first indication of a developing lesion was an increase in pigmentation. These pigmented areas appeared in the form of macules, papules, raised plaques, or larger areas of

hyperpigmentation. The macules and papules (1 to 2 mm in diameter) usually occurred in clusters and sometimes coalesced into larger lesions. The latter were characteristically found on the scalp, neck, and the antecubital fossae. The raised plaques varied in size from a few millimeters to several centimeters in diameter, were thick, rough, dry, and had a leathery feel. This type of lesion occurred predominantly on the feet and in the antecubital fossae and to a much lesser extent on the neck. Areas of increased pigmentation also occurred on the limbs, trunk, and on the face.

The majority of the lesions were superficial without blisters. A few days after appearance, dry, scaly desquamation occurred in the central portion of the hyperpigmented areas. The desquamation left a pink to white epithelium not remarkably different in texture from the sur-

Table 3.1 LESIONS IN RONGELAP GROUP

Type of Lesion	Per Cent of Total in Age Group having Indicated Lesion				Median Time of First Observation of Lesions*
	Age 0-5 (13 people)	Age 6-15 (13 people)	Age 16 & Over (38 people)	Total Group (64 people)	
Epilation					
1 plus	7.6	38.4	13.8	17.2	17
2 plus	38.6	30.7	5.5	17.2	17
3 plus	53.8	23.0	8.3	22.0	16
Total	100.0	92.1	27.6	56.2	16
Skin Lesions					
Anus-Groin	38.4	0.0	0.0	7.8	17
Scalp	100.0	100.0	37.0	62.5	18
Neck	69.2	76.9	68.0	70.3	21
Axilla	61.5	7.6	15.7	23.4	21
Antecubital Fossae	30.7	38.4	34.2	34.4	28
Hands-Wrists	30.7	23.0	18.4	21.8	33
Feet	23.0	53.8	53.0	45.3	28
Arms	15.3	15.3	10.3	12.5	31
Legs	7.6	23.0	4.3	7.8	33
Trunk	15.3	23.0	4.3	9.4	33
Nail Pigmentation	61.5	100.0	95.0	89.0	38

* Post-exposure days.

rounding skin. As the desquamation proceeded outward, the areas developed a characteristic appearance of a central depigmented area fringed with a hyperpigmented zone. At a later stage, pigmentation began in the central areas and spread outwards. After a few weeks the cycle was completed, leaving in most instances a relatively normal appearing skin.

Approximately 20 per cent of the Rongelap group developed lesions which were more severe. These lesions might be considered as comparable to second degree thermal burns. The deeper lesions occurred principally on the feet and to a lesser extent on the scalp and neck and in one case on the ear. Blister formation was not common. However, on the feet, some large bullae (blisters) appeared. After a few days, the hyperpigmented lesions showed wet desquamation with weeping and crusting, leaving depigmented raw surfaces of varying area. Some of these lesions became secondarily infected. Epithelium rapidly covered the ulcerated areas within a week to 10 days. Pigmentation followed during the next few weeks. As healing occurred many of the more severe lesions (particularly on the neck and antecubital fossae) developed a thickening of the skin with an "orange peel" appearance and a dusky, grayish-brown color (see Plate 3.4).

In Table 3.1 is tabulated the incidence of the various types of lesions in the Rongelap group with a breakdown as to age and the median time of appearance. Similar data are presented in Table 3.2 for the Ailinginae and Rongerik groups.

3.2.2 Description and Illustration of Specific Lesions

The scalp and forehead lesions appeared 12 to 14 days following exposure along with epilation in the Rongelap group. The skin lesions were maculopapular with a spotty distribution. They were concentrated in the areas of epilation. These lesions are illustrated in Plates 3.12 to 3.16. Scalp lesions continued their appearance over a period of about a month. Thus, groups of lesions in various stages were present in the same individual. This was particularly notable among the children. The incidence of scalp lesions was greatest in the 0 to 15 year group (see Table 3.1).

The neck lesions were the most common and began their appearance a few days after the scalp lesions. These lesions appeared as hyperpigmented macules and papules which spread and coalesced into raised plaques. The lesions usually appeared first on the side and front of the neck and spread backwards. They were more common and more severe in women. Plates 3.1 through 3.4 and 3.9 illustrate neck lesions in the various stages of development. Some of the deeper neck lesions tended to occur in women where their thick hair touched their necks.

Axillary lesions were maculopapular, less abundant, and developed simultaneously with the neck lesions. These lesions are illustrated in Plates 3.10 and 3.11. The axillary lesions were more common in the young children.

Antecubital fossae lesions appeared about a week later than did the neck and axillary lesions. These are illustrated in Plates 3.9 and 3.10.

The foot lesions developed later than the lesions of the antecubital fossae. These lesions were located mainly on the dorsum of the foot between or on the toes. They were initially characterized by large pigmented plaques with subsequent bullous formation and in eight cases raw crusting lesions of varying degree followed the bullae. The foot lesions were not as common in children under 5 years of age as in the older age groups. Sequential lesions in a 14-year old girl are shown on Plates 3.5 to 3.7. One of the more severe foot lesions is shown in Plate 3.8.

Lesions of the hands, arms, legs, and trunk were less common, less severe, and developed later. Areas of increased pigmentation were scattered over the abdomen, chest, arms, legs, and face. Increased pigmentation of the sides of the face is illustrated in Plate 3.4. A striking early lesion was an erythematous, weeping, excoriating lesion surrounding the anus which occurred in several of the babies and a few of the older people. These lesions were severe initially but healed rapidly.

Most of the Marshallese had multiple lesions. The combination of epilation and the contrasting hyperpigmented and depigmented areas adjacent to normal skin presented a striking appearance. The multiple lesions are illustrated in Plates 3.8 to 3.11.

3.2.3 Severity and Time Appearance of Lesions in the Various Exposure Groups

It is not feasible to quantify accurately the severity and extensiveness of the skin lesions in the various groups. However, it was the uniform opinion of all observers that the most severe and extensive lesions occurred in the Rongelap group. The lesions that developed in the Ailinginae group were much less severe and extensive, and the Rongerik group (Americans) had only mild lesions. Skin lesions were completely absent in 60 per cent of the Rongerik group, as opposed to approximately 10 per cent in both the Rongelap and Ailinginae groups. Transepidermal necrosis occurred in 20 per cent of the Rongelap and in 5 per cent of the Ailinginae people. No lesions of this severity were seen in the Rongerik group.

The lesions appeared earlier in the Rongelap group suggesting a higher dose. The comparison of the incidence of epilation and neck lesions and time of appearance is illustrated in Fig. 3.1 for the Rongelap and Ailinginae groups.



Plate 3.1 Early hyperpigmented maculopapular neck lesions at 15 days. Case 39, age 15, F.



Plate 3.2 Neck lesions at 28 days. Wet desquamation. White color is calamine lotion. Case 78, age 37, F.



Plate 3.3 Repigmenting superficial neck lesions at 40 days. Hyperpigmented areas not completely desquamated. Case 24, age 15, F.



Plate 3.4 Healed neck lesions at 77 days showing dusky pigmentation of back of neck. Case 39, age 15, F.



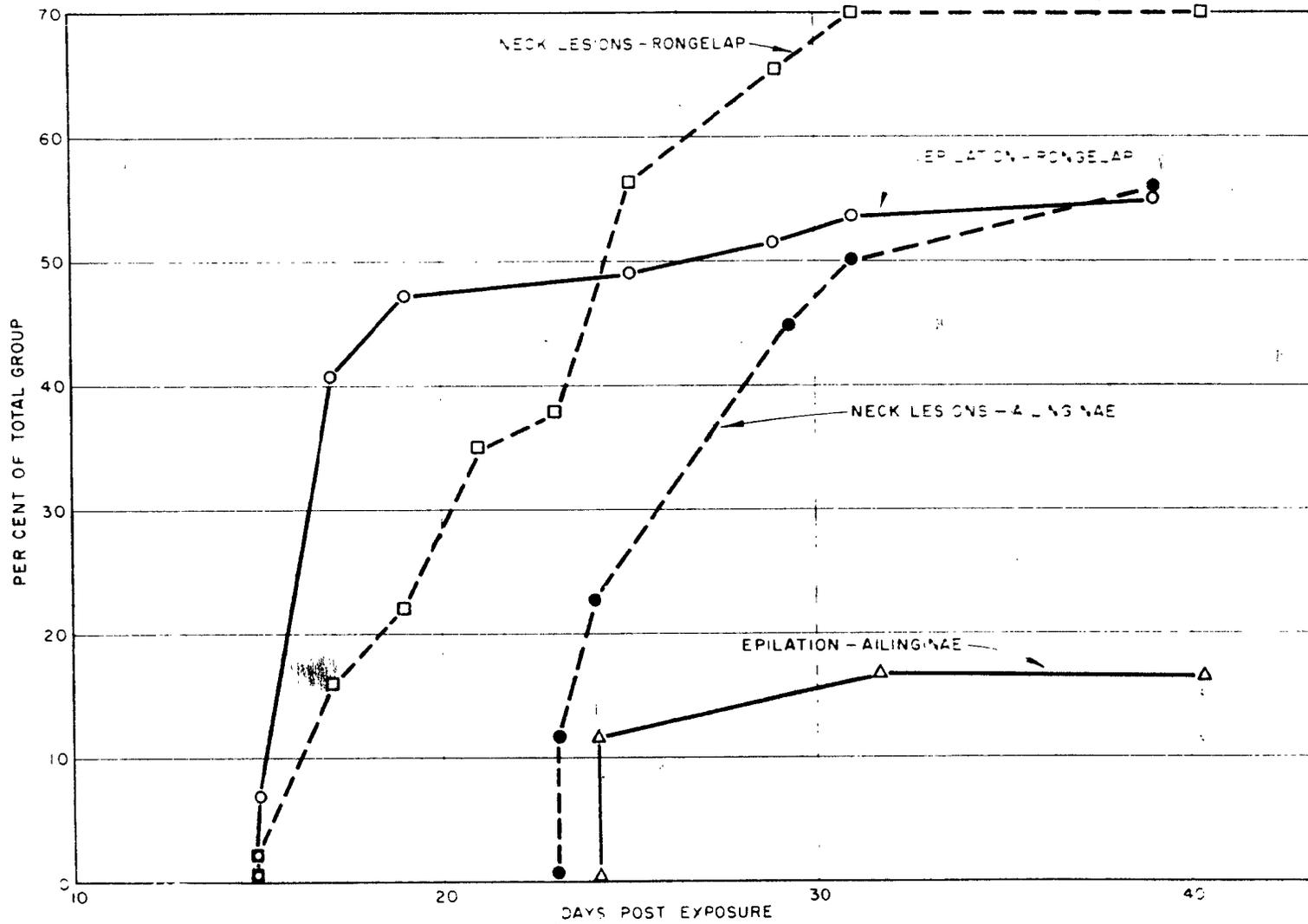


Fig. 3.1 Comparison of the Incidence and Time of Appearance of Epilation and Neck Lesions in the Rongelap and Ailinginae Groups

3.2.4 Histopathology

Seven biopsies were taken of neck lesions and one of an axillary lesion during the third to fourth week after exposure on the Rongelap people. The lesions biopsied at that time were in the hyperpigmented stage with little or no desquamation (shedding) having occurred. Most of these biopsies were taken from individuals who showed lesions of greater than average severity. A second group of biopsies from the Rongelap group (repeated in three individuals) were taken; four at the seventh week post-exposure and five at the eighth-week period. These were taken from the neck and antecubital fossae. All of these lesions had desquamated and the depigmented skin had repigmented to a dusky, gray color with some thickening ("orange-peel" appearance) of the skin. Biopsies were not taken from open lesions or from the feet for fear of infection. All biopsy wounds healed rapidly with no secondary complications.

The microscopic findings are summarized as follows:

Early biopsies - 3rd to 4th week. Epidermis

Transepidermal damage was noted with a few intervening arcades showing less damage (Plates 3.18 and 3.19). The epidermis in the most extensively involved areas showed considerable atrophy with flattening of the rete pegs. In places the epidermis was reduced to a thickness of 2 to 3 cells (Plates 3.20 and 3.21). The cells of the malpighian layer showed pleomorphic nuclei, pyknosis and cytoplasmic halos, giant cells and in a few instances multinucleated cells. Pyknosis of cells of the basal layer was commonly seen. Focal disorganization of the malpighian and basal layers was usually present in the more extensively damaged arcades. Cells laden with pigment were frequently present throughout the epidermis and intercellular pigment was noted in some sections. The stratum granulosum was usually atrophic or even absent. Imperfect keratinization with parakeratosis was visible in all sections. Hyperkeratosis was also seen. The stratum corneum was loosely fibrillated.

The arcades of minimal damage were usually found in areas where sweat ducts approached the epidermis (Plate 3.19). There was an apparent increase in the number of cells and mitotic figures in the neck of the ducts and the adjoining areas where regeneration was underway. In these areas the stratum granulosum was near normal width and pigmentation of the basal cells noted in the more severely damaged areas was lacking.

Changes in the dermis were largely confined to the pars papillaris (Plates 3.18 through 3.21). Mild edema in some cases was noted. Capillary loops were often indistinct and when discernible they frequently were associated with an increased number of pericytes. The endothelial cells showed swelling and were polygonal in shape. Telangiectatic changes (dilated blood vessels) were noted in areas where the overlying epidermis showed greatest damage with lymphocytic infiltration surrounding the telangiectatic spaces. Chromatophores, filled with melanin pigment, were prominent in the superficial dermis. The fine elastic fibrils running into the pars papillaris were often altered or absent.

Little if any damage was seen below the superficial pars reticularis. The hair follicles were narrow (Plate 3.22) and in most instances devoid of shafts in this region. There was some telangiectasis of the capillary spaces bounded by lymphocytes and mononuclear phagocytes in the superficial pars reticularis. Some of the large elastic fibers in this region showed slight swelling in some instances. No damage to fibrocytes or collagen fibers was noted.

Second series, 7th and 8th weeks post-exposure. Epidermis

In general, reparative processes of the epidermis appeared to have been fairly good, except for a few persistent areas of atrophy with narrowing of the epidermis and finger-like downgrowths of the stratum malpighii (Plate 3.24). These occurred in areas of greatest epidermal narrowing and the cells showed rather prominent pigment content. There were many outward epidermal excrescences covered by thickened stratum corneum which was still loosely laminated-- such phenomenon producing a wrinkled appearance which probably accounted for the "orange-peel" like appearance of the skin noted grossly in the areas biopsied (Plate 3.22). In almost all instances the basal layer was intact with little or no disorganization noted. There were a few scattered areas in which occasional epithelial cells with pyknotic nuclei and perinuclear cytoplasmic halos occurred in the stratum granulosum and malpighian layers (Plate 3.23). There were occasional arcades in which the epidermis, particularly the stratum granulosum, appeared to be actually widened. These occurred almost predominantly in relationship



Plate 3.5 Hyperpigmented raised plaques and bullae on dorsum of feet and toes at 28 days. One lesion on left foot shows deeper involvement. Feet were painful at this time.



Plate 3.6 Lesions 10 days later. Bullae have broken, desquamation is essentially complete, and lesions have healed. Feet no longer painful.



Plate 3.7 Lesions 6 days later showing repigmentation except for small scar on dorsum of left foot at site of deepest lesion.

Plates 3.5-3.7 Sequential changes in foot lesions Case 67, age 14, F.

to the contiguous sweat gland ducts at sites where the latter penetrated the epidermis. A narrow zone of parakeratinosis and amorphous debris was still present between the stratum granulosum and the loosely laminated stratum corneum.

Dermis

The capillary loops in the dermal papillae, although present, were not uniformly distinct. Pericytes remained in increased number but fewer lymphocytes were present. Generally, there appeared to be slight telangiectasis of the capillaries in the pars papillaris and the superficial pars reticularis (Plate 3.24). There was some edema of the pars papillaris (Plate 3.22). Scattered pigment laden chromatophores were irregularly distributed in the papillary layer (Plate 3.23). In some cases hair shafts in the superficial pars reticularis were quite narrow, atrophic, and occasionally absent; in others the hair shafts appeared normal. Small hair follicles (Plate 3.22) and secretory sweat ducts in some cases showed mild atrophy.

Biopsies of three pigmented mild lesions were taken from two of the white Rongerik Americans. Only one of the three gave evidence of damage, which was nominal and confined to the epidermis.

3.2.5 Symptomatology and Treatment

On the day of exposure, itching and burning of the skin was prevalent. This subsided and for a period of 10 to 14 days or longer there was neither subjective nor objective evidence of skin injury. Itching and burning reappeared either prior to or in the early pigmentation stage. With the deeper lesions there was also pain. Pain was rather marked with the foot lesions. During the painful period some of the foot lesions were also hot and presented a brawny edema. A common complaint was a tenderness in the great toes medial to the nails. However, visible lesions in this area were infrequent. This symptom usually preceded the appearance of gross lesions elsewhere on the feet. Many of the individuals who developed painful foot lesions were observed walking on their heels for several days. The painfulness of the foot lesions may have resulted from their greater severity, and may have been accentuated by the dependent nature of the foot. Some of the lesions of the neck and axilla were painful when turning the head or raising the arms. The acute reaction and pain subsided after a few days. There were no constitutional symptoms.

The treatment of skin lesions was largely non-specific. Most of the superficial lesions were treated with calamine lotion with one per cent phenol, which in most cases relieved the itching, burning or pain. A few of the painful hyperpigmented lesions not relieved by calamine with phenol were treated with pontocaine ointment, with apparent success. When the epithelium desquamated the itching was relieved by daily washing with soap and water and the application of a water soluble vanishing type ointment which kept the injured skin soft and pliable. Raw areas, which became secondarily infected, were treated by washing with soap and by the application of aureomycin ointment. Bullous lesions of the feet were left intact as long as no symptoms were present. If painful, the bullae were aspirated with sterile techniques to remove the clear straw-colored fluid. A single aspiration was adequate since the bullae did not refill. One foot lesion developed an extensive, raw weeping ulcer. Prophylactic penicillin was given for two days, during which time the lesion developed healthy granulation tissue. Some of the lesions of the skin of the foot remained thickened, less pliable, and painful after desquamation. This was relieved by the use of vaseline or cocoa butter to soften the tissues. One persistent ear lesion did not heal after desquamation. This was treated with warm boric acid compresses and washing with surgical soap to remove the eschar. Granulation tissue formed, and epithelium was slowly growing in from the edges of the ulcer when the initial observation period was terminated 74 days after exposure. Upon resurvey six months after exposure healing was complete, with a depigmented scar remaining as evidence of the previous ulcer.

3.3 EPILATION

The incidence of and time of appearance of epilation in the Rongelap and Ailinginae groups is illustrated in Tables 3.1, 3.2 and Fig. 3.1. Epilation was first observed on the 14th post-

exposure day in the Rongelap group and was confined to the head. The epilation was divided arbitrarily into three degrees of severity. "1+" epilation indicates loss of hair without obvious thinning; "2+" indicates a loss of hair sufficient to cause obvious thin spots; and "3+" indicates an extensive epilation with bald spots. Table 3.1 illustrates that there was a greater degree of epilation in the children (0 to 15 years). Over 90 per cent developed epilation of some degree in the 0 to 15 years group, compared to only 28 per cent in the older age group. The preponderance of scalp lesions in the areas of epilation indicates that radiation from the fallout ma-

Table 3.2 LESIONS IN AILINGINAE AND RONGERIK GROUPS

Type of Lesion	Ailinginae (18 people)		Rongerik (28 people)	
	Per Cent of Total with Lesions	Mean Time of Appearance*	Per Cent of Total with Lesions	Mean Time of Appearance*
Epilation	16.7	27	3.5†	42
Lesions of:				
Scalp & Face	38.9	26	10.7	32
Neck and Shoulders	61.0	27	14.3	30
Back	0.0		7.1	28
Axilla	22.2	24	3.5	23
Antecubital Fossae	11.1	28	25.0	29
Hand & Wrist	5.6	38	3.5	47
Feet	16.7	33	3.5	43
Legs	5.6	44	0.0	
Nail				
Discoloration	77.7	38	17.9	40

(All Negroes)

* Days post-exposure.

† One case claimed slight epilation. Questionable.

terial on the skin is primarily responsible for the epilation. In the Ailinginae group only three cases of mild epilation developed in children (Table 3.2).

Slight regrowth of hair was observed in all individuals nine weeks after exposure. Hair regrowth was complete and normal six months after exposure.

3.4 NAIL PIGMENTATION

An unexpected observation was the discovery of a bluish-brown pigmentation of the fingernails which was first well documented on the 23rd post-exposure day. The discoloration began in the semilunar area of the fingernails (to a lesser extent in the toenails), and tended to spread outward sometimes in streaks. As the discolored area grew outwards the semilunar area usually became clear. In a few cases, detachment of the end of the nail from the nail bed was observed when the pigmentation reached the end of the nail. Plate 3.17 shows pigmented bands in the nails at 77 days. The discoloration of the nails was seen in 89 per cent of the Rongelap and 78 per cent of the Ailinginae group. It appeared to be a radiation response peculiar to the dark-skinned races since it was seen in all of the American Negroes in the Rongerik group and in none of the white men. This lesion was not observed in the Utrik group nor in the control Marshallese. Since the nail pigmentation occurred in individuals without skin lesions, it appeared to be the result of a more penetrating component of radiation than contact radiation which predominantly produced the skin lesions.



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Plates 3.8-3.9 Extensive lesions in boy, age 13, case 26.

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3.5 FACTORS INFLUENCING THE SEVERITY OF SKIN LESIONS

3.5.1 The Characteristics of the Fallout Material

The fallout material was composed mainly of calcium oxide from coral, with adherent fission products. The skin lesions observed were undoubtedly the result primarily of beta radiation from fallout material deposited on the skin, with minimal or no contribution from chemical irritation. The gamma dose to the skin was small compared to the beta dose, and thus was relatively unimportant in producing the lesions.

3.5.2 Energy of Beta Particles

From available data on the fallout material it has been calculated that 50 to 80 per cent of the beta rays during the exposure period were soft with an average energy of about 100 kev. Since 80 microns of tissue produces 50 per cent attenuation of such radiation,¹ the greater portion of energy was dissipated in the epithelium which is roughly 40 to 70 microns in thickness. The remaining 20 to 50 per cent of the beta rays were of higher energy, with an average of approximately 600 kev. The latter would penetrate well into the derma since it takes 800 microns of tissue to produce 50 per cent attenuation.^{1,2} In addition, a wide spectrum of gamma energies irradiated the skin. Approximately 10 per cent of the total gamma spectrum was below 80 kev which would be absorbed largely in the superficial layers of the skin. The remainder of the gamma spectrum is distributed between 100 and 1600 kev with a large proportion between 600 and 800 kev.

3.5.3 Physical Dose to the Skin

There is no practical way to estimate the physical dose to the areas of skin where lesions were found. The entire surface of the body of the Rongelap group received approximately 175 r from gamma irradiation derived from fission products distributed on the ground, trees, and buildings. To this 175 r would have to be added the beta component. In view of the high beta to gamma ratio in fission products, one might expect the total beta surface dose to the skin to be large. The maximal skin doses from the plane field of radiation are estimated in chapter 1. To these doses must be added the contribution of the material deposited on the skin. The latter can not be calculated, or estimated biologically with any degree of accuracy. A rough approximation of dose received at the hair follicles can be made as follows:

The hair follicles must have received a dose comparable to the known minimal epilating dose of about 400 r for 200 kvp X-rays. Since regrowth of hair occurred, the upper limit of dose at the depth of the hair follicle must not have exceeded the permanent epilating dose of around 700 r of 200 kvp X-ray.³

3.5.4 Protective and Aggravating Factors

The individuals who remained indoors or under the trees showed some protection as compared to those who were in the open during the period of the fallout. Those who went swimming or bathed were also protected to varying degrees. Small children who went wading developed fewer foot lesions. Clothing, even a single layer of cotton material, offered almost complete protection, as was demonstrated by the fact that lesions developed almost entirely on the exposed parts of the body.

Since the lesions predominate in areas where perspiration is abundant such as folds of the neck, axillae, and antecubital fossae, it seems likely that the abundant perspiration produced by a hot, humid climate tended to cause the material to concentrate and adhere to these areas. In addition, the coconut oil hair dressing used by the Marshallese acted as an effective collecting agent for the radioactive material. This was proved since the hair was the most highly contaminated part of the body. The concentration of radioactive material on the hair may have been responsible for the large number of scalp lesions, epilation and the large number and severity of neck lesions in women.

There was a delay of two and one-half days before satisfactory decontamination was possible. The presence of radioactive materials on the skin during this period increased the dose to the skin. However, the dose rate fell off rapidly and decontamination would have to have been very prompt in order to be effective.

3.6 CORRELATION WITH HEMATOLOGICAL FINDINGS

Attempts at correlation of the severity and extensiveness of skin lesions with maximum depression of platelet, lymphocyte, and neutrophil counts were made for individuals in the Rongelap group. No positive correlation was found. Thus the contamination of the skin apparently did not significantly contribute to the total-body dose of irradiation.

3.7 DISCUSSION

There has been little previous experience with radiation dermatitis resulting from exposure to fallout material from nuclear detonations, and the general consensus until now has been that the hazard from fallout material was negligible. With the Hiroshima and Nagasaki detonations, fallout material was not a problem since the bombs were exploded high in the air. The flash burns of the Japanese were purely thermal.

From the present experience it is quite evident that following detonation of a large scale device close to the ground, serious exposure of personnel may occur from fallout material, even at considerable distances from the site of detonation. The incident described in this paper is the first example of large numbers of radiation burns produced by exposure to such fallout material.

Knowlton, et al.⁴ described burns of the hands of four individuals who were handling fission product material following an experimental detonation. Also, following the Alamogordo detonation, there were a number of cattle that developed lesions due to deposit of fallout material on their backs.⁵ In addition, there were a number of sheep that developed lesions closely resembling radiation burns following a Nevada detonation. However, Lushbaugh⁶ reported that the histopathological characteristics of these lesions did not conform in all respects to radiation dermatitis. It is of considerable interest to compare the present experience with that accepted in the past as the typical course of radiation burns of the skin.

The gross lesions of the hands described by Knowlton, et al. occurred from an exposure of about one hour, resulting in doses between 3000 and 16,000 rep of beta radiation (maximum energy about 1 Mev) with a small gamma component considered to be insignificant. The lesions were described as developing in four phases: (1) An initial phase which began almost immediately after exposure and consisted of an erythema with tingling and burning of the hands, reaching a peak in 48 hours and subsiding rapidly so that by 3 to 5 days there was a relative absence of signs; (2) A second phase which occurred from about the 3rd to the 6th or 8th day and was characterized by a more severe erythema; (3) The third phase at 8 to 12 days, which was characterized by vesicle and bullae formation. The erythema spread to new areas during the following two weeks, and the active process subsided by 24 to 32 days. The bullae dried up, and desquamation and epithelization took place in less severely damaged areas; (4) The fourth phase or chronic stage was characterized by further breakdown of skin with necrosis in areas which were damaged sufficiently to compromise the blood supply. Atrophy of the epidermis and loss of epithelial structures took place, which necessitated skin grafting in some cases.

Robbins, et al.⁷ reported six cases accidentally exposed to scattered cathode rays (beta) from a 1200 kv primary beam with exposure time of about 2 minutes and a rough estimation of dose to the skin of between 1000 and 2000 rep. The lesions described were similar to those reported by Knowlton, et al.⁴ with a primary erythema developing within 36 hours; secondary erythema with vesiculation and bullae formation appearing about 12 to 14 days later; and, in the more severely affected, a tertiary phase characterized by further breakdown of the skin. In comparison with severe roentgen ray reactions these investigators stress the unique periodicity of cathode ray burns, relative absence of deep damage to the skin, less pain, rapidity



Plates 3.10 3.11 Whole body view of lesions in same case as plates 3.8 3.9.

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of healing, and absence of pigmentation. These points would apply to the Marshallese except for the multiphasic reactions and pigmentation. Crawford⁸ reports a case of cathode ray burns of the hands which were similar to those described by Robbins, et al.⁷

Experimental beta radiation burns in human beings have been reported by Low-Beer⁹ and Wirth and Raper.¹⁰ Both investigators used P³² discs applied to the flexor surfaces of the arms, forearms, or thighs for varying lengths of time. Low-Beer reported "monophasic" skin reactions. He found that a calculated dose of 143 rep to the first millimeter of skin, ignoring self-absorption, produced a threshold erythema. Dry, scaly, desquamation was produced by 7200 rep in the first millimeter and bullous, wet desquamation was produced by 17,000 rep to the first millimeter. Erythema developed in 3 to 4 days, followed later by pigmentation and desquamation with higher doses. Recovery was observed with doses of 17,000 rep. The lesions later showed depigmented centers with hyperpigmented edges (also seen in the present lesions).

Wirth and Raper¹⁰ produced primary erythema within 6 hours after exposure to a dose of 635 to 1180 rep of P³² radiation. Minute vesicles with dry, spotty desquamation were noted with 1180 rep at about the 5th to 6th weeks post-exposure.

Following the detonation on 1 March 1954, 23 Japanese fishermen were contaminated with fallout material. Apparently they were exposed to roughly the same total-body dose of radiation as were the Rongelap group. The skin lesions which developed are described by Morton, et al.^{11,12} Lesions developed which were similar in most respects to those seen in the Marshallese people, and were characterized by pigmentation, desquamation with depigmentation, spotty epilation of the head and ulcerations developing particularly on the scalp, ears, neck, and hands (the latter probably from handling contaminated fishing lines). Erythema and vesicle formation, as well as inflammation of the eyes were more prominent than in the Marshallese. Pigmentation apparently was not as prominent in the Japanese. The lesions appeared earlier than in the Marshallese (about 7 to 8 days post-exposure). As in the Marshallese the lesions occurred mainly on exposed parts of the body not protected by clothing.

In addition to the Marshallese and Japanese, several Navy men on ships in the test area developed a few small pigmented lesions of "belt-line" distribution, apparently due to fallout material.

The lesions reported in this paper when compared to radiation lesions described in the past presented certain unique features which merit further discussion.

The early symptoms of itching and burning of the skin and eyes were probably due mainly to skin irradiation from the fallout material. However, the chemical nature of this material may have contributed to the irritation. It has been noted¹³ that irritating chemicals applied during or shortly after irradiation enhance the effects of radiation.

The lack of prominence of an erythema was notable, particularly in view of the severity of some of the lesions that developed. Wilhelmy¹⁴ states that erythema only occurs when the dose reaching the papillary layer exceeds a certain level. Perhaps the dose to the dermis was insufficient to evoke the response. On the other hand, the darkness of the skin and the development of hyperpigmentation may have masked an erythema. Microscopically, a superficial hyperemia was not a notable finding.

Wirth and Raper¹⁰ point out that they were impressed in their studies on P³² radiation of the human skin with the difficulty of distinguishing between true erythema and tanning, particularly in the skin of brunette individuals. It was unfortunate that color filters were not available to aid in distinguishing an erythema as suggested by Harris, et al.¹⁵

In general the latent period before development of obvious signs and symptoms for radiation injury to the skin is inversely proportional to the dose of radiation.^{16,17} In the present series of cases the relatively long latent period is suggestive of a low dose of radiation. However, the wide spectrum of beta energies and particulate distribution of radioactive material drastically altered the depth dose, as compared with that in previous experience; hence strict comparisons cannot be made. The later development of lesions in the Ailinginae and Rongerik groups as contrasted with the Rongelap people is in keeping with the relative severity of lesions noted.

A unique feature of the present cases was the appreciable differences in the latent periods observed for lesions on various parts of the body. These differences cannot be explained entirely on the basis of severity, since the severe foot lesions developed after most other lesions. However, the severity might have been in part due to the dependent position of the foot rather than greater radiation injury. Lesions on flexor surfaces in general preceded lesions on extensor surfaces. The present data suggest that the latent period and radiation sensitivity of various skin areas may differ. Previous work has shown that flexor surfaces with thinner epidermis are in general more sensitive than extensor surfaces with thicker epithelium.¹³

The destructive and atrophic changes of the epidermis, disturbances in keratinization, and atrophy of hair follicles are characteristic of histopathologic radiation injury of the skin.^{9,13,18-21} Severe injury to the dermis and blood vessels was not observed. The minimal dermal injury with severe epidermal injury is in keeping with the low energy beta component present and the marked decrease in depth dose over a distance of a few microns from the surface.

Hyperpigmentation of injured areas was a consistent finding in the Marshallese, the Japanese, and the American Negroes. Pigmented lesions were also observed to a lesser extent in the white Americans. Pigmentation of this nature has not been described as a constant characteristic of radiation damage to the skin.

There is no satisfactory explanation for the darker dusky-gray color that appeared in some of the skin lesions as healing progressed. The color changes may be due to alterations in local pigment production, vascular changes, or a thinning of the epidermis, rendering it more translucent with resultant darker appearance of the pigment layer. Later biopsies may explain this phenomenon.

There are features of the lesions described that appear unique, e.g., the absence of visible multiphasic responses, the presence of early hyperpigmentation, the long latent periods, and the severe epidermal injury with minimal dermal injury. It is possible that differences may in part be on a racial basis.* In addition, the marked difference in histologic response of the epidermis and dermis in the present series is in marked contrast to the usual radiation response of the skin produced by high energy X- or beta-rays.

In Table 3.3 are listed the approximate minimal surface skin doses required to produce recognizable epidermal injury in animals. It is apparent from the table that beta ray energy is of considerable importance in determining the degree of injury. A number of assumptions, including knowledge of the beta ray spectrum from the fallout, would have to be made if these data were to be used to estimate biologically the beta dose received by the Marshallese. The difference in dose between that required to produce threshold skin damage and that for permanent damage in pigs is 500 to 1000 rep.³⁰

It is impossible to estimate the probability of development of radiation cancer at the site of the healed lesions. The absence of scarring, telangiectasis, and extensive chronic vascular lesions tends to improve the prognosis since the foregoing are usually observed to precede the development of radiation cancer.

A favorable prognosis is also suggested by the following evidence: an analysis of 1100 individuals exposed to low voltage X-ray for dermatological conditions revealed no evidence of cancer induction 5 to 23 years after treatment. MacKee¹³ states that epitheliomata rarely develop after a single dose of radiation to the skin. Lastly, the incidence of skin cancer in Negroes is one sixth to one ninth the incidence in Caucasians.³³

If neoplasia can develop purely as the result of epidermal irradiation, the incidence of late cancers may be enhanced since the dose of radiation and the visible gross and microscopic injury to the epidermis greatly exceeded that to the dermis. Since many children and young adults were involved, the life expectancy of a large number of the exposed people will exceed the long induction period for development of radiation cancer observed in radiologists. Long

* Reported clinical experience with radiation skin lesions is based predominantly on the response of white-skinned people, whereas the type of lesions described herein, with one clear cut exception, were observed in Japanese, Marshallese (negroid), and American Negroes. The exception was a dark brunette individual.



Plate 3.12 Desquamation of back of scalp at 28 days. Epilation occurred earlier in desquamated area (See Plate 3.14). Note persistent ulceration of left ear.

Plates 3.12-3.13 Scalp lesions in man, age 41, case 79. Head has been shaved



Plate 3.13 Eighteen days later. Scalp lesions have healed with repigmentation of skin. No hair growth evident in epilated areas. Ear ulceration persists with little improvement

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exposure to tropical sunlight, potentially carcinogenic in itself, may increase the probability of neoplastic change. The influence of the sub-lethal whole body exposure received by these people on skin cancer induction is not known.

The transverse band of pigmentation that was observed in the fingernails has been previously observed by Sutton³⁴ in a negress who received 150 r of soft X-ray to the hands. The

Table 3.3 SURFACE DOSES IN REP REQUIRED TO PRODUCE RECOGNIZABLE EPIDERMAL INJURY

Investigator	Animal	Isotope	Average Energy (Mev)	Surface Dose (rep)
Henshaw, et al ²⁷	Rats	P ³²	0.5	1,500 - 4,000
Raper & Barnes ²⁸	Rats	P ³²	0.5	4,000
Raper & Barnes ^{2b}	Mice	P ³²	0.5	1,500
Raper & Barnes ^{2b}	Rabbits	P ³²	0.5	5,000
Lushbaugh ⁶	Sheep	Sr ⁹⁰	0.3	2,500 - 5,000
Moritz and Henriques ³⁰	Pigs	S ³⁵	0.05	20,000 - 30,000
Moritz and Henriques ³⁰	Pigs	Co ⁶⁰	0.1	4,000 - 5,000
Moritz and Henriques ³⁰	Pigs	Cs ¹³⁷	0.2	2,000 - 3,000
Moritz and Henriques ³⁰	Pigs	Sr ⁹⁰	0.3	1,500 - 2,000
Moritz and Henriques ³⁰	Pigs	Y ⁹¹	0.5	1,500 - 2,000
Moritz and Henriques ³⁰	Pigs	Y ⁹⁰	0.7	1,500 - 2,000

nature of the pigment is not known. Since it occurred in all exposed American Negroes, many of the Marshallese, and none of the American whites, it is a radiation response peculiar to negroid races. The pigmentation was apparently produced by as little as 75 r of gamma radiation since the American Negroes developed the phenomenon in the absence of significant contamination of the hands.

3.8 CONCLUSIONS

As a result of this accident the following conclusions can be drawn with respect to beta damage to the skin.

- a. Serious skin contamination of personnel from fallout may occur many miles from the detonation of a nuclear device. Resultant radiation damage to the skin may be the major radiation effect under conditions where early evacuation from the field of radiation reduces the whole body exposure.
- b. Fairly extensive skin lesions resulting from fallout beta radiation apparently produce little or no systemic or hematological effects.
- c. Decontamination of the skin must be prompt to be effective because of the initial high beta dose rate.
- d. A latent period of a few days to three to four weeks may elapse before signs and symptoms of skin damage are evident.
- e. Clothing and/or any type of shelter gives almost complete protection to the skin.

NOTE

For purposes of documentation the following color plates (Plates 3.1 through 3.24) numbered with letters *a*, *b*, *c*, *c1*, *d*, *e*, *f*, and *g* are considered to be pages 47-57, 59, and 61-62.

REF ID: A66110

COLOR PLATES

Plates 3.18 through 3.21 show lesions at 22 days post-exposure (Plates 3.18 through 3.20, Case 26; Plate 3.21, Case 63).

Plate 3.18 (X100, H&E) Epidermis: Extensive transepidermal damage with less involved zones on either side. Loose lamination of stratum corneum, absence of stratum granulosum. Parakeratinization with exfoliation of pigment containing cells. Disorganization of the malpighian layer. Dermis: Mild edema of pars papillaris with indistinct capillary loops. Moderately pronounced perivascular cellular infiltrate (lymphocytes and mononuclear phagocytes), in superficial corium with mild telangiectasis.

Plate 3.19 (X100, H&E) Epidermis: Arcades of minimal damage occur in relation to excretory ducts of sweat glands. Stratum granulosum of good width and shows scant alteration. Underlying stratum malpighii shows marked decrease in pigment. In the deeper portion of the overlying, loosely laminated stratum corneum moderate amounts of pigment, however, are present. One narrow arcade of more severe transepidermal damage shows alteration of the stratum granulosum with intercellular edema, pyknosis, swollen nuclei, and pigment scattered throughout. The latter is especially dense in the contiguous parakeratotic material. Dermis: A moderate cellular infiltrate, chiefly perivascular, is most pronounced in the superficial pars reticularis where there is a mild telangiectasis.

Plate 3.20 (X400, H&E) (Case #26) Transepidermal damage with disorganization of the malpighian layer. Stratum granulosum absent. Malpighian and basal layer only two to three cells thick with exfoliation of pigment outward toward parakeratinized zone adjacent to stratum corneum. Pigment laden chromatophores and histiocytes in pars papillaris of corium. Latter is edematous and infiltrated by moderate numbers of lymphocytes, mononuclear phagocytes and scattered pigmented leukocytes. Capillary loops indistinct.

Plate 3.21 (X100, H&E) (Case #63) Transepidermal damage with disorganization of the malpighian layer. Slight parakeratosis. Migration or exfoliation outward of pigment. Loose lamination of stratum corneum. Pigment laden chromatophores and histiocytes in superficial pars papillaris of corium. Marked cellular infiltration of pars papillaris. Slight telangiectasis of superficial pars reticularis.

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Plate 3.18



Plate 3.19



Plate 3.20



Plate 3.21

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COLOR PLATES

Plates 3.22 and 3.23 show lesions at 53 days post-exposure.

Plate 3.22 (X100, H&E) (Case #75) Loose lamination of stratum corneum with outward papillary projections and resultant "rugose" appearance. Stratum granulosum of good width. Basal and malpighian layers distinct with pigment present. Slight edema of corium with mild telangiectasis and slight increase in perivascular lymphocytes and pericytes. Small, atrophic hair follicle adjacent to sebaceous gland-- in mid pars reticularis.

Plate 3.23 (X400, H&E) (Case #75) Same as 3.22. Occasional perinuclear cytoplasmic halos in mid stratum granulosum. Loosely laminated stratum corneum. Pigment laden chromatophores in superficial corium along with occasional lymphocytes and mononuclear phagocytes.

Plate 3.24 shows lesions (second biopsy) at 46 days post-exposure.

Plate 3.24 (X100, H&E) (Case #39) Narrow rugose epidermis with papillary extensions downward of stratum malpighii. Latter are heavily laden with melanotic pigment. Slight telangiectasis of pars papillaris and pars reticularis of dermis. Occasional pigment laden chromatophores in superficial dermis.

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Plate 3.22



Plate 3.23



Plate 3.24

CHAPTER 4

HEMATOLOGY

4.1 INTRODUCTION

Since it is generally agreed that a depression in the formed elements of the peripheral blood is the most useful practical clinical index of the degree of exposure to ionizing radiation, a systematic study of the leukocytes and platelets was relied upon as a major aid in evaluating the clinical status, severity of the radiation injury, and prognosis of the exposed individuals. Animal experimentation had previously shown that the rate of development and magnitude of the depression were equally important in evaluating the severity of radiation injury. Since there had been no previous exposure of human beings to significant amounts of fallout radiation, no hematological data known to be strictly applicable* were available for use as a guide in the evaluation of the exposed Americans and Marshallese. Accordingly emphasis was placed on systematic serial studies, utilizing a few highly standardized hematologic determinations to insure that individual and group trends would have maximum validity. Since it was known that the Utrik group had received a very small dose of radiation compared to the other exposure groups, less extensive determinations were carried out on these people.

4.2 GENERAL METHODS

Hematological examinations included total leukocyte, neutrophile, lymphocyte and platelet counts, and hematocrit determinations. Whenever possible an entire exposure group was studied in a single day. Occasionally two days were required to complete the larger groups.

Capillary blood was used, usually obtained from the finger but occasionally from the heel or ear. Two pipettes each for total leukocyte and platelet counts were filled. From each pipette a single hemocytometer chamber was filled. All pipettes were rotated for 10 minutes, and the cells were allowed to settle for 10 minutes in the hemocytometer chamber before counting. A 3 per cent acetic acid diluting fluid was used for total leukocyte counts. The blood was diluted with 1 per cent ammonium oxalate for platelet counts and counted in flat bottom hemocytometers using a dark phase contrast microscope.³⁵ Two blood smears were made with each examination, using a beveled end glass slide for spreading. One blood smear was fixed in methyl alcohol. The other was stained by Wright's method, from which a 100 cell differential count was made. Hematocrits were performed using heparinized capillary tubes. One end of the capillary tube was heat sealed and the tube was centrifuged in an ALOE centrifuge at 12,500 rpm for 5 minutes.

*A large literature on the hematologic effects of radiation exists; however, these data were not relied upon for direct comparison and evaluation of the exposed individuals for reasons that will be indicated later in the discussion.

Every effort was made to maintain uniform procedures in every phase of the laboratory work. The number of personnel changes for a given procedure was held to a minimum; personnel drawing blood from a single puncture were sufficient in number to allow all samples to be taken in rapid succession. All time intervals were rigidly controlled.

4.3 METHODS OF TREATING DATA, CONTROL GROUPS

Pre-exposure blood counts were not available on the exposed Marshallese or Americans; hence the individuals could not be used as their own controls. In order to estimate the severity of the hematologic response it was necessary to establish control groups as comparable as possible in respect to age, race, sex, background, and habits. A Marshallese control group living on Majuro, comparable with respect to age and sex to the Rongelap people, was used as

Table 4.1 HEMATOLOGICAL RESULTS, CONTROL GROUPS

Control Group	Age (yr)	No. in Gp.	WBC ($\times 10^3$)	Neutrophils ($\times 10^3$)	Lymphocytes ($\times 10^3$)	Platelets ($\times 10^4$)	Hematocrit* (Per Cent)		
							M	F	Comb
Majuro	0-5	22	13.2	4.8	7.4	38.8	38.5 (10)	37.3 (12)	37.8 (22)
	6-15	14	10.6	4.8	5.1	38.6	41.3 (6)	40.6 (8)	40.9 (14)
	15-50	63	9.4	4.8	4.0	30.9	46.8 (29)	40.4 (34)	43.3 (63)
	>50	14	9.6	5.0	3.7	30.7	43.6 (10)	41.8 (4)	43.0 (14)
	>5	91	9.7	4.8	4.1	33.6	45.2	40.6	
	<15	37				38.5			
	>15	76				30.8			
Kwaj- Americans	>18	28	7.8	4.1	3.1	23.8		44.9	

* Numbers in parentheses indicate number of individuals in the group.

the Marshallese control group. For comparison with the exposed Americans, blood counts were made on approximately 85 American males, on duty at Kwajalein. All who had not been on duty in the tropics for more than two months were excluded since the exposed Americans on Rongerik had been in the area for about two months before exposure. In addition several who were recently associated with radioactive materials were excluded. The resulting smaller group of 67 was used as the Kwaj-American control group.

Preparatory to analyzing the hematological results on the exposed Marshallese, data from the control Majuro Group were examined to determine if there was an age or sex dependence in the hematologic observations (Table 4.1). Although the neutrophile count was independent of age, the lymphocyte counts were significantly higher in children below the age of 5. Similarly the platelet count was higher in the younger age groups; however, a relative depression appeared to occur at about age 15.

The total leukocyte, neutrophile and lymphocyte counts were independent of sex. The hematocrit of females was lower than that of males, particularly in the child bearing age group. The age and sex dependency of these endpoints is comparable to that in published data.^{36,37}

To obtain valid comparisons within and among the various exposure groups, they were stratified in accordance with age or sex dependency noted for the control groups. Although each individual in all groups was studied hematologically, those Marshallese with serious long-standing diseases were omitted from the analysis. A total of two from the Rongelap and two from the Majuro groups were omitted on this basis.

In the following descriptions and comparisons of the data, findings in the exposed groups are usually expressed in terms of per cent of the appropriate control group. It should be noted, however, that in observational studies of this kind, unknown factors could possibly account for part of the differences noted between the control and exposure groups even though all possible measures were taken to select a comparable control group. In addition, it was not possible to obtain more than a single blood sample on each control individual. For these reasons, statistical tests of significance were applied mainly to time changes within an exposure group, and not to differences between control and exposure groups. For the purpose of detecting significant changes in the hematological pattern, nonparametric tests (i.e., statistical tests for which it is not necessary to specify the functional distribution of the variate under study) were used.³⁸⁻⁴³ The advantages of nonparametric methods have been summarized.⁴⁴

4.4 HEMATOLOGICAL FINDINGS, GENERAL

Total leukocyte, neutrophile, lymphocyte, monocyte, platelet and eosinophile counts for the several exposure groups are given by day and by age in Tables 4.2 to 4.5. The mean total white count, neutrophile, lymphocyte and platelet counts at the times of peak depression (time over which counts were consistently the lowest) are shown in Tables 4.6 and 4.7 (pp 78 and 79) for each individual in the Rongelap and Ailinginae groups, respectively. Hematological findings as a function of time and age are shown also in Figs. 4.1 to 4.8. The cumulative distribution curves for the various exposure groups, using the average of counts obtained over the period of maximum depression (days 39 to 51 for leukocytes; days 26 to 30 for platelets) are shown in Figs. 4.9 to 4.11.* In the figures emphasis is placed on the individual blood elements rather than on the total leukocyte count, since the component elements have distinct and different time trends after irradiation.

Table 4.2 RONGELAP GROUP MEAN BLOOD COUNTS BY DAY AND BY AGE

P.E. Day	W.B.C. ($\times 10^3$)		Neutrophiles ($\times 10^3$)		Lymphocytes ($\times 10^3$)		Platelets ($\times 10^6$)		Monocytes ($\times 10^3$)		Eosinophiles ($\times 10^3$)	
	<5	>5	<5	>5	<5	>5	<15	>15	<5	>5	<5	>5
	3	9.0	8.2	6.4	4.7	1.8	2.2			0.8	0.3	0.1
7	4.9	6.2										
10	6.6	7.1	3.5	4.5	2.6	2.1	27.5	22.1	2.9	1.7	1.6	1.6
12	5.9	6.3	3.5	3.9	2.1	1.7			4.2	5.4	1.9	1.9
15	5.9	6.5	3.2	4.1	2.4	1.9	26.1	19.8	3.0	2.3	1.1	1.3
18	6.7	7.2	3.4	4.7	2.4	2.1	23.0	19.6	2.7	1.7	3.5	1.6
22	7.0	7.4	4.3	5.0	2.6	2.1	16.2	14.7	1.9	2.0	2.3	1.8
26	5.7	6.1	3.0	3.9	2.3	1.8	12.6	10.9	1.9	1.6	1.8	1.3
30	7.6	7.8	4.0	5.3	3.2	2.1	13.3	11.5	1.5	0.9	3.4	2.2
33	6.5	6.2	3.1	3.8	3.2	2.0	17.9	14.6	1.7	1.6	2.6	2.2
39	5.7	5.5	3.0	3.3	2.6	2.0	23.6	21.5	0.9	0.9	0.5	1.0
43	5.2	5.2	2.0	2.6	2.9	2.3	25.0	21.8	1.1	1.1	1.4	0.8
47	5.9	5.8	2.6	3.3	3.1	2.4	25.8	20.6	1.0	1.0	1.1	0.5
51	6.7	5.6	2.6	3.5	3.4	2.1	24.2	18.2	2.5	1.6	0.8	0.7
56	7.0	6.0	3.5	3.5	3.7	2.4			1.7	1.2		
63	7.7	6.0	3.9	3.6	3.7	2.3	21.8	19.1	0.5	0.9	0.3	0.6
70	7.6	6.5	3.8	4.0	3.3	2.2					3.4	1.9
74							28.1	21.1				
Majuro Controls	13.2	9.7	4.8	4.8	7.4	4.1	38.5	30.8	2.0	2.0	9.5	4.7

*In the Utirik group the cumulative distribution curve for platelet counts only is presented since hematological determinations in this group were not made during the 39 to 51 day period, used for leukocyte comparisons among the other groups.

Table 4.3 ALLINGINAE GROUP MEAN BLOOD COUNT BY DAY AND BY AGE

P.E. Day	W.B.C. ($\times 10^3$)		Neutrophiles ($\times 10^3$)		Lymphocytes ($\times 10^3$)		Platelets ($\times 10^4$)		Monocytes ($\times 10^3$)		Eosinophiles ($\times 10^3$)	
	<5	>5	<5	>5	<5	>5	<15	>15	<5	>5	<5	>5
3	6.0	7.0	3.0	5.0	2.8	2.2			0.8	1.6	0.5	0.4
7	5.5	6.8										
10	6.3	7.3	4.2	4.2	1.9	2.2	22.2	21.1	3.8	2.1	2.6	1.6
12	6.3	7.6	1.8	4.7	3.1	2.2			3.4	5.8	4.4	2.6
15	7.1	7.0	2.3	4.5	4.2	2.2	25.7	23.0	3.7	2.6	2.3	1.4
18	6.8	7.8	2.9	5.0	3.5	2.4	28.8	22.0	2.3	1.5	3.2	2.3
22	8.9	8.7	5.3	5.4	2.7	2.9	24.7	19.6	1.5	2.4	5.8	2.4
26	8.4	7.0	4.8	4.4	3.2	2.2	21.4	14.3	2.3	2.4	0.6	1.6
30	9.6	8.6	5.3	6.2	3.7	2.0	21.0	14.7	1.9	1.9	4.1	2.0
33	7.7	7.8	3.3	5.2	3.5	2.2	26.8	18.0	2.8	2.2	6.0	1.9
39	7.5	6.2	2.9	4.2	4.7	1.9	31.0	22.2	1.1	1.7	2.7	1.6
43	6.9	6.5	2.7	3.6	3.9	2.7	28.5	22.1	0.6	1.4	2.8	0.6
47	7.3	6.7	3.5	3.8	3.4	2.7	25.8	23.9	2.2	1.9	1.5	0.7
51	8.4	6.3	3.8	3.6	4.0	2.2	28.5	21.6	2.7	2.8	2.2	1.0
54	4.6	6.3	2.8	3.5	3.2	2.5	29.8	21.2	1.5	1.9	1.8	0.8
Majuro Controls	13.2	9.7	4.8	4.8	7.4	4.1	38.5	30.8	2.0	2.0	9.5	4.7

Table 4.4 UTIRIK GROUP MEAN BLOOD COUNT BY DAY AND BY AGE

P.E. Day	W.B.C. ($\times 10^3$)		Neutrophiles ($\times 10^3$)		Lymphocytes ($\times 10^3$)		Platelets ($\times 10^4$)		Monocytes ($\times 10^3$)		Eosinophiles ($\times 10^3$)	
	<5	>5	<5	>5	<5	>5	<15	>15	<5	>5	<5	>5
4	9.4	8.2	4.7	4.2	4.9	3.2			0.6	0.2	2.0	1.2
14	10.0	8.6	4.1	3.2	5.1	2.9			4.9	4.2	3.6	2.7
19							37.7	31.5				
29	10.1	9.7	4.9	5.8	4.8	3.2	33.2	28.6	2.2	1.7	3.1	2.0
Majuro Controls	13.2	9.7	4.8	4.8	7.4	4.1	38.5	30.8	2.0	2.0	9.5	4.7

Table 4.5 RONGERIK GROUP MEAN BLOOD COUNT BY DAY

P.E. Day	W.B.C. ($\times 10^3$)	Neutrophiles ($\times 10^3$)	Lymphocytes ($\times 10^3$)	Platelets ($\times 10^4$)	Monocytes ($\times 10^2$)	Eosinophiles ($\times 10^2$)
1	9.6	6.1	3.3		0.1	1.6
8	6.6	4.3	2.1		1.9	0.5
9	6.2	4.0	2.0		2.0	0.4
10	6.3	3.8	2.2		2.2	0.3
11	6.2	3.9	2.1		1.8	0.5
12	6.0	3.7	2.1		1.5	0.7
13	6.1	3.7	2.1		1.7	1.2
15	6.1	3.8	2.0		1.7	1.3
16	8.1	4.7	2.9	22.0	2.8	2.5
19	7.9	4.8	2.7	22.2	2.4	2.1
23	6.7	4.2	2.1	17.9	1.6	1.4
28	7.2	4.1	2.5	14.4	2.0	2.1
33	6.7	4.1	2.2	16.1	1.8	2.2
39	6.8	3.8	2.7	20.1	1.4	1.5
43	7.6	4.4	2.9	21.8	1.4	2.0
47	7.8	4.6	3.1	20.2	2.7	1.5
51	5.7	3.2	2.2	18.8	2.4	1.7
Kwajalein Controls	7.8	4.1	3.1	23.8	2.6	2.7

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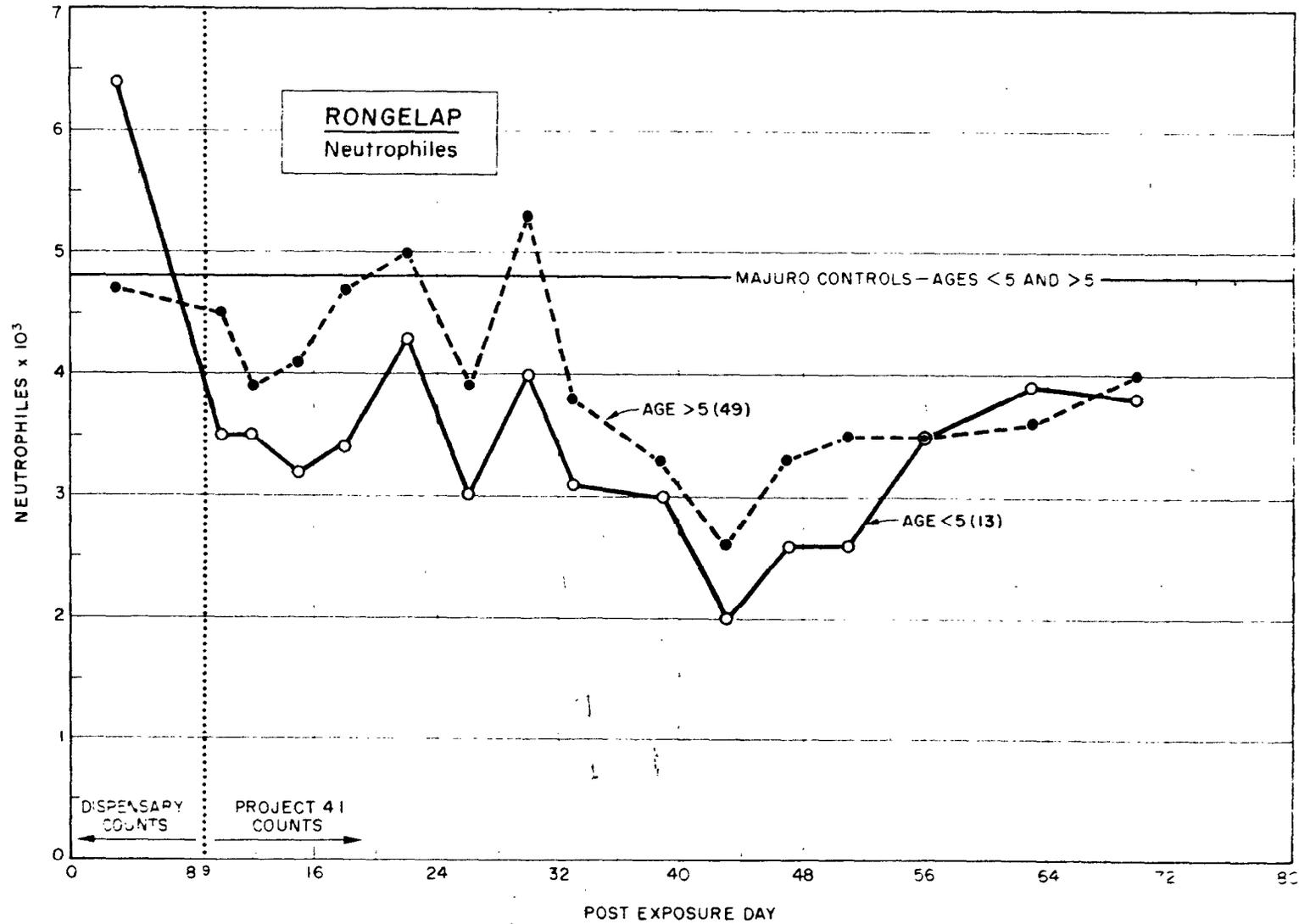


Fig. 4.1 Serial Changes in Neutrophile Counts of Rongelap Group for Those Less Than 5 Years and Greater Than 5 Years of Age

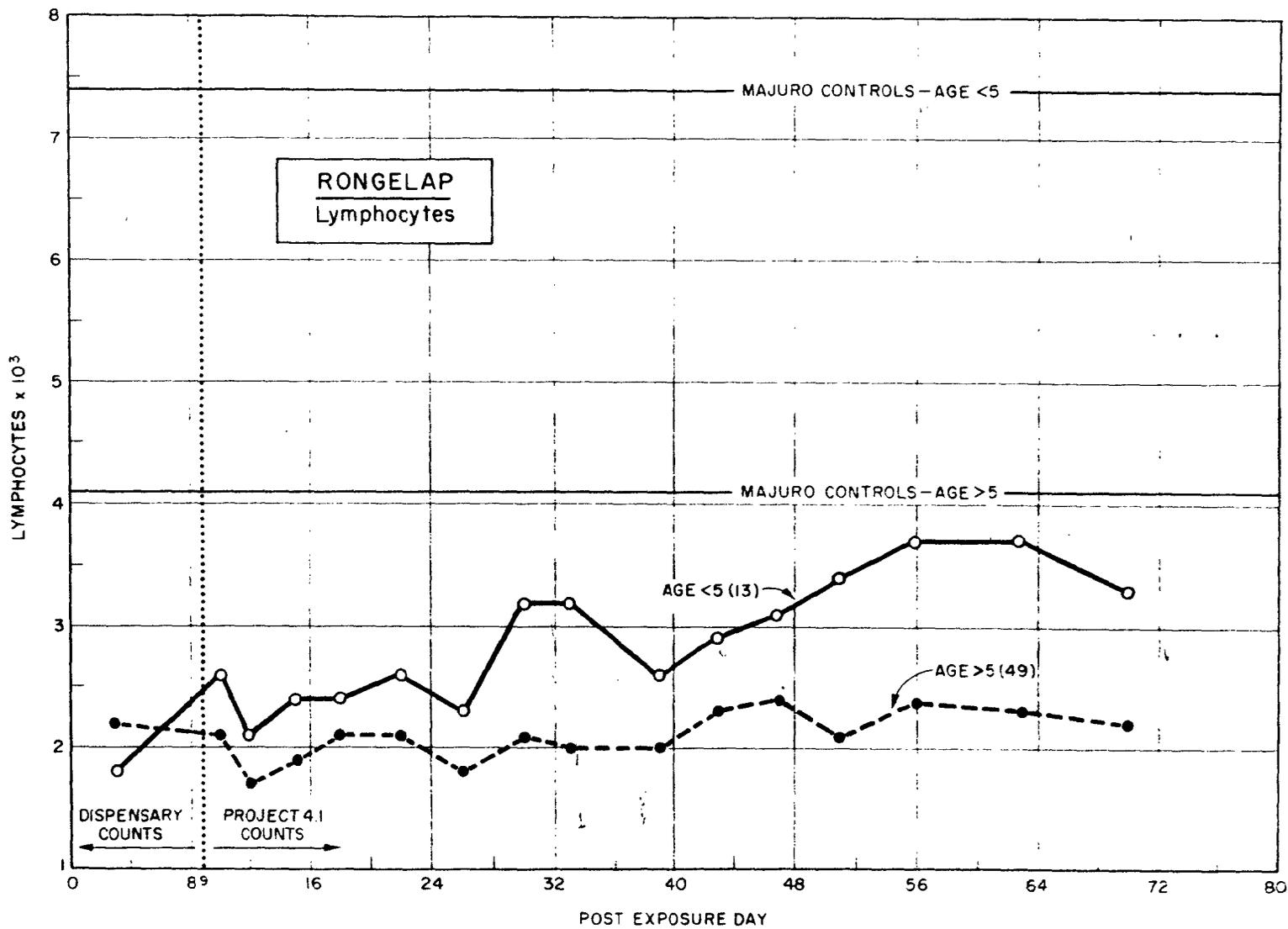


Fig. 4.2 Serial Changes in Lymphocyte Count of Rongelap Group for Those less Than 5 Years and Greater Than 5 Years of Age

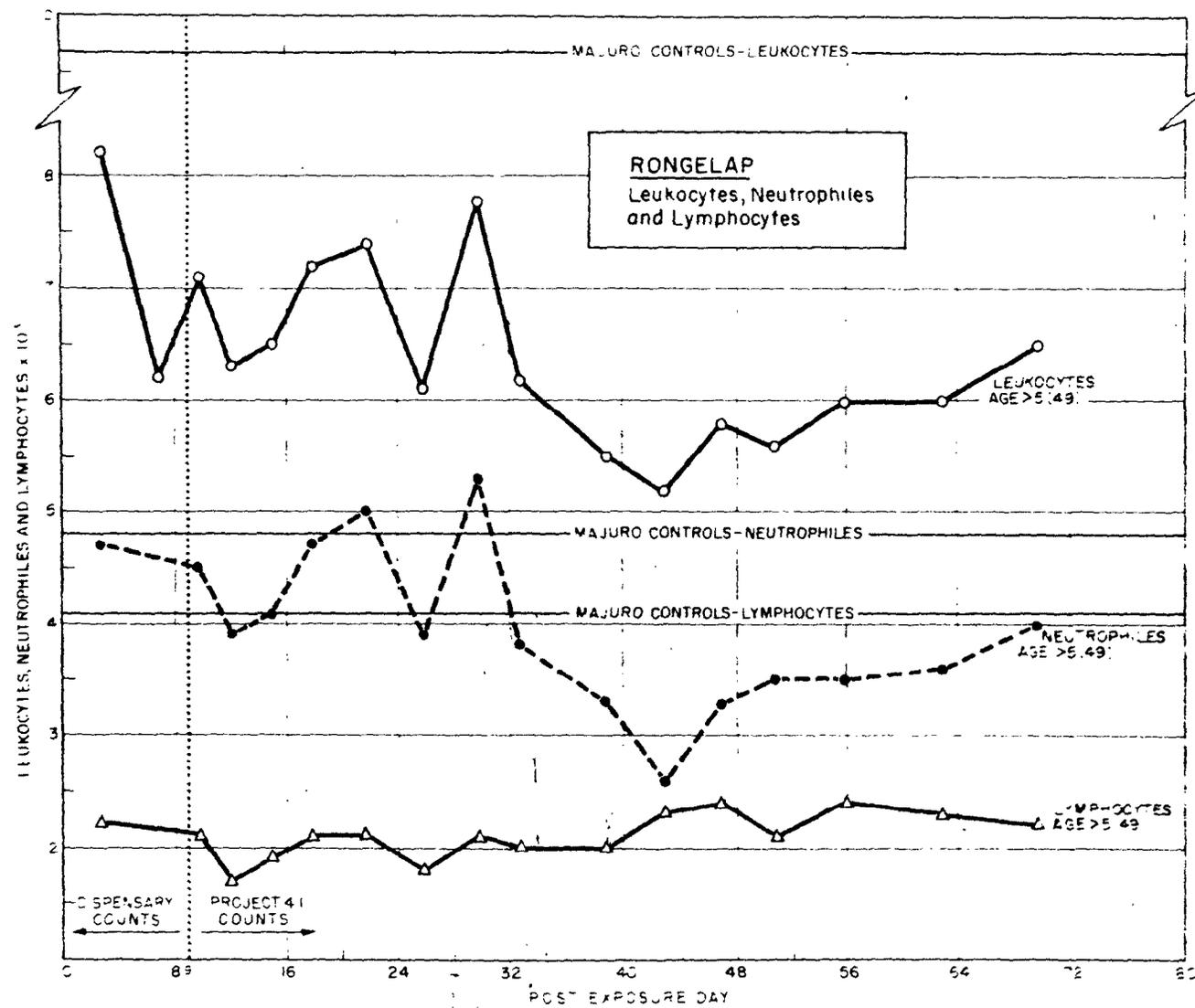


Fig. 4.3 Comparative Serial Changes in the Total Leukocyte, Neutrophile, and Lymphocyte Counts in Those Greater Than 5 Years Old, Rongelap Group

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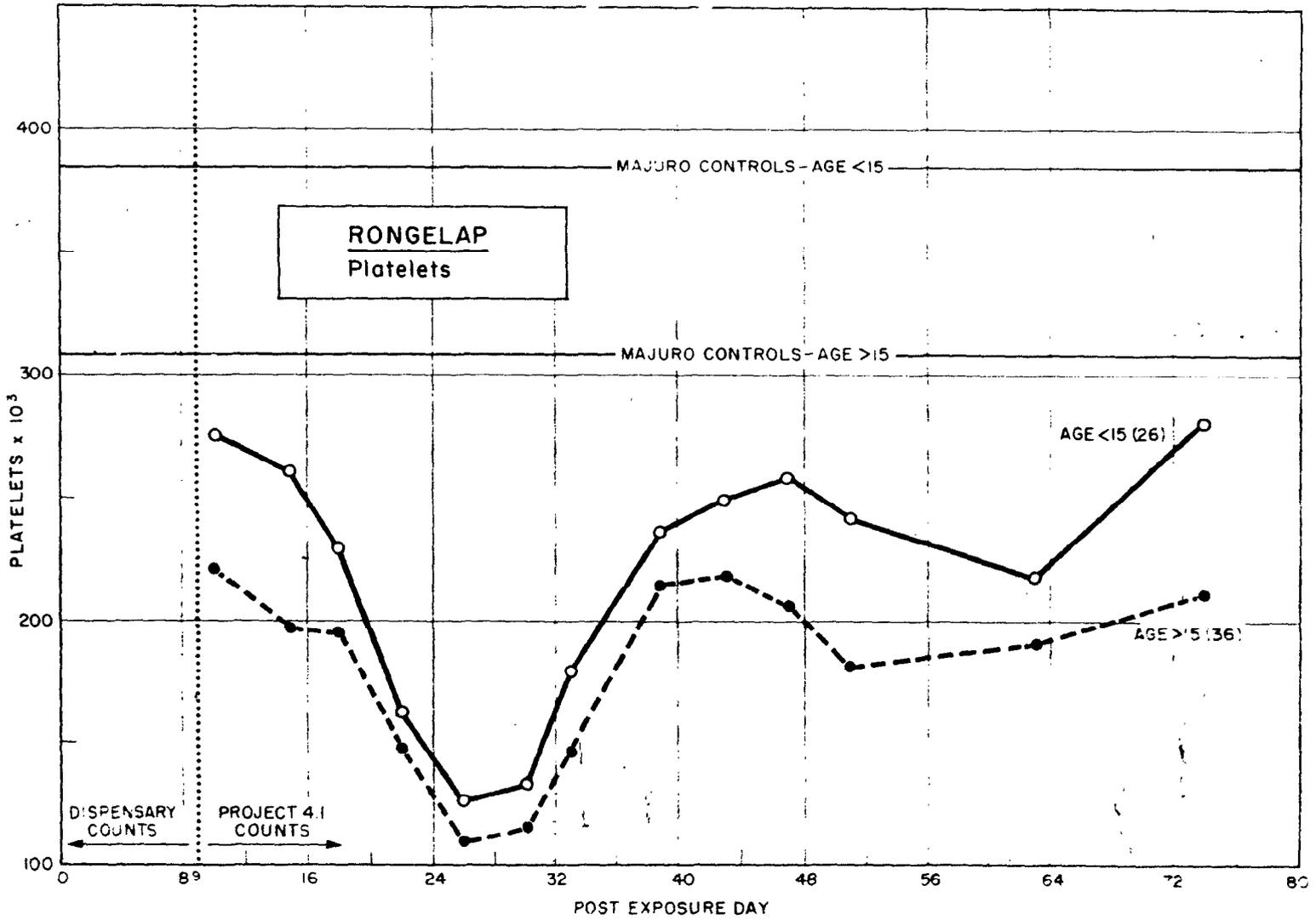


Fig. 4.4 Serial Platelet Changes in Those Less Than 15 Years and Greater Than 15 Years of Age of the Rongelap Group

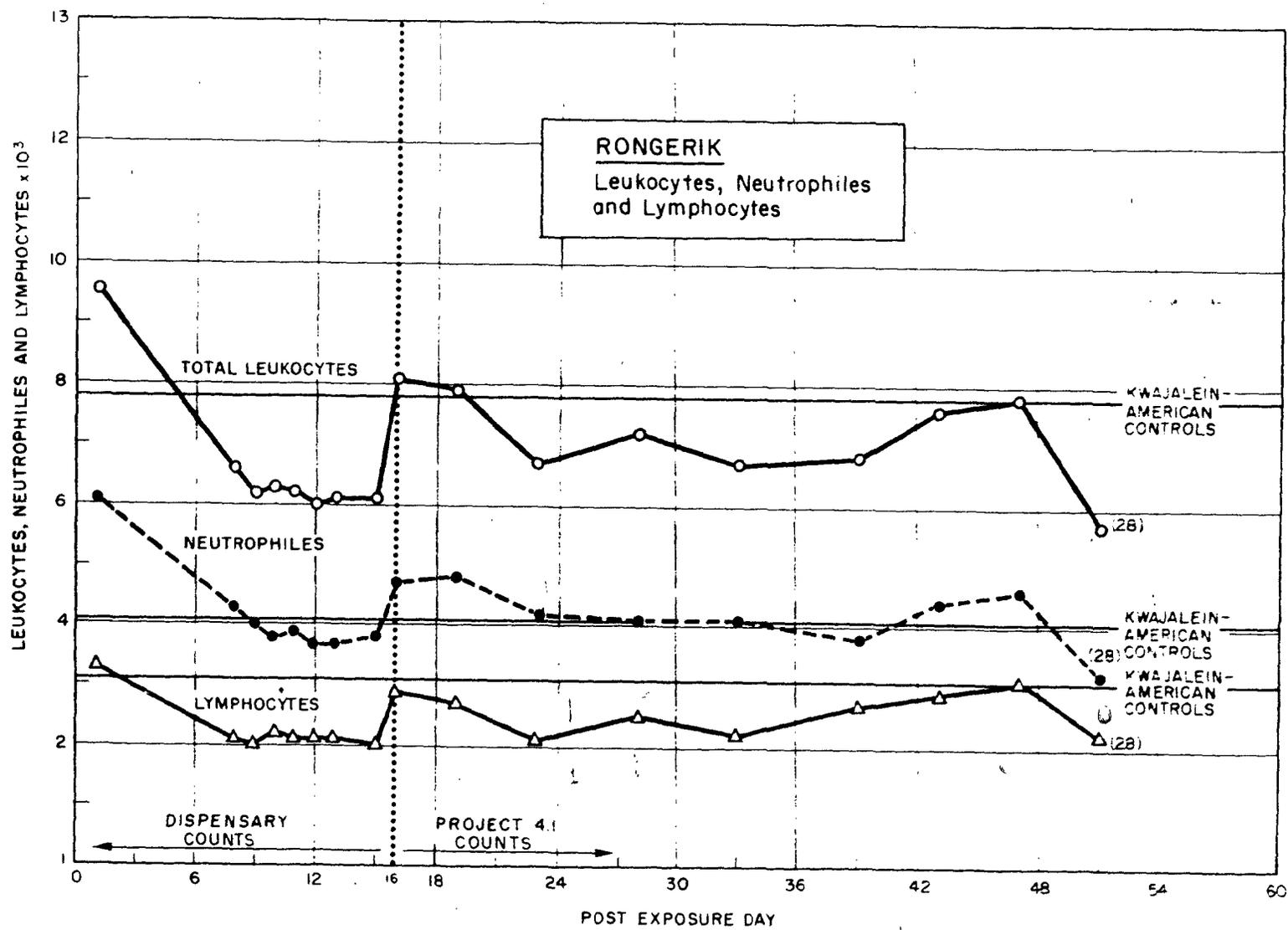


Fig. 4.5 Serial Total Leukocyte, Neutrophile, and Lymphocyte Count in Exposed Americans

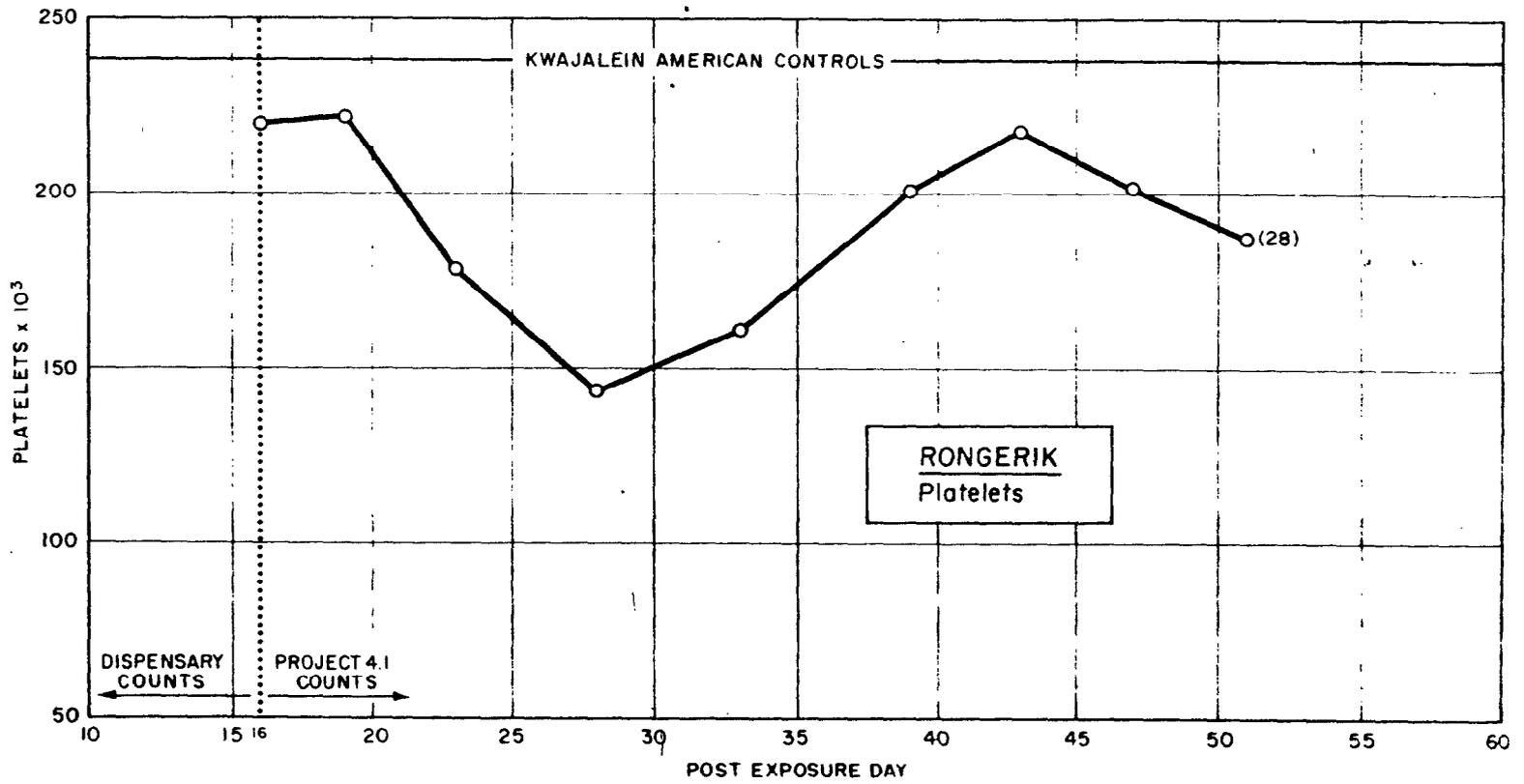


Fig. 4.6 Serial Platelet Counts in Exposed Americans

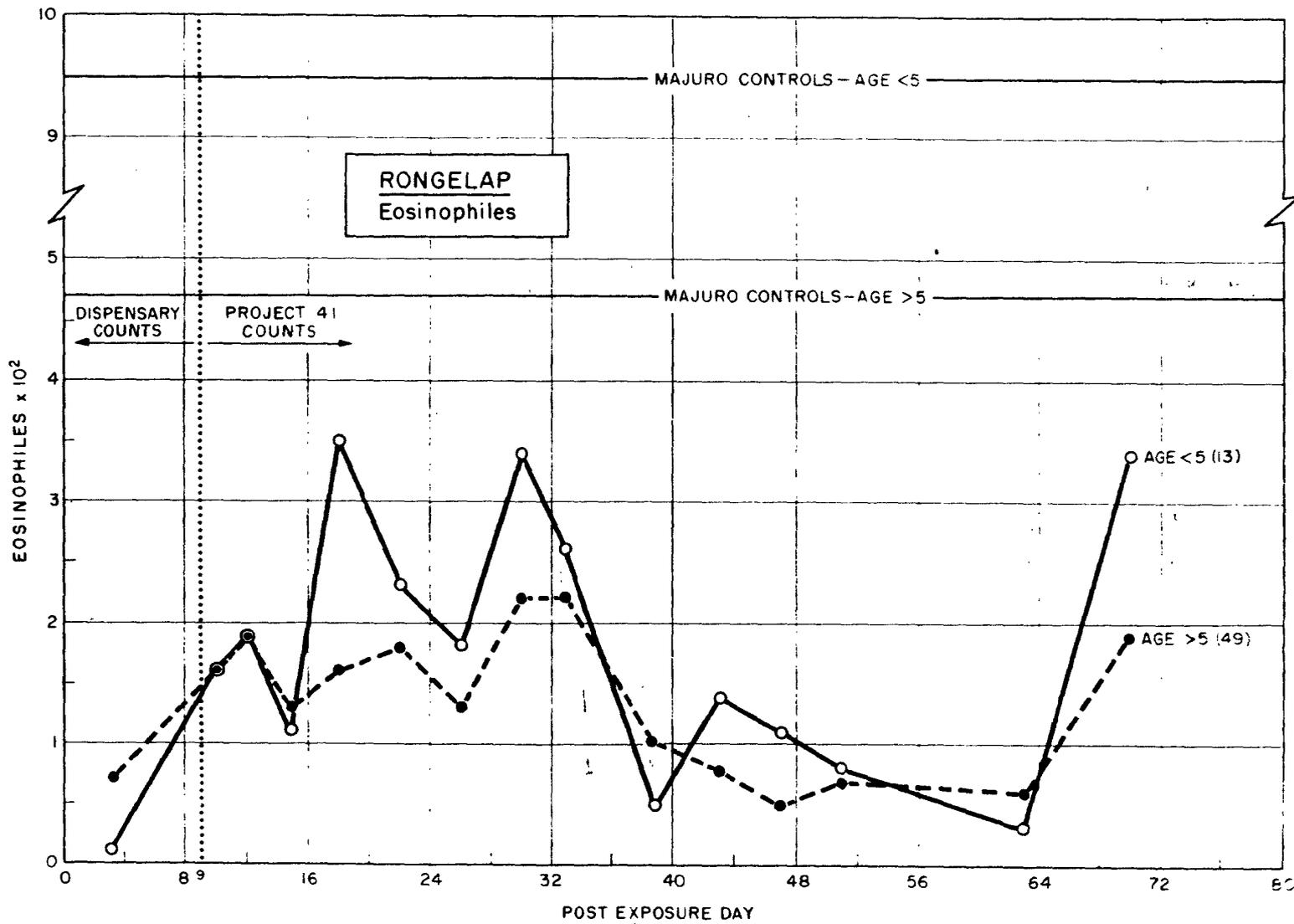


Fig. 4.7 Serial Eosinophile Counts on Those Less Than and Greater Than 5 Years of Age of the Rongelap Group

SECRET

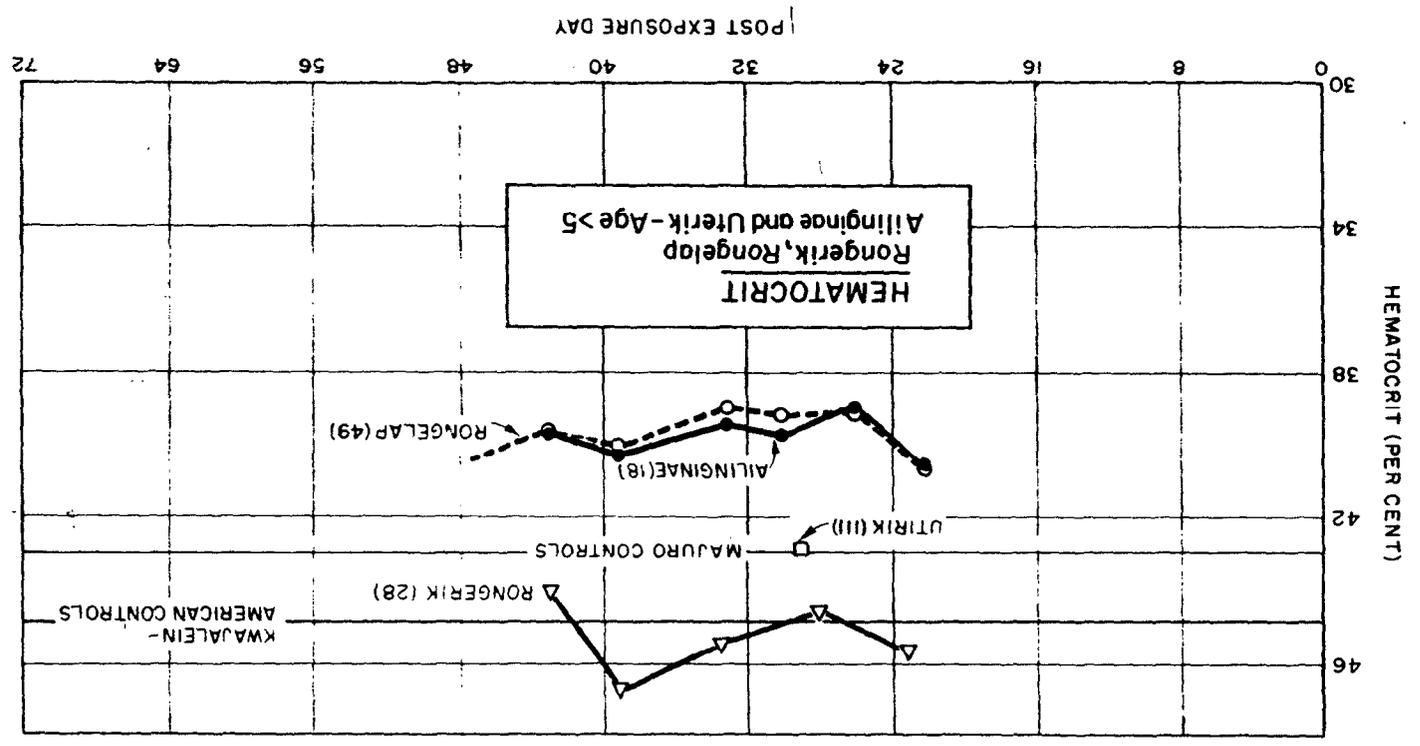


Fig. 4-8 Serial Hematocrits for All Exposed Groups

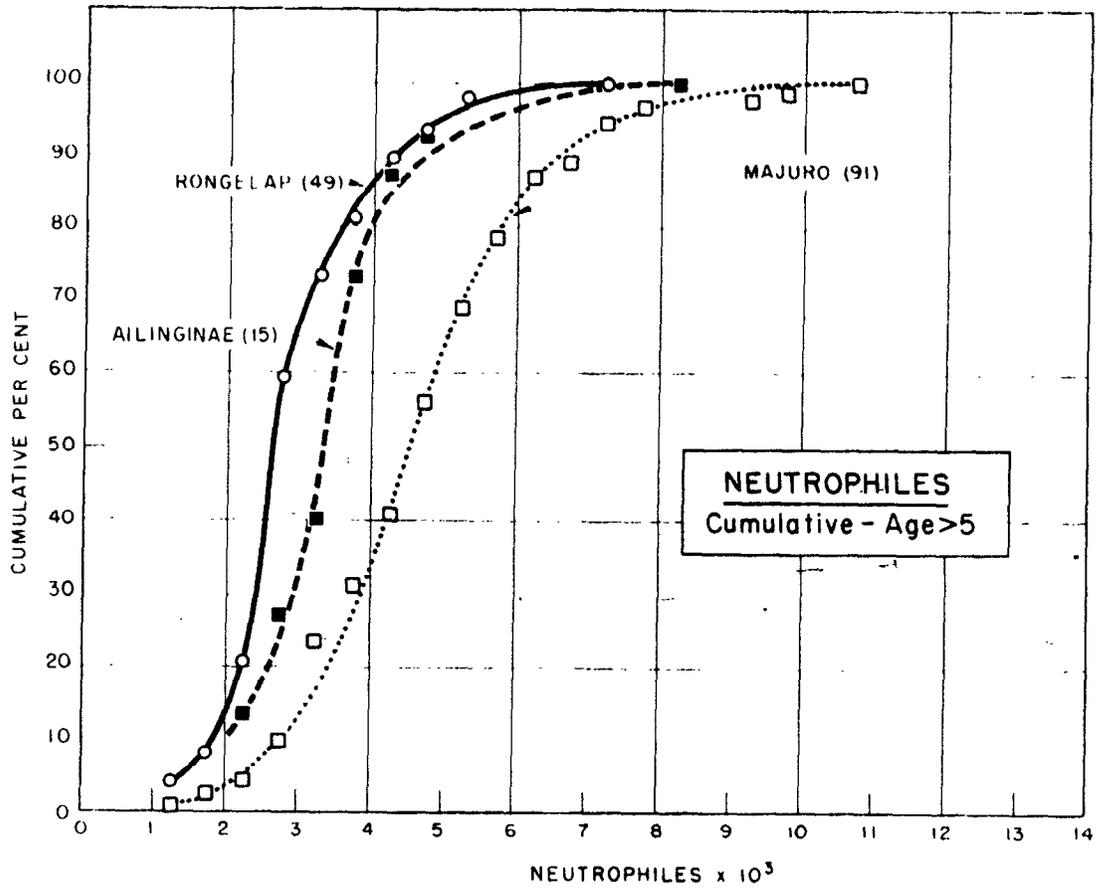
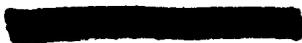


Fig. 4.9 Cumulative Neutrophile Counts for the Rongelap, Ailinginae and Majuro Groups at the Time of Peak Depression



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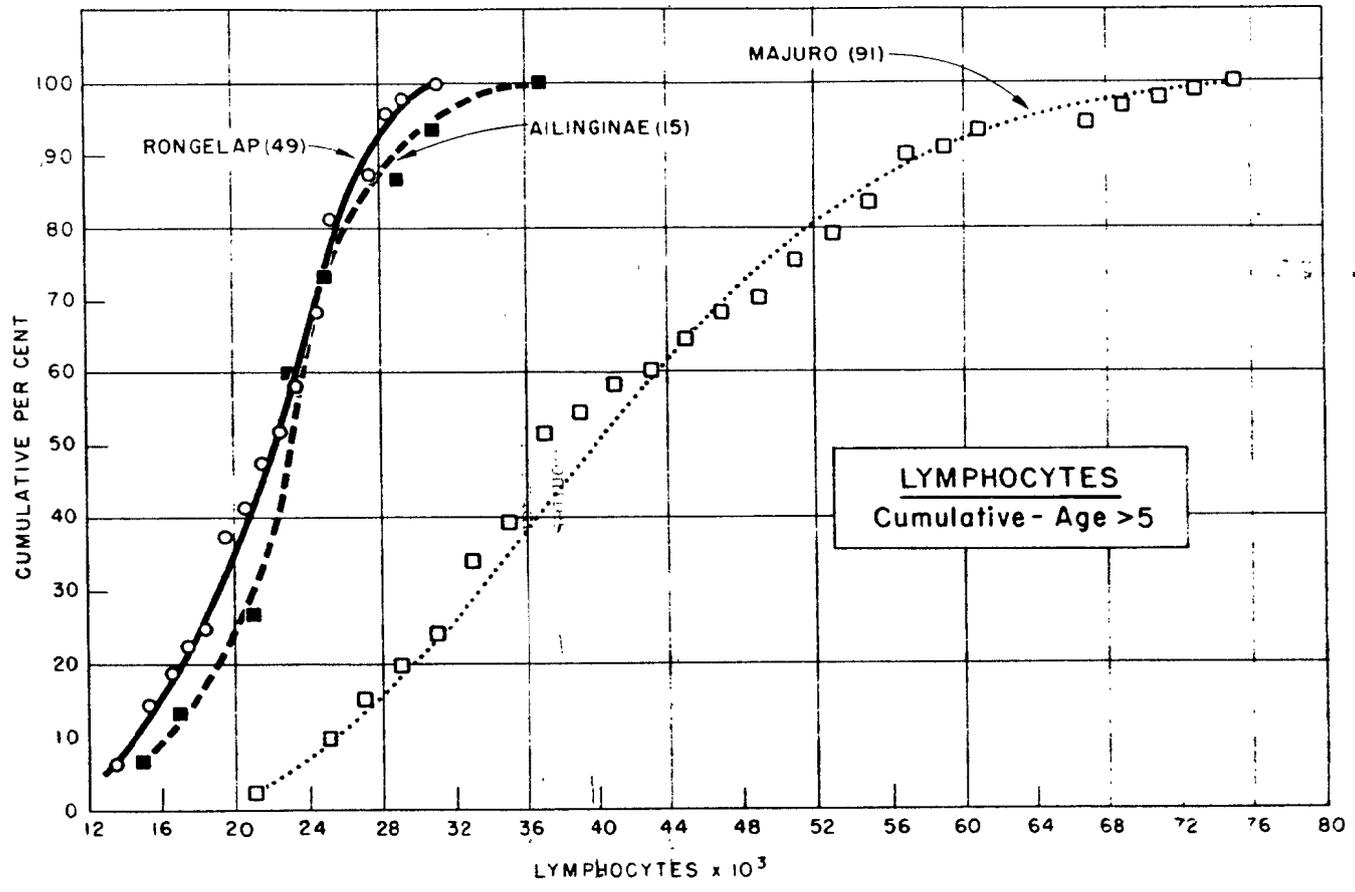


Fig. 4.10 Cumulative Lymphocyte Counts for the Rongelap, Ailinginae and Majuro Groups at the Time of Peak Depression

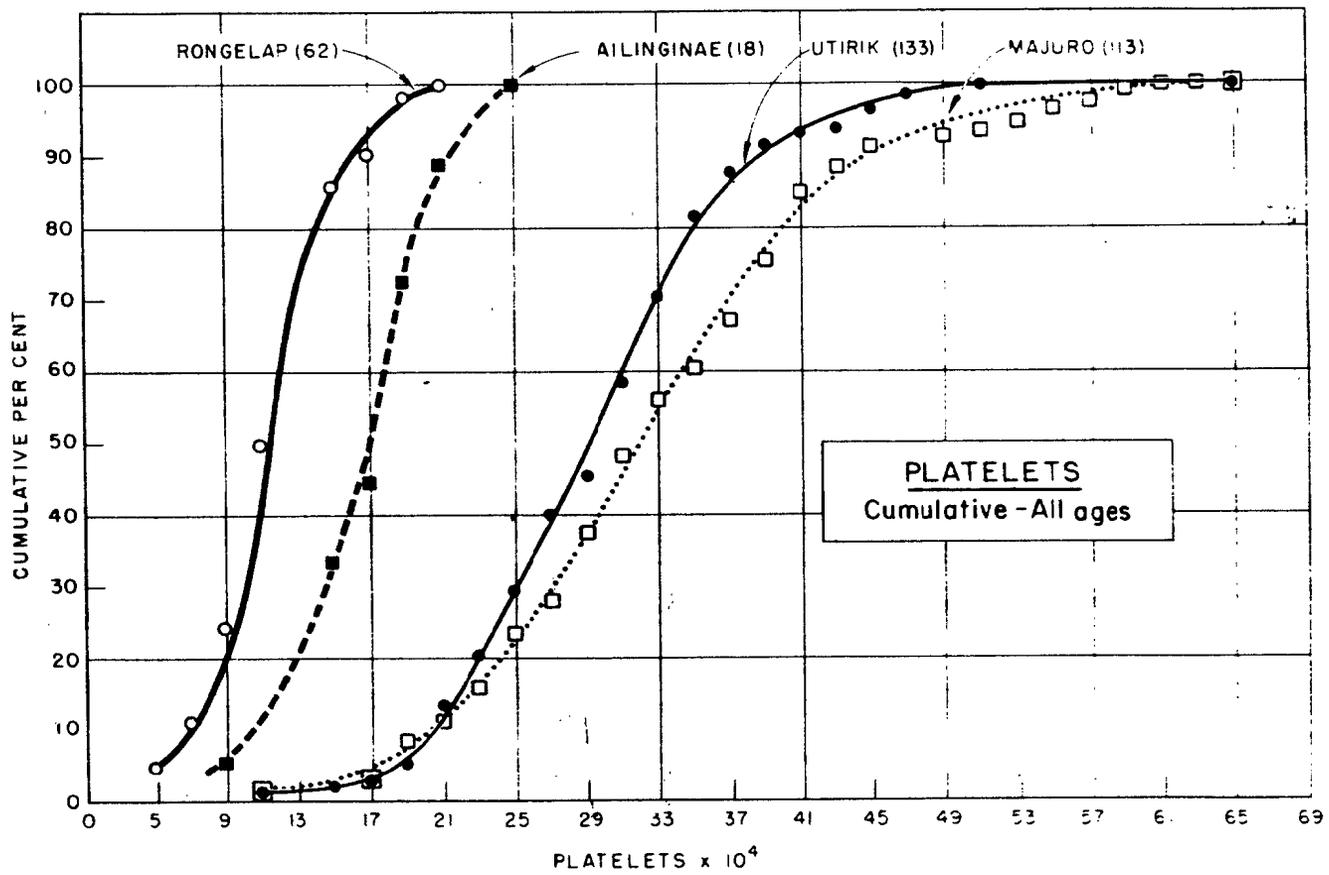


Fig. 4.11 Cumulative Platelet Counts for the Rongelap, Ailinginae, Utirik and Majuro Groups at the Time of Peak Depression

Table 4.6 RONGELAP GROUP MEAN BLOOD COUNTS AT TIME OF PEAK DEPRESSION

Case No.	W.B.C. (Average from Day 39 to 51)	Platelets $\times 10^3$ (Average from Day 26 to 30)	Neutrophils (Average from Day 39 to 51)	Lymphocytes (Average from Day 39 to 51)
Age Less Than 5				
2	7220	110	2870	406
3	7320	155	2770	420
6	5020	115	2570	266
17	6230	105	3350	265
19	5650	115	3070	240
21	4750	85	2670	195
23	7150	195	4100	280
32	5450	95	2600	265
33	5800	85	1670	357
42	5500	80	2520	292
54	4750	145	2620	195
65	6050	105	2520	240
69	4770	115	1420	317
Age 5 to 15				
15	3920	200	1470	232
20	5020	120	3020	195
24	5620	195	3450	197
26	6020	145	3470	232
35	5100	140	2700	215
36	5120	130	2520	247
39	4720	165	2900	155
47	7220	120	4720	225
61	5600	105	2500	297
67	5120	115	2970	192
72	4100	185	1800	197
75	4200	110	2320	172
76	5750	150	2800	287
Age Greater Than 15				
4	6420	130	2650	355
7	5220	195	2520	252
9	5470	125	2700	242
10	4550	105	2770	157
11	3120	85	1570	135
12	4670	150	3270	127
13	4050	105	2370	152
14	4570	55	2770	170
18	6100	45	4320	165
22	4470	130	2500	190
25	6250	110	4050	212
27	6620	110	3600	265
30	5700	85	3920	160
34	5900	125	3650	235
37	5970	130	3120	257
40	5600	140	2450	287
46	4620	135	2350	210
49	6620	180	4050	222
52	5820	160	2970	246
55	4400	135	1450	272
56	6170	125	3520	255
57	5020	55	2020	270
58	4750	80	2600	185
60	6970	160	4050	247
62	8300	110	5170	282
63	4270	65	2560	152
64	5600	70	3220	205
66	6100	145	2820	312
68	4600	120	2400	202
71	7950	105	4950	270
73	3970	60	2630	126
74	9900	155	7250	255
78	5400	95	3350	195
79	7800	70	5120	250
80	5670	100	2920	252
82	5250	130	2620	247

RECORDED

Table 4.7 AILINGINAE GROUP MEAN BLOOD COUNTS AT TIME OF PEAK DEPRESSION

Case No.	W.B.C. (Average from Day 39 to 51)	Platelets $\times 10^3$ (Average from Day 26 to 30)	Neutrophils (Average from Day 39 to 51)	Lymphocytes (Average from Day 39 to 51)
Age Less Than 5				
6	9750	215	3470	5600
8	8350	185	3520	4350
44	4570	180	2350	2070
Age 6 to 15				
48	6220	210	2970	3150
53	6170	240	3700	2500
81	4700	240	2320	2150
Age Greater Than 15				
1	6170	175	3570	2370
16	4670	195	2200	2270
28	6270	115	3720	2270
29	6750	115	4100	2220
31	5650	145	2950	2450
41	5120	110	3050	2270
43	6150	215	3700	2000
45	5650	180	4170	1470
50	7050	95	3970	2900
51	7750	170	4620	2950
59	12400	105	8120	3670
70	5070	185	3000	1750

4.5 RONGELAP GROUP

The absolute neutrophil count of both the younger and older age groups fell during the second week to a value, approximately 70 to 80 per cent of that of the controls (see Fig. 4.1). Following the depression of the total neutrophil count during the second week, the values were unstable until the fifth week. At this time the beginning of a second drop ($P < 0.01$) was noted for both age groups, and a low value of approximately 50 per cent of controls was reached. The count was maintained at approximately 75 per cent of control values from the seventh week to the end of the study. Although both age groups followed the same general time pattern of response, the lower age group was below that of the older group throughout most of the observation period.

The absolute lymphocyte count of the older age group (Fig. 4.2) had fallen by the third day to a value approximately 55 per cent of the control group. This value was maintained throughout the study, and there was no definite evidence of an upward trend during the study. The values for the younger age group likewise fell before the third day to a value approximately 25 per cent of the control, following which there was a significant upward trend. With the total lymphocyte count, there is a consistent difference between the two age groups. However, during the first four weeks the difference is accentuated when expressed as per cent decrease because of the relatively high lymphocyte levels in the lower age control group. After this period the differences expressed as per cent are less marked since recovery was more rapid in the younger age group.

The cellular elements chiefly responsible for the fluctuations in total white blood cell count can be determined by comparing the total white, neutrophil and lymphocyte counts (Fig. 4.3). It is seen that the lymphocyte count remained essentially constant throughout the period of study, while the total neutrophil count fluctuated with a pattern essentially identical to that

of the total white blood count (coefficient of correlation of 0.9). Thus the fluctuations in total count were due to changes in the neutrophile count. This was true of both the older and younger age groups. It can be seen from Table 4.2 that the neutrophile count was consistently greater than the lymphocyte count in the older age group. In the younger groups, differences in the neutrophile and lymphocyte count were less marked and on six occasions the lymphocyte count was greater than the neutrophile count.

Platelets were first counted 10 days after exposure, at which time platelet values of the older age groups were approximately 70 per cent of the control group (Fig. 4.4). Following this, the platelet count fell reaching a low of approximately 35 per cent of control value during the fourth week. The platelet count rose during the 5th week and reached the value noted for the initial counts on the 10th day. A second decrease in the platelet count ($P < 0.01$) developed during the 7th and 8th weeks, and values remained at approximately 70 per cent of the control group during the remainder of the observation period. The pattern of platelet counts in the below 15-year group was remarkably similar to that noted in the older age groups. Differences between the age groups were less apparent if the platelet counts were expressed as per cent of the control group.

4.6 AILINGINAE GROUP

In this group there were only three individuals below age 5. For this reason, remarks will be confined essentially to the older age group.

The absolute neutrophile count fluctuated around the control value for the first six weeks of observation (Table 4.3). At this time the counts began to fall, and a value approximately 75 per cent of the control count was reached and maintained throughout the duration of the observation period. The lymphocytes in this group fell to a value of 55 per cent of normal during the first week. The counts then fluctuated around this value throughout the period of observation, and no definite upward trend of the lymphocyte count was noted during the period of observation.

As was noted in the Rongelap group the lymphocyte counts remained at an essentially constant low level throughout the period of observation. The total leukocyte count in this group also reflected changes in the neutrophile count.

The platelet counts in the Ailinginae group were low, approximately 75 per cent of normal, when first enumerated on post-exposure day 10. The counts remained at this level during the second and third week; however, a definite fall in count was noted during the fourth week when a low value 45 per cent of control was attained. The counts returned during the fifth week to a value approximately 70 per cent of the control level, where they remained for the duration of the observation period. A secondary fall, as observed in the Rongelap group was not detected.

4.7 UTIRIK GROUP

In the greater than 5 age group the total white blood cell and neutrophile counts were depressed slightly below control values during the first and second weeks (Table 4.4). The lymphocyte counts were below control levels consistently, and the total white count equal to the control value obtained on day 29 was due to a neutrophilic leukocytosis.

Platelet counts on the 29th day were significantly lower than on the 19th day and were lower than control values. The 29th day coincides with the time of maximum depression for the more heavily exposed groups.

4.8 RONGERIK GROUP (AMERICANS)

The neutrophile count in general reflected the time course of the total leukocyte count (Fig. 4.5). Neutrophiles accounted almost entirely for the marked rise in total count on post-exposure day one, and the values for absolute neutrophile count fluctuated near the control

values thereafter in the course of the study. The lymphocyte counts fell to below control levels in the first few days, and remained at a level approximately 75 per cent of the control value throughout most of the remainder of the observation period.

In the Rongelap and Ailinginae groups the fluctuations in the total leukocyte count were accounted for almost entirely by changes in the total neutrophile count. The Rongerik group differed since the changes in total leukocyte count were reflected almost equally in the lymphocyte and neutrophile count. The significance of this difference in response in the two groups is not apparent.

The platelet counts (Fig. 4.6) were not markedly depressed when the initial counts were taken during the third week. At the end of the third week, however, the platelet count began to fall to reach a low of approximately 60 per cent of control levels at the end of the fourth week. The value then returned to approximately the control level at the start of the seventh week, following which a second depression was noted. The platelet counts were at a level of 80 per cent of the control value at the time of the last observation during the eighth post-exposure week.

4.9 MONOCYTES AND EOSINOPHILES, ALL GROUPS

From Table 4.2 it is seen that the mean monocyte count for the Rongelap group rose abruptly from an early value below control levels to a well defined peak on day 12, following which it fluctuated at values below the control level for the duration of the observation period. A similar time trend was noted in the Ailinginae and Utirik groups.

The eosinophile count in the older age individuals, Rongelap group, rose from very low levels observed on day 3 to values approximating 35 per cent of control during the second week, where it remained from the third to the fifth week (Fig. 4.7). The counts then decreased ($P < 0.01$), and remained at a value approximately 15 per cent of control throughout the remainder of the study. The time trend of response was similar in the younger age individuals, however, changes in the younger age group were relatively greater if considered in terms of the control values. Similar trends in eosinophile count were not evident in other exposure groups.

It is possible that the rise in eosinophiles represents that reported as occurring "... two to three weeks after short-wave-length radiation".⁴⁶

4.10 HEMATOCRIT, ALL GROUPS

The hematocrit values for all exposed groups are shown in Table 4.8 and in Fig. 4.8 (a detailed breakdown of hematocrit by age and sex for control groups is given in Table 4.1). When hematocrits were first done on the 22nd day, mean values for the Rongelap and Ailinginae groups were below those of the control population. A significant trend in values after this time could not be detected statistically.

4.11 MORPHOLOGY OF PERIPHERAL BLOOD

Significant morphological cellular changes, with the exception of a small number of abnormal mononuclear cells* seen in a number of individuals during the period of neutropenia, were not observed. Similarly altered cells have been observed previously.⁴⁶ Complete evaluation of these changes in the present study would necessitate an exhaustive serial study of the hematology slides.

*There was considerable difference in opinion in respect to classification of these cells. They were classified as atypical monocytes, degenerating lymphocytes, atypical myelocytes, monocytoïd lymphocytes, and lymphocytes in transition to myelocytes. At the time of this report there was no unanimity of opinion in respect to classification and significance of these cells.

Table 4.8 HEMATOCRIT, ALL EXPOSURE GROUPS

Day	Rongelap						Allinginae						Utrik		Rongerik
	Males		Females		Comb		Males		Females		Comb		Comb	Comb	Males
	<5*	>5	<5	>5	<5	>5	<5	>5	<5	>5	<5	>5	<5	>5	Adults
22	38.1	42.5	38.0	39.1	38.1	40.7	37.5	40.6	37.0	39.4	37.3	40.6			
23															45.7
26	35.8	40.3	35.3	38.0	35.6	39.1	36.5	43.2	38.0	36.7	37.0	38.9			
28															44.5
29													36.9	42.9	
30	38.1	40.5	34.3	37.7	36.4	39.2	36.0	44.6	32.0	37.2	34.7	39.7			
33	36.8	41.0	34.8	37.3	35.9	39.0	35.5	43.8	38.0	37.2	36.3	39.4			45.4
39	36.4	41.6	36.0	37.6	36.2	40.0	35.0	45.6	35.0	37.6	35.0	40.3			46.7
43	35.7	41.0	36.2	37.6	35.9	39.6	36.0	45.2	35.0	37.0	35.7	39.7			44.0
47		42.8		38.3		40.3									
Controls	38.5	45.2	37.3	40.6	37.8	43.0	38.5	45.2	37.3	40.6	37.8	43.0	37.8	43.0	44.9

*Age in years.

4.12 COMPARISON OF HEMATOLOGICAL FINDINGS IN CHILDREN AND ADULTS, RONGELAP GROUP

It is seen from Tables 4.2 to 4.4 and Figs. 4.1, 4.2, and 4.4 that differences in the degree of depression of cellular elements were present between children and adults. In Table 4.9, the mean values of the neutrophile, lymphocyte and platelet counts at time of peak depression for each element are given in terms of absolute count and per cent of appropriate control value (mean platelet counts were calculated for the less than 5 and greater than 5 age groups for this comparison).

Table 4.9 COMPARISON BY AGE OF MEAN NEUTROPHILE, LYMPHOCYTE AND PLATELET COUNTS IN THE RONGELAP GROUP AT THE TIME OF PEAK DEPRESSION

Type of Cell	Absolute Count $\times 10^3$		Per Cent of Control	
	Age <5	Age >5	Age <5	Age >5
Neutrophile	2.7	3.1	56	64
Lymphocyte	2.9	2.2	40	54
Platelets	115	122	30	36

It is seen that in terms of absolute counts, the children showed a greater depression of the neutrophile count, and the same degree of depression of the platelet counts and less depression of the lymphocyte count. These differences can be most easily described at the time of peak depression. Expressed as per cent of control, all elements were affected more markedly in the younger age group. These results would indicate that children are more sensitive to radiation, or that other biological or physical factors resulted in a relatively greater effect.

4.13 DISCUSSION

4.13.1 General

An estimation of the severity of radiation damage incurred can be attempted by comparing the present results with previous hematological data on total body exposure. The present data

represent the only large series in which systematic serial counts on the same individuals have been possible, and thus they comprise the most complete data available on human beings exposed in the high sublethal range. It is also of importance, therefore, to examine the present results in conjunction with past experience in an effort to gain a better understanding of the hematological response of human beings exposed to penetrating radiation in the sublethal range.

In the following discussion it will be generally assumed that the hematological effects noted were due primarily to the penetrating gamma radiation received. The beta radiation injury of the skin may have contributed to fluctuations in the white count during the period of active lesions during the third, fourth, and fifth week, but is considered not to have contributed significantly to depression of any peripheral elements (see chapter 3). The degree of internal contamination with fission products (see chapter 5) was probably too small to contribute significantly to the early hematological effects observed. Although it is not possible to say with certainty that these added factors did not materially affect the hematological pattern seen, it will become evident in the discussion that the changes observed are not inconsistent with those to be expected from exposure to penetrating radiation alone. Thus the hematological changes noted are considered to be the result of a single exposure to penetrating gamma radiation, delivered at rapidly decreasing dose rates over a period of approximately two days. Unless otherwise stated all discussion will be limited to the adult Rongelap group.

The principle sources of previous data available for comparison, and the characteristics and limitations of each are summarized in Table 4.10. Perusal of the table will make apparent the difficulties involved in attempting strict comparisons; however, some statements can be made despite the obvious limitations. For easy reference, "normal" values for peripheral blood counts, from the present data and from the literature are presented in Table 4.11.

4.13.2 Comparison with the Japanese Data

The limitations stated in Table 4.10 apply to the Japanese low dose groups* E to H in particular, in which values given^{46,47} are pooled and include individuals located at the time of the bombing such that they may not have received significant exposure. Hence, while the pattern of change with respect to time is of value, absolute counts probably are high. The time course of hematological change in the Rongelap people corresponded most closely with these low exposure Japanese groups in which definite signs of severe radiation exposure were present in some individuals but in which essentially no mortality occurred (initial hemotological studies on the Japanese terminated at 15 weeks). The early period up to approximately six weeks was characterized by considerable variation in total white count in both the Rongelap and Japanese people. This fluctuation may be associated with the presence of thermal or other injuries in the Japanese or the active skin lesions in the Marshallese, or may correspond to the "abortive rise" noted for animals following exposure.^{51,54} From the sixth week until the termination of the acute studies on the Marshallese during the tenth week, the Japanese and Marshallese counts remained at similar levels.

The neutrophile count in both the Japanese and Marshallese in general paralleled the total white count. The lymphocyte count in both groups was depressed early and remained depressed at values of approximately 2000 until week 10. The high value of 2692 reported for the Japanese for weeks 12 to 15 must be suspected of being high for the reasons given in section 4.13.2.

Three characteristics of the Japanese hematological trends should be pointed out: a) while high dose exposure groups with significant mortality showed early depression with a definite low point at four weeks, the lower dose groups showed no definite minimum at four weeks but rather a continued depression until the eighth or ninth weeks. b) While the mean values for total white and neutrophile counts for even the heavily exposed groups had returned to within

*The Japanese casualties were divided into groups A to H on the basis of degree of exposure as determined roughly by distance from the hypocenter and approximate degree of shielding. In groups E to H essentially no mortality ascribable to radiation exposure occurred in the first 3 or 4 months.

Table 4.10 CHARACTERISTICS OF AVAILABLE DATA ON THE HEMATOLOGICAL EFFECTS OF PENETRATING RADIATION

Source of data Characteristics	Japanese Bombings ^{46,47}	Clinical Radiotherapy ⁴⁸	Laboratory Accidents ^{49,50}	Exposed Marshallese	Large Animals ⁵¹
Numbers in Groups	Large	Small	Small	Large	Large
Adequacy of Controls	Fair	Poor	Poor	Good	Very good
Serial Counts	No	Yes	Yes	Yes	Yes
Counting Techniques*	Fair	Fair	Good	Good	Good
Chance of bias due to sampling techniques	Large	Large	Large	Small	Small
"Normal" individuals	Yes	No	Yes	Yes	Yes
Internal contamination	None	None	None	Minimal	None
Additional trauma (burns etc.)	Yes	No	Yes	Yes	No
Species extrapolation necessary	No	No	No	No	Yes
Type of radiation	Gamma, some neutrons	Hard x rays, gamma	Gamma, neutrons, x rays, betas	Gamma, beta to skin	Hard x rays, gamma
Dosage estimation	Poor	Good	Poor	Fair	Good
Single exposure	Yes	Usually no	Yes	Yes	Yes
Dose rate	Instantaneous	~5r/min	Instantaneous	Varying, ~5r/hr	~10r/min
Body region	Total body	Usually partial body	Total and partial body	Total body; beta to skin	Total body
Dosage Range	Sublethal & lethal	Sublethal	Sublethal & lethal	Sublethal	Sublethal & lethal
Geometry	Narrow beam	Narrow beam	Narrow beam	360° field	Narrow beam
Depth dose curve	Moderate fall off	Variable	Rapid fall off	Essentially flat	Variable rapid fall off to flat

*Same technicians for all counts; rigidly standardized techniques throughout etc.

Table 4.11 MEAN PERIPHERAL BLOOD COUNT VALUES FOR SEVERAL CONTROL POPULATIONS ($\times 10^6$)

Source of data Determination	Japanese Kure,		Americans ³⁷	Americans ³⁸	Kwaj-	Majuro Controls
	1947-1948 ⁵²	1948-1949 ⁵³			American Controls	
Total White Count	9.9	9.5	7.4	7.0	7.8	9.7
Neutrophiles	5.5	5.0	4.4	4.3	4.1	4.8
Lymphocytes	2.9	2.8	2.5	2.1	3.1	4.1
Monocytes	0.6	0.6	0.3	0.4	0.3	0.2
Eosinophiles	0.9	1.0	0.2	0.2	0.3	0.5
Basophiles		0.1	0.0	0.0	0.0	0.1
Platelets				250†	238	308

*Age 21 years

†The mean value for 50 normal young American men, using the technique employed in the present study was 257,000

the "normal" range for individual counts by the eighth or ninth week, neither of these values for either the high or low exposure groups had returned to the mean levels of any of the control populations listed in Table 4.11 at this time, nor by the end of the study at 15 weeks.* c) Lymphocyte counts remained depressed at least through the 12th week and probably through the 15th week.

The present findings in the Marshallese are in accord with these characteristics, namely, a) total white cell and neutrophile counts showed no definite minimum at four weeks as evidenced in Japanese groups A to D, but rather fluctuated during the first few weeks with minimum mean counts occurring in the sixth week or later. b) Neutrophile counts were unstable over the first five weeks, and recovery to control levels was not complete by the 10th week. c) Lymphocyte counts remained depressed throughout the period of observation.†

Platelet data in the Japanese are not sufficient to allow more than rough qualitative comparisons. This is unfortunate since changes in platelet counts in the present studies appeared to show a more consistent pattern than did the leukocyte counts. Platelet counts on one individual considered as atypical response in a non fatal Japanese⁴⁷ indicated an apparent low at approximately day 30. This time trend agrees with that seen in the Marshallese and Americans exposed to fallout radiation.

It is worthy of note that the period of peak incidence of purpura in the Japanese victims occurred between the 25th and 30th day, which corresponds to the time of maximum platelet depression in the exposed Marshallese.

4.13.4 Comparison with Data from Laboratory Accidents

Although in the Los Alamos and Argonne accidents^{49,50} the type of radiation and the conditions of exposure were markedly different from either the Japanese or the Rongelap situations, a large component of penetrating gamma radiation was received and thus attempts at comparison may be of value. Some findings in the hematological responses are pointed out: a) a uniform early rise in white and neutrophile counts over the first few days, similar to that seen early in the Rongerik American group was observed uniformly.‡ b) Of three high-exposure but non-lethal cases, the total white and leukocyte counts continued to show some degree of depression into the seventh week or beyond. c) The lymphocyte counts in individuals exposed to as little as 50 rem showed an initial marked depression. In most cases the lymphocyte counts remained at low levels throughout the period of observation. d) Platelet counts were done by a different method, and absolute counts are therefore not comparable. However, of the three high-dose survivors, times of maximum depression were not inconsistent with the value of 30 days obtained in the present studies.

The Argonne Laboratory accident⁵⁰ involved four individuals who were estimated to have received 136, 127, 60, and 9 rep, respectively. The findings in the two highest exposed individuals in general were consistent with those in the present study. An initial neutrophilic leukocytosis was followed by fluctuations in total count, with low values continuing into the seventh week. Recovery was not complete by the 20th week. The lymphocyte count depression was rapid and marked, and recovery was not evident by the 20th week. Minimum values for the platelet count were obtained between the 25th and 31st day.

4.13.5 Comparison with Animal Data

The time trends and severity of peripheral blood count change following total body radiation in animals has been critically examined recently,⁵¹ and the following general conclusions are presented.

*Counts 2 years later were not significantly different from control Japanese values.⁵²

†Counts on the Rongelap people 6 months after exposure showed no elevation of the mean total white count, neutrophile or lymphocyte counts over values obtained during the 10th week.

‡No counts were taken on the Rongelap and Ailinginae groups during this early period.

a) An initial rise in total white count (reflected in the neutrophile count) may occur. There after the magnitude of depression of the total white and neutrophile counts, and within limits their duration are a function of radiation dose: A secondary or abortive rise in the total white count (reflected in the neutrophile or lymphocyte count) may occur, followed by a second decrease. There is little species difference in the rate of depression of the total white or neutrophile count at comparable doses; however, the rate of recovery and time for complete recovery is quite different in various species. Small animals (mouse, rat, hamster) show relatively complete recovery to control levels, even at doses in the lethal range, by the end of the fifth week or earlier. Data on dogs are inadequate to indicate when recovery is complete; however, return to control levels at high dose levels had not occurred by the fifth week. Swine require 9 to 15 or more weeks for complete recovery.

b) The response of lymphocytes is essentially identical in all animal species. Depression can be detected within a few hours, and recovery from the minimum values (achieved in 36 to 48 hours) requires longer than does neutrophile recovery. Lymphocytes fall to very low levels at doses well below the lethal range, and increasing dose results in no or minimal further decrease in count. Lymphocyte depression appears to have no causal relationship with acute radiation deaths.

c) Platelet counts have been studied most extensively in dogs.⁵⁶ As with neutrophiles, the rapidity and magnitude of depression is a function of dose below the lethal range. Maximum depression occurs by the 9th or 10th day with doses in the high lethal range, by the 10th to 15th day at sublethal levels. Recovery begins during the third week, but is not complete by the 30th day when most studies have been terminated. Insufficient data are available to indicate the time required for complete recovery.

Considerable evidence including studies in the mouse using splenic homogenates, induced bacterial infections and spontaneous infections have indicated that critical neutrophile levels exist below which survival is correlated with the absolute neutrophile count following whole body irradiation. From data on dogs, it appears that survival is likely unless neutrophile counts remain below 1200 cells for a period of time.

Platelet data on dogs indicate that animals with external purpura have platelet counts of 50,000 or below.

Insufficient data on large animals are not as yet available to quantify the extent of maximum depression of either the neutrophile or platelet counts as a function of dose in the sublethal range. The response of the platelet count in the present study was much less subject to fluctuation than were the neutrophile or lymphocyte counts. For the preceding reasons, systematic investigation of the platelet and leukocyte counts in large animals as a function of dose in the sublethal range are indicated.

It is not possible to say at present whether severity of exposure, or of radiation damage correlates better with absolute levels of peripheral blood count, or with degree of change from control or pre-exposure levels. Some evidence on this point can be gained by comparing the degree of depression of the neutrophile and platelet counts in the Ailinginae and Rongelap groups, both of which had essentially the same calculated exposure but for which control hematological values were considerably different (the lymphocyte count is not suitable for comparison since degree of depression was essentially the same in these groups and the higher-dose Rongelap group). At the time of peak depression for each element, both the neutrophile and platelet counts were essentially identical in terms of absolute counts, but considerably different in terms of the respective control values. Thus some evidence is afforded that absolute counts, rather than counts relative to control values, may be the more reliable index of exposure in this dose range.

4.13.6 Approximation of Minimal Lethal Dose for Man

Some indication of severity of exposure can be gleaned from a comparison of minimum individual counts in Japanese groups exposed at Hiroshima and Nagasaki in which fatalities occurred. In general, a significant number of deaths was encountered only in individuals whose neutrophile count fell below 1000. In the Rongelap group 42 or approximately 50 per cent had

neutrophile counts below 2000 at some time during the observation period, and 10 per cent had counts below 1000. By this criterion, then, the effective dose received by the Rongelap people approached the lethal range.

In the dog and mouse,⁵⁶ approximately 50 to 100 r are required to lower the neutrophile count by 1000 cells min^{-3} in the high sublethal dose range. If these data can be applied to man, an additional 50 to 100 r would have placed the dose in the lethal range. On the other hand, however, it is clear from the present data that neutrophile counts between 1000 and 2000 in human beings are well tolerated. Human beings with these levels of neutrophiles show no clinical evidence of illness, are physically active, and do not need prophylactic antibiotic therapy.

The Rongelap people are estimated to have received 175 r as calculated from dose rate readings measured in air in the plane fission product field. From the preceding paragraph it is seen that an additional 50 to 100 r of laboratory radiation or an average of 75 r, probably would have resulted in some fatality. Correcting this average value geometry,* it follows that the minimal lethal dose (MLD) for man exposed in a fission product field is approximately 225 r measured in air.

It is possible also to estimate the added increment of dose that would have resulted in some mortality among the Rongelap people from consideration of the minimum platelet counts observed, the platelet levels in dogs exposed in the high sublethal range,⁵⁵ and the estimated rate of decrease of platelet level with increasing dose in this dosage range. Such an analysis leads to the same conclusions as those derived from neutrophile data.

4.13.7 Peripheral Counts as an Index of Severity of Exposure

The relative value of the several hematological determinations in estimating the degree of exposure, as well as the approximate dose ranges over which maximum sensitivity for each determination exists, can be estimated by comparing the degree of hematological change among the several exposure groups. The relative degree of change in neutrophiles, lymphocyte, and platelets can be seen in Tables 4.2 to 4.5 and Figs. 4.9 to 4.11. Lymphocyte counts were depressed appreciably even in the low-exposure Utrik group. In the higher dose groups, however, with widely different physical estimates of exposure the lymphocyte counts showed essentially identical degrees of depression. The lymphocyte counts of the Rongelap and Ailinginae groups were constantly depressed at a level of approximately 2000 cells. Thus while sensitive at very low doses, this endpoint may be a poor index of the degree of exposure at higher doses. The total neutrophile count of the Rongelap group was consistently more depressed than was that of the Ailinginae group and the difference was of the order of 500 to 1000 cells. However, day-to-day wide fluctuations in the neutrophile counts occurred. Accordingly this endpoint appeared to be of limited usefulness as an index of relative exposure severity except when counts on groups to be compared are performed at the same time.

The platelet count showed a more systematic trend than did the neutrophile count. Differences between the low-dose Utrik group and controls at the time of maximum depression for all groups could be detected, and appreciable differences existed between the means for the Marshallese higher exposure groups. Platelet counting is as easily carried out and more reproducible than leukocyte counts.^{35,57} Thus the platelet count may prove to be a useful index of degree of exposure throughout the sublethal range.

The above considerations are in accord with previous findings on human beings and animals.

*From geometric and depth dose considerations set forth in chapter 1, 1 r measured in air in a fission product field is equivalent in its effect on man to approximately 1.5 r of penetrating x- or gamma radiation under geometric conditions usually used in the laboratory. Thus the minimal lethal dose for man exposed to penetrating radiation under the usual laboratory conditions would be approximately 335 r.

4.13.8 Conclusions

1. Consideration of the degree of depression of peripheral cellular elements indicates that exposure of these Rongelap people was moderately severe, probably within 50 and at most 100 r of the level where some fatalities would have resulted.

2. The degree of effect evidenced in the Rongelap people is not inconsistent with the physical estimates of gamma dose received. Beta lesions of the skin, and the low levels of internal radioactive contamination observed are considered not to have contributed significantly to the hematological changes seen.

3. The extensive serial hematological data obtained, considered in connection with previous data, allow reasonably accurate characterization of the hematological response of human beings exposed to single doses of penetrating radiation in the high sublethal range. The time course of events is different from that observed in large animals and may be described as follows:

a) The total white count increases during the first two or more days and then decreases below normal levels. The total count then fluctuates over the next five or six weeks, with no definite minimum and with some values above normal (the presence of thermal or beta lesions, or other acute processes during this time may account in part for these fluctuations). The count becomes stabilized during the seventh or eighth weeks at low levels, and minimum counts probably occur at this time. A definite trend upward is apparent in the ninth or tenth weeks; however, complete recovery may require several months or more.

b) The neutrophile count parallels the total white blood cell count. Complete return to normal values does not occur for several months or more. The initial rise in total white count is due to a neutrophilic leukocytosis.

c) The drop in lymphocytes is early and profound. No evidence of recovery may be apparent several months after exposure, and return to normal levels may not occur for months or years.

d) The platelet count, unlike the fluctuating total leukocyte count, falls in a regular fashion and reaches a low on the 30th day. Some recovery is evident early; however, as with the other elements, recovery may not be complete several months after exposure.

4. As an index of severity of exposure, particularly in the sublethal range, the total white or neutrophile counts are of limited usefulness because of wide fluctuations and because several weeks may be required for maximum depression to become evident. The lymphocyte count is of more value in this regard particularly in the low dose range, since depression occurs within hours of exposure. However, since a marked depression of lymphocyte counts occurs with low doses and since further increase in dose produces little more depression, this index is of little value at the higher doses.

5. Platelet counts showed a regular pattern of change in the present studies, with the same time of maximum depression in all exposure groups and with the degree of depression roughly proportional to the calculated doses. It appears, therefore, that the platelet count has considerable promise in the sublethal range as a convenient and relatively easy direct method of determining the degree of exposure.

CHAPTER 5

INTERNAL RADIOACTIVE CONTAMINATION

5.1 INTRODUCTION

A study of the nature and extent of the internal radioactive contamination in the exposed human beings was initiated on the 16th day post-detonation, with the collection of 24-hour urine samples from the Rongelap, Ailinginae and American groups. Additional information on the extent and nature of the radioactive contamination was obtained from domestic animals, foodstuffs, water and soil collected on the contaminated atolls and sent to the U. S. Naval Radiological Defense Laboratory, the Naval Medical Research Institute, the New York Operations Office of the Atomic Energy Commission, and the Los Alamos Scientific Laboratory for gross activity measurements and radiochemical analyses. Data on soil, water, and plants were necessary to determine the feasibility of utilizing the foodstuffs and living area of the contaminated atolls. Long term studies of the domestic animals obtained are being conducted at the NRDL, in order to obtain information on the possible acute and delayed hazard from internal radiation.

The main findings of the internal contamination study in the human beings and their environment are presented in this report. A detailed report on the nature and extent of the internal radiation hazard in human beings and animals, as well as the contamination of the environment will be presented in an addendum report.

5.2 PHYSICAL ENVIRONMENT STUDIES

A comparison of the food, water and soil samples from Rongelap, Utirik and Rongerik indicated a much higher level of contamination on Rongelap than on the other islands. The activity appeared to be distributed in the same manner on all the islands. In general, the contamination was associated with very fine particulate matter and was uniformly distributed throughout samples of earth, thatch, and grass. Significant amounts of beta activity ($1 \mu\text{c}$ per plant) as well as fissionable material ($1 \times 10^{-4} \mu\text{gm}$) were present on the external surfaces of plants at 42 days, but only small amounts of beta and no alpha activity were detected in the edible portions. Coconut tree sap, an important item in the native diet, was found to have beta activities of the level of $1 \mu\text{c/liter}$ on Rongelap at two months after detonation. Fish collected from the Rongelap lagoon were found to have very high levels of activity (0.3 to $3 \mu\text{c}$ per fish) as late as 120 days post-detonation.

The activity found in soil was determined to be associated mainly with fallout particles of relatively large diameter. Rongelap water samples from both cisterns and a well (Figs. 5.1 and 5.2) had activities of about $1 \mu\text{c/liter}$ at 30 days. Samples of thatch roofing exhibited the highest levels of activity in the physical environment. The presence of fissionable material was detected on thatch, grass and on plant food.

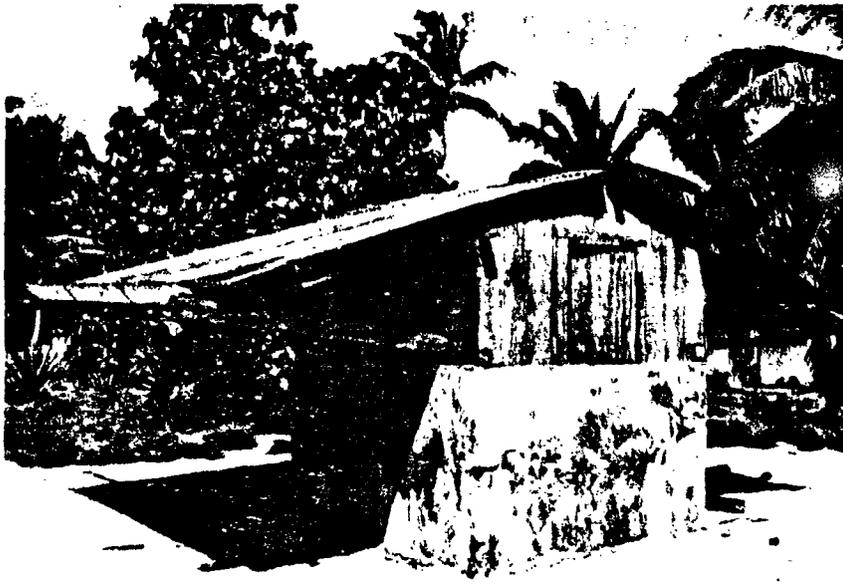


Fig. 5.1 Well, Rongelap

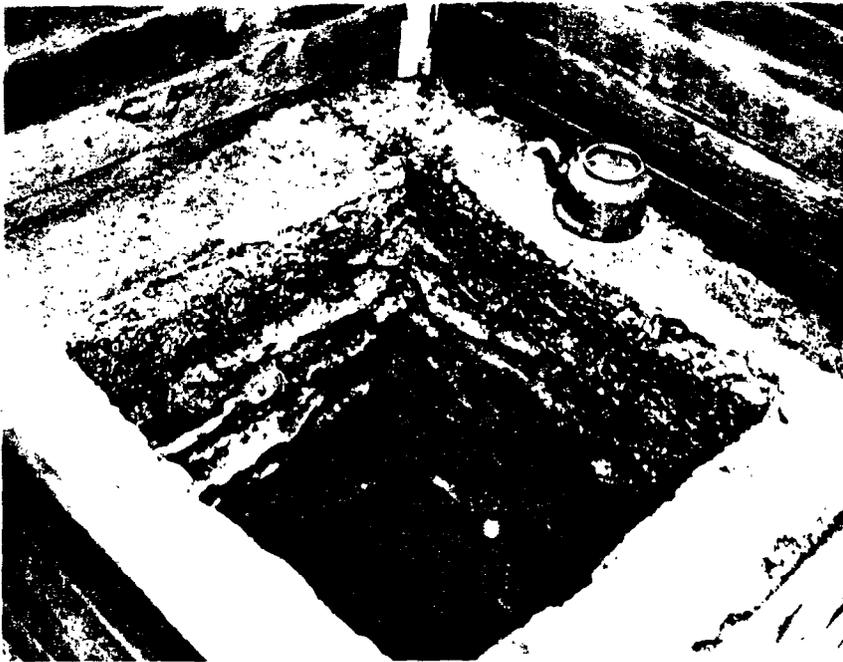


Fig. 5.2 Cistern, Rongelap

5.3 ANIMAL STUDIES

The studies on the Rongelap and Utrik animals included measurement of internal radioactive contamination and radiochemical analysis of excreta and tissues. The acute hematological and pathological effects of the exposure, as well as long term effects on these animals, are being studied. In addition, fertility and hatchability studies as well as radiochemical analysis of eggs laid by hens from Rongelap were performed.

These studies have provided information on the extent and nature of the internal contamination in the exposed human beings. For example, radioautographs of animal bones prepared by NRDL and Argonne National Laboratory⁶⁸ (Figs. 5.3 and 5.4) indicate the pattern of skeletal distribution of fission products, particularly the high concentrations of radioelements in the epiphyseal region of the long bones. Further, information on these animals (which received a much higher external radiation dose than was received by the native group and over 10 times the internal deposition) should be of considerable prognostic value for the human beings.

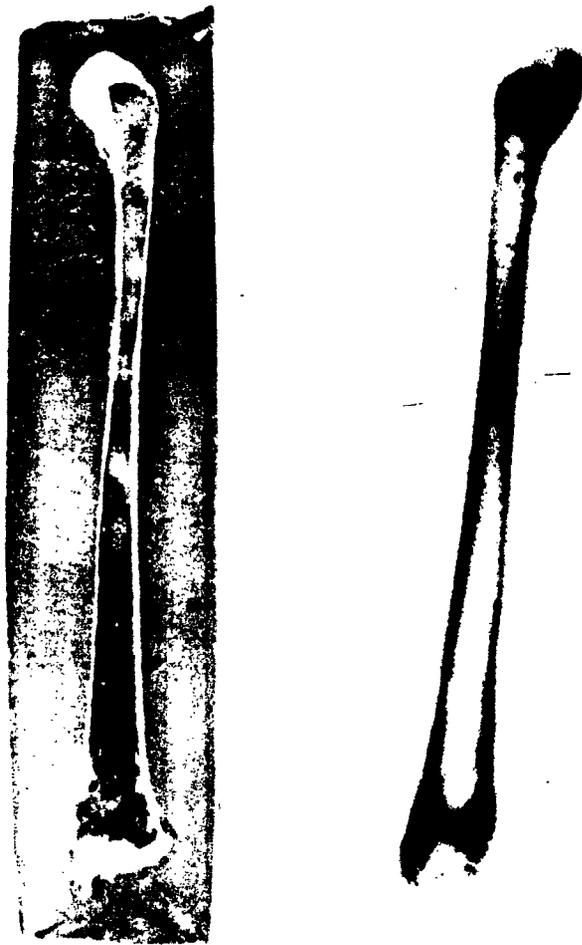


Fig. 5.3 Autoradiograph of Rongelap Chicken Tibia, $1\frac{1}{2} \times$, 21 hr Exposure. Animal Sacrificed 45 Days Post-exposure (Argonne National Laboratory Photo).



Fig. 5.4 Autoradiograph of Rongelap Pig Tibia, 42 hr Exposure. Animal Sacrificed 58 Days Post-exposure.

5.4 HUMAN STUDIES

5.4.1 Excretion

Gross beta activity measurements on the urine samples from Rongelap, Ailinginae, and Rongerik indicate significant contamination in the majority of samples. The variation in beta activity excreted 24 hours among individuals in any one group is quite large (Table 5.1). If the mean values of urine activity of the adults of the three groups six weeks after exposure are compared, it will be seen that the Rongelap group had the highest activity, 1208 d m 24 hours. The Ailinginae group had 553 d/m/24 hours, and the American group had 309 d, m 24 hours. A group of the Japanese fishermen exposed to fallout from the same detonation had a urine activity comparable to that of the Rongelap adult group.

The mean activity of the individuals under 15 years of age appears to be considerably lower than that of the adult group. An examination of the group collections analyzed by the various laboratories involved showed fairly consistent levels of activity for the individual from day to day. The variation which occurs is not considered to be excessively large for this type of study. The activity as function of time fell off rapidly due to the large component of Ba^{140} - La^{140} and other short lived radioisotopes. For example the Rongelap group had a mean activity of 1208 d/m/24 hours at 46 days and activity of 339 d/m/24 hours at 90 days. At six months post-exposure the Rongelap urine was found to contain a barely detectable amount of beta activity. The results of radiochemical studies of urine indicate that Sr^{88} , Ba^{140} , and the rare earth group apparently constitute 77 per cent of the total beta activity at 46 days. Strontium-89 contributes 40 per cent of the total beta activity, Ba^{140} — 11 per cent and the rare earth

Table 5.1 GROSS BETA ACTIVITY IN URINE OF RONGELAP GROUP
 (Urine Collected on 46th Post-exposure Day and Values Corrected for Decay
 Back to Collection Day)

Case No.	Total Volume 24 hr	Beta Activity d/m/24 hr	Case No.	Total Volume 24 hr	Beta Activity d-m 24 hr
Age 5 years			Age 16 years		
2	120	712	4	455	634
3	150	894	7	810	1700
5	155	313	9	355	201
23	40	223	10	980	549
33	260	0	11	450	1583
54	80	285	13	340	1677
69	455	301	14	780	2460
Mean	165	404	18	455	1670
			22	47	77
			30	960	438
			34	750	570
			37	480	792
			40	550	1450
			46	330	495
			49	425	0
			52	780	0
			55	320	1080
			56	700	3220
			57	550	1095
			58	750	2170
			60	810	580
			62	980	1985
			63	635	2260
			66	855	1715
			68	300	2010
			71	290	1450
			73	230	0
			78	965	52
			79	465	2038
			80	540	1353
			82	670	2140
Mean	439	758	Mean	581	1208

group—25 per cent (of which 11 per cent is La^{140} , in equilibrium with Ba^{140} at this time). Fissionable material was not found in significant amounts in any of the urine samples analyzed. Iodine-131 was found only in samples analyzed at early time intervals, due to its relatively short physical half life.

5.4.2 Estimate of Body Burden

For estimating the human body burden of the various fission products, a semi-empirical approach, involving extrapolation from animal data, was used. Two Rongelap pigs were killed after their average 24 hour urinary excretion of fission products was determined. All tissues were analyzed for total beta activity, as well as for Sr^{89} , Ba^{140} , and the rare earth content (Table 5.2). By applying the ratio of animal body burden/urine excretion activities to the Rongelap native urine samples analyzed at about the same post-detonation time, a total human body burden of 0.33 μc , for post-exposure day 82 was obtained. If this total human body burden

Table 5.2 RADIOCHEMICAL ANALYSIS OF TISSUES AND URINE OF RONGELAP PIGS

(All Values Corrected for Decay to Day of Sacrifice, the 82nd Post-exposure Day)

Sample	Both Activity d/m/total Sample			
	Gross Activity ($\times 10^{-3}$)	Sr ⁹⁰ ($\times 10^{-3}$)	Ba ¹⁴⁰ ($\times 10^{-3}$)	Total Rare Earth ($\times 10^{-3}$)
Pig #24 (25.8 kgm)				
Skeleton	8890	5660	660	1010
Liver	31	0.40	0.33	6.4
Colon & Contents	12	5.0	2.4	3.2
Lung (Alveolar)	1.5	0.22	0.20	0.8
Stomach	1.2	0.22	1.1	1.3
Intestine (Small)	2.3	0.62	0.50	0.51
Kidney	3.3	0.21	0.42	0.74
Remaining Tissues	690			
Total	9630	5667	665	1020
Urine Sample, 24 hr (5/27)	13	8.7	1.2	1.6
Urine Sample, 24 hr	9.6			
Average for days 82 to 88 post-exposure				
Pig #25 (22.7 kgm)				
Skeleton (total)	8600	5100	530	690
Liver	27	0.53	0.20	5.5
Colon & Contents	16	5	3.2	4.9
Lung (Alveolar)	1.1	0.26	0.23	0.33
Stomach	2.0	0.29	0.13	0.30
Intestine (Small)	2.6	0.83	0.88	0.88
Kidney	3.1	0.14	0.19	0.52
Remaining tissues	220			
Total	8870	5107	534	702
Urine Sample, 24 hr (5/27)	6.2	4.4	0.40	0.54
Urine Sample, 24 hr	8.1			
Average for days 82 to 88 post-exposure				
Average Per cent of Individual Radioelements		Sr ⁹⁰	Ba ¹⁴⁰	Total Rare Earth
Skeleton		62	6.8	9.7
Total Body		58	6.5	9.0
Urine (24 hr)		69	7.9	10.5

is separated into its components using the ratio of individual isotope activities in the urine or in the total body of the animals at this time, the following values for the Rongelap group, 82 days post-detonation were obtained:

$$\begin{aligned} \text{Sr}^{90} &= 0.19 \mu\text{c} \\ \text{Ba}^{140} &= 0.021 \mu\text{c} \\ \text{Rare Earth Group} &= 0.030 \mu\text{c} \\ \text{Others} &= 0.09 \mu\text{c} \end{aligned}$$

The excretion rate of Sr and Ba can be described empirically by a power function, $A_t = A_1 t^{-n}$ where n is 0.4 and A_t is the activity remaining in the body at time t (days). The exponent was derived from Cowan, ⁵⁹ Sr⁹⁰ human inhalation data, and it corresponds very closely

to the exponent found in the human radium data of Norris⁶⁰ and Lönner⁶¹. Using this formulation the individual skeletal body burdens extrapolated back to day one are as follows:

$Sr^{90} - 1.6 \mu c$
 $Ba^{140} - 2.7 \mu c$
 Fissionable material negative

Based on the Sr^{90} analysis, the Hunter-Ballou fission data⁶² and the retention data of Hamilton,⁶³ the following estimates of skeletal body burden in the Rongelap group on day 1 were made:

- a) rare earth group - 1.2 μc
- b) I^{131} - 6.4 mc

The integrated dose to the thyroid from I^{131} and the shorter lived iodine isotopes (I^{132} , I^{133} , and I^{135}), assuming a 20 per cent uptake/24 hours was 180 rep. The Ailinginae values were then approximately one-half and the American were one-fourth of these values.

On the basis of a radiochemical analysis on pooled urine samples collected from a cross section of the Rongelap and American populations, 16-18 March, Los Alamos reported⁶⁴ the following estimate of fissions associated with material inhaled and/or ingested by the Rongelap native group:

$I^{131} - 5.5 \times 10^{13}$ fissions
 $Sr^{90} - 1.2 \times 10^{13}$ fissions
 $Ba^{140} - 1.0 \times 10^{13}$ fissions

From these data, using certain assumptions as to the uptake and retention of these radioelements by the body, the following estimate of body burden at one day was derived:

	Rongelap (μc)	American (μc)
Sr^{90}	2.2	0.42
Ba^{140}	0.34	0.27
Ru^{103}	0.013	0.015
Ca^{46}	0.19	0.04
I^{131} , and short lived I^{131} equivalents	5.1 mc	1.9 mc
Fissionable material	0.016 μgm	

The initial body burden of I^{131} and short lived iodine isotopes, energetically equivalent to I^{131} is 5.1 mc. The estimated total integrated dose to the thyroid from the iodine isotopes assuming a 20 per cent uptake/24 hours and with corrections for decay of the very short lived isotopes was calculated to be 150 rep for the Rongelap natives and 50 rep for the Americans.

It can be seen that with widely different approaches to the estimation of the body burden, the results obtained are very similar with the exception of the Ba^{140} estimate.

An attempt to detect bone-fixed radioactive emitters by means of sensitive film badges taped below the knee over the epiphysis of the tibia on 40 exposed Marshallese yielded negative results.

5.4.3 Internal Radioactive Decontamination

Ethylene diamine tetracetic acid (EDTA) has been shown to be the most effective chemical agent to date for mobilizing fission products from the skeleton and for increasing their excretion rate.^{65,66,67} This chelating agent was therefore used in a decontamination therapy attempt on a group of seven Rongelap individuals having relatively large amounts of internally deposited radioelements. Oral administration of calcium EDTA, 1 gm/25 lb body weight daily

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for three days increased the over-all mean activity in the 24 hour urines of the treated people by a factor of 2.5 times compared with the pretreatment control excretion. Since the rate of excretion of activity at this time was about 0.1 per cent/24 hours, the observed increase in excretion did not significantly alter the total body burden. This study and previous animal work suggest that EDTA, perhaps administered I.V. and at early intervals, might have been of considerably greater value in increasing the excretion of some of the internally deposited fission products.

5.5 DISCUSSION

The fallout material probably entered the exposed people through both the inhalation and ingestion routes. The relatively large particle size indicates that the probability of inhalation of airborne contamination was small. This is based on the observation that in general only particles 0.1 to 3 μ in diameter reach the alveoli on inhalation and that larger particles are filtered out by the nose and upper respiratory passage and swallowed.⁴⁸ In addition, the lungs and air sacs of chickens autopsied in the period 24 to 100 days post-detonation showed low levels of contamination, while the gastrointestinal tract and its contents were relatively active. The high levels of activity found in the liver are also compatible with this hypothesis indicating ingestion as the route of entry. Autopsy findings on five pigs indicate substantially the same results.

In general the mean gross beta activities in the urine of Rongelap and Ailinginae groups were found to be roughly proportional to the calculated external dose and therefore to the concentrations of airborne fission products. However, a comparison of mean beta activities of the Ailinginae and American groups show the latter to have a somewhat lower amount of internal contamination than the former though the groups received approximately the same calculated external dose. The difference may well lie in the fact that the Ailinginae people continued to eat contaminated food and drink contaminated water up to the time of evacuation. The American personnel probably ingested less contaminated food. Their water was distilled and stored in closed containers which precluded the possibility of radioactive contamination. It is also possible that indoctrination of the American group as to the inhalation hazard and perhaps their more adequate shelter was responsible for the lower amount of internal contamination received. Radioanalysis of urine samples of the Japanese fishermen, who were exposed to the same fallout, indicated that they had received approximately the same amount of internal contamination as did the Rongelap adult group. The lower urine activities found in the Marshallese individuals under 15 years may indicate that with active bone growth occurring, the internally deposited fission products were more firmly fixed and therefore less readily excreted. It does not seem likely that the children were subjected to any less internal radioactive contamination than were the adults.

5.6 CONCLUSION

The degree of internal radiation hazard was too low to have contributed significantly to the acute radiation syndrome observed. Efforts to correlate individual body burdens with their clinical or hematological findings (platelet and white blood cell levels) were unsuccessful. Due to the ability of the skeletal system to concentrate fission products, any injury to the body from these internal emitters would most likely be determined by the radiation effect on the bone. The concentration and type of internal radioactive contaminants, however, minimize the probability of any significant long term effects from the internal radiation.

The possibility of synergistic effects from the combined exposure to external and near tolerance doses of internal radiation is also very slight. The occurrence of such a synergism has been demonstrated where animals were subjected to relatively high doses of internal emitters (2 μ c/gm) in addition to high external doses. The only suggestion of such a combined effect in this situation was from the hematological response observed in these exposed human beings, which did not follow precisely the time course characteristically seen in lower mammals after short exposure to external radiation.

CHAPTER 6

RECOMMENDATIONS

1. It is recommended that establishment of a medical team on a continuing basis, prepared to handle emergency situations connected with radiation, be considered. Qualified personnel should be designated by name; minimal clinical and research procedures considered essential should be established and necessary equipment should be stockpiled.

2. It is recommended that steps be taken to obtain additional data in the laboratory in the following categories:

(a) The hematological response of large animals, and preferably man, exposed to radiation producing the depth-dose curve seen with fallout gamma radiation from a plane field. Data well beyond the usual 30-day period, and preferably for at least a year after exposure, are necessary.

(b) The response of human skin to beta radiation under conditions of different beta energies, source sizes, and source geometries.

REFERENCES

1. Henriques, J., *J. Lab. Invest.*, Vol. 1, No. 2: 153, 1952.
2. Glendenin, L. E., *Nucleonics*, Vol. 2, No. 1: 12, 1948.
3. Warren, Shields, *Arch. of Path.* 35: 304, 1953.
4. Knowlton, N. P., Leifer, E., Hogness, T. R., Hempleman, L. H., Blaney, L. F., Gill, D. C., Oaks, W. R., and Shafer, C. L., *J.A.M.A.*, Vol. 141, No. 4: 239, 1949.
5. Comar, C. L., Hansard, S. L., Hobbs, C. S., Hood, S. L., Paysinger, J., Plumlee, M. P., Sikes, D., West, J. L., UT-AEC-1, 1953.
6. Lushbaugh, C. E., Spalding, J. F., and Hale, D. B., Report on sheep losses adjacent to Nevada Proving Grounds, AEC report Jan. 6, 1954.
7. Robbins, L. L., Aub, J. C., Cope, O., Cogan, D. G., Langohr, J. L., Cloud, R. W., and Merrill, O. E., *Radiology* 46: 1, 1946.
8. Crawford, S., *Arch. of Dermat. and Syph.* 27: 579, 1933.
9. Low-Beer, B.V.A., *Radiology* 47: 213, 1946.
10. Wirth, J. E., and Raper, J. R., Biological effects of external beta radiation, Zirkle, R. E., Chapter 12, McGraw-Hill Book Co., Inc., First Edition, New York, 1951.
11. Morton, J. J., Lewis, J. J., Shimomura, S., Fujii, T., Sears, M., and Tsuchitori, K., Atomic Bomb Casualty Commission, Preliminary Report, March 26, 1954.
12. Morton, J. J., Lewis, J. J., and Shimomura, S., Atomic Bomb Casualty Commission, Supplementary Report, Apr. 19, 1954.
13. MacKee, G. M., Cipollaro, A. C., and Montgomery, H. M., X-Ray and radium treatment of diseases of the skin, Fourth Edition, Lea and Febiger, Philadelphia, 1947.
14. Wilhelmy, E., *Strahlentherapie*, 55: 498, 1936.
15. Harris, M., Leddy, E. T., and Sheard, C., *Radiology* 19: 233, 1932.
16. Halkin, H., *Arch. of Dermat. and Syph.*, 65: 201, 1903.
17. Goldberg, S. V., Cited in *Das Radium in der Biologie und Medizin* by London, J.S., Leipzig, 1911, page 56.
18. Bloom, W., and Bloom, M. A., *Radiation Biology*, Hollander, Vol. I, Part II, pg. 1119, McGraw-Hill Book Co., Inc., New York, 1954.
19. Warren, Shields, *Physio. Rev.*, 24: 225, 1944.
20. Nödl, F., *Strahlentherapie*, 92: 576, 1953.
21. Walbach, S. W., *J. of Med. Res.*, 16: 415, 1909.
22. Raper, J. R., Zirkle, R. E., and Barnes, K. K., Atomic Energy Commission Report MDDC-439, Feb. 12, 1946.
23. Snider, R. S., *Histopathology of Irradiation*, Bloom, W., First Edition, Chapter 4, McGraw-Hill Book Co., Inc., New York, 1948.
24. Raper, J. R., Henshaw, P. S., and Snider, R. S., Atomic Energy Commission Report MDDC-580, June 6, 1946.
25. Raper, J. R., and Barnes, U. K., Biological effects of external beta radiation, Zirkle, R. E., Chapter 4, First Edition, New York, 1951.
26. Raper, J. R., Henshaw, P. S., and Snider, R. S., Atomic Energy Commission Report MDDC-578, April 25, 1946.

27. Henshaw, P. S., Snider, R. S., and Riley, E. F., *Radiology* 52: 401, 1949.
28. Lushbaugh, C. C., Stover, J. B., and Hale, D. B., *Cancer*, Vol. 6, No. 4: 671, 1953.
29. Trum, B. F., Personal communication of work in progress.
30. Moritz, A. R., and Henriques, F. W., *J. Lab. Invest.*, Vol. 1, No. 2: 167, 1952.
31. Raper, J. R., Wirth, J. E., and Barnes, K. K., *Biological effects of external beta radiation*, Zirkle, R. E., Chapter 2, First Edition, New York, 1951.
32. Sulzberger, M. B., Baer, R. L., and Borota, A., *Atomic Energy Commission Report AECU-1616*. No date.
33. Dorn, H. F., *Illness from cancer in the United States*, Public Health Report, Reprint No. 2537, Govt. Printing Office, Jan. 1944.
34. Sutton, R. L., Jr., *J.A.M.A.*, Vol. 150, No. 3: 210, 1952.
35. Brecher, G., and Cronkite, E. P., *Journal of Applied Physiology*, 3: 365, 1950.
36. Wintrobe, M. M., *Clinical Hematology*, Lea and Febiger, Phil., 1951.
37. *Standard values in Blood*, Air Force Technical Report No. 6039, 1951.
38. Walsh, J. E., "Applications of some significant tests for the median which are valid under very general conditions," *Jour. Amer. Stat. Assoc.*, Sept. Vol. 44 (1949), pp 342-355.
39. Rosenbaum, S., "Tables for a nonparametric test of location," *Annals of Math. Stat.*, March, Vol. 25 (1954) pp 146-150.
40. Rosenbaum, S., "Tables for a nonparametric test of dispersions," *Annals of Math. Stat.*, December, Vol. 24 (1953) pp 663-668.
41. Dixon, W. J., "The statistical sign test," *Jour. Amer. Stat. Assoc.*, December, Vol. 41, (1946) pp 557-566.
42. Quenouille, M. H., Chapter 3, "Ordering Test," Academic Press Inc., Publishers, New York, 1952.
43. White, C., "The use of ranks in a test of significance for comparing two treatments," *Biometrics*, 8, 33 (1952).
44. Moses, L. E., *Nonparametric statistics for psychological research*, *Psych. Bull.*, 49: 122, March 1952.
45. Minot, G. R., and Spurling, R. G., *The effect on the blood of irradiation, especially short wave length Roentgen Ray Therapy*, *Am. J. Med. Sci.*, 168: 215, 1924.
46. Oughterson, A. W., Leroy, G. V., Liebow, A. A., Hammond, E. C., Barrett, H. L., Rosenbaum, J. D., and Schneider, B. A., *Medical effects of atomic bombs. The Report of the Joint Commission for the Investigation of the Effects of the Atomic Bomb in Japan. Vols. III and V. Atomic Energy Commission Documents, NP3038 and NP3040*, 1951.
47. LeRoy, G. V., *Hematology of Atomic Bomb Casualties*. *Arch. Internal Med.*, 86: 691, 1950.
48. Dunlap, C. E., III, *Effects of radiation on the blood and hematopoietic tissues, including the spleen, the thymus and the lymph nodes*, in Warren, S.: *Effects of radiation on normal tissues*. *Arch. Path.*, 34: 562, 1942.
49. Hempelmann, L. H., Lisco, H., and Hoffman, J. G., *The acute radiation syndrome: A study of nine cases and a review of the problem*. *Ann. Internal Med.*, 36: 279, 1952.
50. Hasterlik, R. J., *Clinical report of four individuals accidentally exposed to gamma radiation and neutrons*. Argonne National Laboratory, Jan. 1953.
51. Cronkite, E. P., *The protective effect of granulocytes in radiation injury*. To be published.
52. Snell, Fred M., and Neel, James V., *Hematologic studies in Hiroshima and a control city two years after the atomic bombing*. *Arch. Internal Med.*, 84: 569, 1949.
53. Yamasoiva, Y., *Hematologic studies of irradiated survivors in Hiroshima, Japan*. *Arch. Internal Med.*, 91: 310, 1953
54. Jacobson, L. O., E. R. Marks and E. Lorenz. *Hematological effects of ionizing radiations*, *Radiology*, 52: 371, 1949.
55. Cronkite, E. P., and G. Brecher, *Defects in hemostasis produced by whole body irradiation*. Josiah Macy, Jr. Foundation, *Transactions of the fifth conference on blood coagulation*, New York, 1952.
56. Carter, R. E., Cronkite, E. P., and Bond, V. P. Unpublished data.
57. Brecher, G., Schneiderman, B. S., and Cronkite, E. P., *The reproducibility and constancy of the platelet count*. *Am. J. Clin. Path.*, 23: 15, 1953.



58. Norris, W. P., L. A. Woodruff, P. F. Gustafson and A. M. Brues, Report on biological specimens collected on Rongelap Atoll in March 1954, ANL-HDY 731.
59. Cowan, F. P., L. B. Farabee and R. A. Love, Health physics and medical aspects of a strontium-90 inhalation incident, Am. J. Roent., Rad. Ther. & Nuc. Med., 67: 805, 1952.
60. Norris, W. P., T. W. Speckman and P. F. Gustafson, Studies on the metabolism of a radium in humans. Unpublished.
61. Looney, W. B., Late effects (25-40 years) of the early medical and industrial use of radioactive materials, Presented in part at the 35th annual session of American College of Surgeons, Chicago, Illinois, April 9, 1954.
62. 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, USNRDL-65, April 1949.
63. Hamilton, J. G., The metabolic properties of the fission products and actinide elements, Rev. Mod. Physics, 20: 718, 1948.
64. Harris, P. S., Personal communication.
65. Foreman, H., and J. G. Hamilton, The use of chelating agents for accelerating excretion of radioelements, AECD-3247, 1951.
66. Cohn, S. H., J. K. Gong and M. C. Fishler, Studies on EDTA treatment of internal radioactive contamination, Nucleonics, 11: 56, 1953.
67. Gong, J. K., W. L. Milne and S. H. Cohn, Studies on the treatment of internal radioactive contamination. IV. Effect of zirconium citrate and EDTA on the distribution and excretion of a mixture of long-lived fission products, USNRDL-426, Dec. 1953.
68. Patty, F. A., Indust., Hyg. and Toxicol., Interscience Publishers, Inc., N. Y., 1948.

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