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MEDICAL SURVEY OF RONGELAP PEOPLE SEVEN YEARS AFTER EXPOSURE TO FALLOUT

ROBERT A. CONARD, M.D., H. EUGENE MACDONALD, M.D., LEO M. MEYER, M.D., STANTON COHN, PH.D., WATARU W. SUTOW, M.D., DAVID KARNOFSKY, M.D., A.A. JAFFE, D.D.S., AND EZRA RIKLON, PRACTITIONER

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The accumulation of data from these surveys is becoming increasingly voluminous. Since conditions have not been favorable for performance of extensive statistical analyses or use of electronic computing procedures to store and manipulate the data, the annual survey reports published by this Laboratory are made as complete as possible. This report, therefore, includes a considerable amount of raw data, much of it in appendices, so that others may have an opportunity to make further calculations if desired.

Summary of Past Findings

Reports have been published on the findings of surveys made at the following times after exposure: initial examination,[1] 6 months,[2] 1 year,[3] 2 years,[4] 3 years,[5] 4 years,[6] and 5 and 6 years.[7] The following is a brief summary of the findings previously reported.

During the first 24 to 48 hr after exposure, about ⅔ of the Rongelap people experienced anorexia and nausea. A few vomited and had diarrhea. Many also experienced itching and burning of the skin, and a few complained of lachrymation and burning of the eyes. Following this, the people remained asymptomatic until about 2 weeks after the accident, when cutaneous lesions and loss of hair developed, due largely to beta irradiation of the skin. It was apparent when the people were first examined, a few days after exposure, that the lymphocytes were considerably depressed and that significant doses of radiation had probably been received. In addition to the whole-body dose of radiation and the beta irradiation of the skin, radiochemical analyses of the urine showed that measurable amounts of radioactive material had also been absorbed internally. The effects of the radiation can best be summarized under three headings according to the mode of exposure: penetrating irradiation, skin irradiation, and internal irradiation.

PENETRATING IRRADIATION

The changes in the peripheral blood of the more heavily exposed Rongelap people who received 175 r will be reviewed in the section on hematological examinations (see Figures 34, 39, and 43, and Appendices 1 and 2). The changes in the Ailingnae and Utirik groups were similar but less marked. Certain unexplained fluctuations have occurred from year to year in the peripheral blood levels of the comparison populations as well as of the exposed groups. The mean *leukocyte* level of the exposed group showed a marked decrease at the time of the 1960 survey (no unexposed people were examined). Depression of the peripheral blood elements as represented by mean population levels occurred as follows.

Lymphocytes fell promptly and by the third day were about 55% of the control values in adults, and slightly lower in children. There was only slight recovery after six months. At 2 years, although further recovery was evident, the mean values of these cells were still found to be below the comparison population levels (75 to 80%). At 3 years the mean lymphocyte counts were slightly below those of the comparison population. At 4 years the mean level appeared to be about the same as that of the comparison population, but many counts remained lower. However, at 5 years, *lymphocyte* levels appeared for the first time to equal those of the unexposed group, except that analyses showed more counts below the average of the unexposed group.

Neutrophil levels fluctuated considerably during the first few weeks but fell gradually to a low of about 50% of comparison population levels by the 6th week after exposure. Slow recovery ensued, but at 6 months they were still slightly below the unexposed levels. However, by 1 year post exposure they had returned to the level of the comparison population and have remained so, with the possible exception of those of children <12 years old, which generally have been lower than those of the unexposed children of comparable age.

Platelets fell to about 30% of the unexposed values by the 4th week. By 6 months they had reached 75% of the controls; at 1 year the mean platelet count was still below that of the control population but higher than at the 6-month survey. Although further increases were apparent at the 2-, 3-, and 4-year examinations, the levels were still below those of the comparison population. At 5 years post exposure, exposed people still had mean *platelet* levels 10 to 15% below those of the unexposed group.

Changes in hematocrit were not remarkable in any of the groups during the period of maximum depression of leukocytes. However, it appeared

MEDICAL SURVEY OF RONGELAP PEOPLE SEVEN YEARS AFTER EXPOSURE TO FALLOUT

Introduction

The results of a medical survey of the people of Rongelap in the Marshall Islands, carried out in March 1961 at 7 years after the accident, are presented in this report. These people had been accidentally exposed to fallout radiation following a detonation of a high yield thermonuclear device during experiments at Bikini in the Pacific Proving Grounds in March 1954. An unpredicted shift in winds caused a deposition of significant amounts of fallout on four inhabited Marshall Islands to the east of Bikini (see Figure 1) and also on 23 Japanese fishermen aboard their fishing vessel, the *Lucky Dragon*. Of the inhabitants of the island of Rongelap, 105 nautical miles away from the detonation, 64 received the largest fallout exposure: an estimated dose of 175 r of whole-body gamma radiation, contamination of the skin sufficient to result in beta burns, and slight internal absorption of radioactive materials through inhalation and ingestion. Another 18 Rongelap people away on a nearby island (Ailingnae), where less fallout occurred, received only an external gamma dose of about 69 r. There were 28 American servicemen on the island of Rongerik further to the east who received about the same amount of radiation as did the Rongelap people on Ailingnae. Lastly, 157 Marshallese on Utirik Island, about 200 miles further east, received about an estimated 14 r of whole-body radiation. The fallout was not visible on this island and no skin effects developed.

The exposed people were evacuated from these islands by plane and ship about two days after the accident and taken to Kwajalein Naval Base about 150 miles to the south, where they received extensive examinations for the following three months. In view of the generally negative findings on the American servicemen, they were later returned to their duty stations. The Utirik people were also allowed to return to their home island, where radioactive contamination was slight enough to allow safe habitation. Because Rongelap Atoll was considered to be too highly contaminated, a temporary village was constructed for the Rongelap people on Majuro Atoll several hundred miles to the south, where they lived for the following 3½ years and were examined at yearly intervals by a special medical team. In July 1957, after careful evaluation of the radioactive contamination situation, Rongelap Island was considered safe for habitation. A new village was constructed, and the Rongelap people were moved there by Navy ship. The annual medical surveys have since been carried out on Rongelap Island.

A group of more than 100 Rongelap people, who were relatives of the exposed people but had been away from the island at the time of the accident, moved back with the Rongelap people to their home island and have served as an ideal comparison population for the studies. Following the initial survey of the Utirik people on Kwajalein in 1954, a repeat survey was carried out in March 1957. In addition, during the past survey, as in the previous surveys, a visit was made to Majuro Atoll to examine a group of children who represent part of the control group used for the growth and development studies of the exposed children.

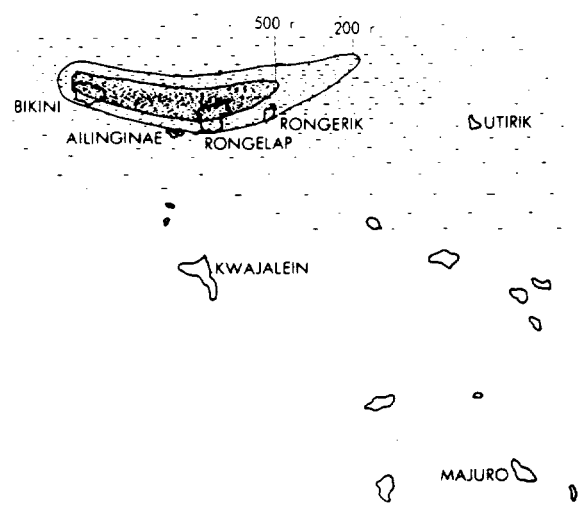


Figure 1. Map of fallout area (March 1, 1954), Marshall Islands.

in the exposed group. It is not known whether this finding is of any significance in relation to their radiation exposure. Slit-lamp observations showed no opacities of the lens characteristic of radiation exposure. As a whole, visual and accommodation levels in the Marshallese appeared to be above the average in the U.S. population.

Dental surveys showed no significant differences in either caries rate or incidence of peridontal disease between exposed and unexposed groups. The poor oral hygiene generally observed in the Marshallese had its usual results, namely, high caries rate in teen-age children, severe peridontal lesions in adults (heavy calculus and loss of alveolar bone), and edentulous mouths in the aged. Radiation exposure did not appear to have affected developing dentition in the exposed children.

Late effects of radiation. Various parameters usually associated with *aging* were measured or estimated on a 0 to 4+ scale (skin looseness, elasticity, and senile changes; greying of the hair and balding; accommodation, visual acuity, and arcus senilis; hearing; cariovascular changes including blood pressure and degrees of peripheral and retinal arteriosclerosis; neuromuscular function; and hand strength). Comparison of these measurements in exposed and unexposed individuals of the same age groups showed no apparent differences. A biological age score was calculated for individuals and groups by use of an average percentage score. *Life shortening* effects of radiation have not been apparent. As noted, the mortality rate was about the same in the exposed as in the unexposed people.

The one case of *cancer* that developed in the exposed group occurred at 5 years after exposure, too soon, it is believed, to bear any particular relation to radiation exposure. *Leukemia* surveys including physical findings, studies of white cell counts and types, alkaline phosphatase staining, and basophil counts of 4000 white cells showed no evidence of leukemia or leukemic tendency. One child in the irradiated group had 3% basophils but no other positive findings. The *cardiovascular* and *arthritis* surveys, as well as the general results of the physical examinations, have not shown any apparent increased incidence of *degenerative diseases* in the exposed people. No radiation-induced *cataracts* have been observed in any of the exposed people.

Genetic effects have not been specifically studied because of the small number of people involved. No apparent radiation-induced genetic changes have been detected on routine physical examination in the first-generation children of exposed parents. If the suggestive evidence of increased miscarriages and stillbirths in the exposed women is true, this may represent a genetic effect of exposure.

BETA IRRADIATION OF THE SKIN

It was impossible to get an accurate estimate of the radiation dose to the skin. Beta burns of the skin and epilation appeared about 2 weeks after exposure, largely on parts of the body not covered by clothing. About 90% of the people had these burns, and a smaller number developed spotty epilation of the scalp. Most of the lesions were superficial; they exhibited pigmentation and dry, scaly desquamation, and were associated with little pain. Rapid healing and repigmentation followed. Some lesions were deeper, showed wet desquamation, and were more painful. A few burns became secondarily infected and had to be treated with antibiotics. Repigmentation of the lesions gradually took place in most instances, and the skin appeared normal within a few weeks. However, in about 15% of the people, deeper lesions, particularly noted on the dorsum of the feet, continued to show lack of repigmentation with varying degrees of scarring and atrophy of the skin. By 6 years the only residual effects of beta radiation of the skin were seen in 10 cases which showed varying degrees of pigment aberrations, scarring, and atrophy at the site of the former burns. Numerous histopathological studies have been made,[1,4,5] and the changes found have been consistent with radiation damage. At no time have changes been observed either grossly or microscopically indicative of malignant or premalignant change. Spotty epilation on the heads was short lived, regrowth of hair occurring about 3 months after exposure and complete regrowth of normal hair by six months. No further evidence of epilation has been seen.

An interesting observation was the appearance of a bluish-brown pigmentation of the semilunar areas of the fingernails and toenails in about 90% of the people, beginning about 3 weeks after exposure. By 6 months, this pigmentation had largely grown out with the nail and had disappeared in most cases. The cause of this phenomenon has not been explained.

that in 1959 the mean *erythrocyte* levels were slightly lower in the exposed people. These blood elements in the Ailingnae group also showed some slight depression below the unexposed levels but not quite so marked as seen in the Rongelap exposed group. A general anemic tendency was noted in the Marshallese, both exposed and unexposed. *Price-Jones curves*, on the average, showed a slight microcytic tendency. *Serum iron* levels were generally normal. The fact that some of the blood elements in the exposed group have not yet returned to the levels in the unexposed group raises the possibility that a residual radiation effect on the bone marrow persists, but other, not immediately apparent, factors may be involved.

Reticulocyte counts have been about the same in the exposed and unexposed people.

Bone marrow examinations on a number of exposed individuals at 6 months and 3 years post exposure showed no abnormalities or deficiencies of cellular elements.

Clinical examinations revealed no disease processes or symptoms which could be attributed to radiation effects, aside from skin lesions, loss of hair, and early symptoms. No specific therapy was given. Epidemics of chicken pox and measles occurred. The diseases encountered were no more severe or frequent in the irradiated group than in the unexposed group, even during the period of greatest depression of peripheral blood elements.

At 3 years post exposure the immune response to primary and secondary tetanus antitoxin was tested and found not to be significantly different in the exposed compared to the unexposed populations.

Four persons in the exposed population died of disease: (1) a 46-year-old man with a hypertensive heart disease which had been present at the time of exposure, who died 2 years after the accident; (2) a 78-year-old man who died, 3 years after exposure, of coronary heart disease complicating diabetes; (3) a 36-year-old man who died of acute varicella, 4 years after exposure, who had received only 69 r, having been on Ailingnae at the time of the fallout; and (4) a 60-year-old woman who died of a cancer of the ovary at 5 years after exposure. There was no apparent relationship between any of these deaths and radiation exposure. Four deaths have occurred in the comparison population. The four deaths that have occurred in the exposed people since exposure represent a *mortality rate* of 8.1 per 1000 population, compared with 8.3 for the comparison population and 6.8 for the Marshall Islands as a whole.

Certain findings were possibly related to the radiation exposure, such as loss in weight of several pounds in most of the people during the first several months after exposure and suggestive evidence of slight lag in growth and development of the children based on studies of height, weight, and bone development (but inconclusive pending verification of more exact ages of some of the children).

In connection with *growth and development studies*, a project on the verification of accuracy of ages of the children has not been completed and, therefore, the suggestive evidence previously presented of possible lag in statural growth in the exposed children must await confirmation. It was noted, however, that in the 6-year chronological age group, three boys and one girl out of five boys and two girls in the exposed group exhibited significantly retarded skeletal maturation as judged by x-ray examination. The birth dates of these children seemed to be fairly well established.

It was difficult to evaluate the effects on fertility. However, a review of the *birth rate* of the exposed group over the past 6 years seems to indicate no noticeable effect of their exposure on fertility. The 24 births represent a rate of 48 per 1000 population, compared with 37.3 for the Marshall Islands (1957). The 20 births over a 3-year period for the comparison population represent a rate of 62 per 1000 population. A somewhat greater incidence of *miscarriages and stillbirths* has been noted in the exposed women, but because of the paucity of vital statistics in the Marshallese and the small number of people involved, the data are not readily amenable to statistical analysis.

A *cardiovascular survey* of the adults (1959) showed no outstanding differences between the exposed and unexposed groups. The people appeared to have less hypertension on the whole than is noted in people in the continental United States.

An *arthritis survey* (1959) showed no great differences between the exposed and the unexposed people, and about the same incidence as is seen in American populations.

Ophthalmological surveys showed no remarkable differences between the exposed and unexposed groups except possibly a slightly greater number of cases of pterygii, pingueculae, and corneal scars

INTERNAL IRRADIATION

Radiochemical analyses of numerous urine samples of the exposed population showed internal absorption of radioactive materials, probably brought about largely through eating and drinking contaminated food and water and to a lesser extent through inhalation. During the first few days when the body levels were at their highest, the maximum permissible concentrations were approached or slightly exceeded only in the case of strontium-89 and the isotopes of iodine. The concentrations were believed to be too low to result in any serious effects. Body levels fell rapidly, so that by 2 and 3 years post exposure, they were far below the accepted maximum permissible level; by 6 months activity in the urine was barely detectable.

In 1957 analyses of bone samples on one of the men who died showed 3.7 strontium-90 units/g calcium. Beginning in 1957, gamma spectroscopy by use of a low-level counting chamber was added to the techniques of radiochemical analysis. The return of the Rongelapese to their home island (which after careful survey was considered safe for habitation, despite a persisting low level of radioactive contamination) was reflected in a rise in their body burdens and increased urinary excretion of certain radionuclides. During the years since the original contaminating event, additional weapons tests held in the area have contributed to the fission products in the environment. Since the diet includes a variety of imported foods, the people are not living in a "closed" environment, and therefore may not be rapidly approaching equilibrium with the environmental fission products, as might be expected under other circumstances.

Body burdens of gamma-emitting fission products (such as Cs^{137} and Zn^{65}) were measured in a whole-body counter and checked by radiochemical analysis of urine specimens. Body burdens of Sr^{90} were estimated from urinary excretion as determined by radiochemical analyses. Both the external dose measurements on Rongelap Island and the levels of radioactive isotopes in the food on the island indicated that some increase in Cs^{137}, Zn^{65}, and Sr^{90} body burdens was to be expected when the people returned there in 1957. The Cs^{137} body burden in 1958 was about 0.68 μC, about 60 times as great as in 1957, and the urinary Cs^{137} level rose by a factor of 140; the mean body burden for 1959 was 0.57 μC. The mean body burden of Zn^{65} estimated from whole-body counting data was, in 1958, after the return to Rongelap, 0.36 μC, 8 times as high as in 1957, and 0.44 μC in 1959. Thus, whereas the Cs^{137} levels appear to have reached a maximum and actually to have dropped in 1959, the Zn^{65} levels have shown a continued increase which is probably related to the long biological half-life of the latter. The Sr^{90} level in 1958 estimated from excretion data was 2 mμC, about 20 times as high as in 1957 before the return to Rongelap. The estimated body burden in 1959 increased to 6.0 mμC, about 20% of the estimated ultimate equilibrium value. Little of the body burden of the exposed group is apparently due to their initial exposure, since at present there is little difference between the levels of the exposed and unexposed populations living on Rongelap Island. When these three isotopes have reached their estimated equilibrium values, the body burdens will still be of small significance in terms of radiation hazard.

OTHER STUDIES

Studies of genetically inherited characteristics. Blood grouping studies in the Marshallese showed a relatively high B gene frequency, a high N gene frequency, an extremely high R^1 gene frequency, and total absence of Kell and Diego factors. These characteristics differ from those of Polynesians and suggest relationship with Southeast Asians and Indonesians. *Haptoglobin studies* showed the frequency of the Hp^1 gene to be higher than in European populations thus far tested and consistent with populations living near the equator. The distribution of haptoglobin types showed the population to be relatively homogeneous. *Transferrins* in all sera were type CC, the common European type. *β-Amino-iso-butyric acid* urinary levels showed the Marshallese to be the highest excretors of this acid of any population thus far reported. Levels in the exposed group were about the same as in the unexposed group, and no correlation was found with body burden level of radionuclides; this indicates that there is probably no correlation with radiation exposure. *Hemoglobin types* were considered normal. *Sickling tests* showed no sickling tendency in any of the people. *Glucose-6-phosphate dehydrogenase* of the red cells appeared to be deficient in the Marshallese. Considerable cau-

tion must be exercised in evaluating the results of these studies on genetically inherited characteristics because of the small number of samples tested. The data do seem to indicate relative homogeneity of the population and closest kinship with people of Southeast Asia. These data also may be useful as a base line should genetic changes appear in later generations, possibly related to radiation exposure.

Results of other laboratory studies included the following: *Serum protein* levels were generally on the high side of normal; electrophoretic patterns showed the increase in proteins was largely due to an increase in the gamma globulin fraction. The reason for this is not apparent. Numerous chronic infections may be an explanation.

Sodium levels in the urine and food indicated about the same consumption of NaCl as in Americans. The generally lower incidence of hypertension in the Marshallese might be related to the fact that the former native diet was probably lower in salt content than the present, more westernized diet. It will be interesting to see whether the incidence of hypertension will later increase.

Serum cholesterol levels (1957, 1959) were somewhat lower in the exposed population than in the comparison or Utirik populations, but were in the low normal range. No abnorally low readings were noted.

Serum creatinine levels (1957) were in the normal range with no abnormal levels noted.

Serum vitamin B_{12} concentrations (1958, 1959) were generally significantly higher than American levels. The possibility of contamination of the samples with bacteria producing vitamin B_{12} must be considered, since myeloproliferative and liver diseases were not seen.

Serum protein bound iodine levels (1957, 1959) were generally slightly elevated. Evidence for thyroid dysfunction was not apparent in the people.

Glucosuria and elevated blood sugar were found in 4 unexposed individuals, which indicated a rather high incidence of diabetes.

A survey for *intestinal parasites* (1958) showed 75% of the people to be infected with various types. For the three major pathogens found, the overall infection rates were, for *Entamoeba histolytica*, 18.2%; for hookworm, 5.5%; and for *Trichuris trichiura*, 34.3%.

Eosinophilia >5% has consistently been noted in about half the people. The fact that half the cases with eosinophilia showed no helminthic infections at all suggests that other factors besides parasitic infections must be responsible. The eosinophilia may be related to chronic fungus and other infections, particularly of the skin.

Complement fixation studies for parainfluenza 1, 2, and 3, respiratory syncitial, psittacosis, and Q fever showed antibodies to all groups of viruses except that for Asian influenza, which probably had not yet seriously involved the people of the Marshall Islands. The antibody titers appeared to be somewhat lower in the exposed people.

DIFFICULTIES ASSOCIATED WITH THE EXAMINATIONS

As mentioned in previous reports, several difficulties were associated with carrying out the examinations as well as interpreting the findings.

1. The language barrier made examinations difficult, since very little English is spoken by the Marshallese. However, there were sufficient English-speaking Marshallese to assist the medical team in most instances.

2. The lack of vital statistics or demographic data on the Marshallese imposed a serious difficulty in interpretation and evaluation of the medical data. Records of births, deaths, etc., have been made by the health aides or magistrates of the villages and supposedly forwarded to the district administrator; however, such records have been incomplete or lost in most instances, and vital statistics are therefore inadequate. Trust Territory officials are now attempting to assemble such data.

3. There is uncertainty on the part of some of the Marshallese as to their exact ages, particularly among the older group. This imposes certain difficulties in interpreting some of the studies to be outlined.

COMPARISON POPULATIONS

During the first 2 years, two separate groups of Marshallese people were used for comparison, each of comparable size to the exposed Rongelap group and matched for age and sex. However, this population was found to be unstable, with a large attrition rate over the 2 years, which made it unsatisfactory. At the time of the 3-year survey, it was found that during the preceding 12 months the Rongelap population at Majuro Atoll had

Figure 3. Rongelap Island, 1961.

doubled because of the influx of relatives who had come back from other islands to live with them. These people had been away from Rongelap Atoll at the time of the accidental exposure. This group matched reasonably well for age and sex and was of comparable size. Since the return of the people to Rongelap, however, this group has about doubled in size.

Since the people are of the same stock genetically, they are uniquely appropriate to serve as a comparison population and have, therefore, been used since 1957.

1961 Survey – Organization and Procedures

The medical team consisted of six medical specialists, five from the United States and one from the Trust Territory; one dentist from the Trust Territory; one Marshallese practitioner; and ten technicians, five from the United States and five from the Trust Territory (Figure 2).

The medical equipment had been sent out to the Islands prior to the team's arrival, and preliminary preparations had been made for logistic support of the operation by the Navy Pacific Missile Range group and the Holmes and Narver contractors at Eniwetok.

Before the survey at Rongelap, the team visited Kwajalein and Majuro for several days to carry out examinations on a number of Rongelap people who had moved to these islands.

The Trust Territory provided a cargo ship, the M/V *Roque* (Figure 5), to transport the team and to serve as a base of operations. In mid-March the ship proceeded from Majuro to Eniwetok, where the medical equipment and the 21-ton steel room for whole-body gamma measurements (Figure 6), which was stored there, were loaded on the ship. The onloading of the latter was a difficult and exacting procedure, but was carried out remarkably smoothly by the Holmes and Narver personnel. The steel room and a wooden room to house the electronic equipment were placed in the cargo hold of the ship, and wooden stairs were installed for access into the hold.

On completion of loading the ship, refueling, and taking on fresh water, the team boarded the ship and proceeded to Rongelap. The one-day trip was quite rough, since the ship was heading into the strong prevailing trade winds, but the steel room and equipment remained secure. The ship dropped anchor about 1000 yards off shore at Rongelap village.

On arrival, an outrigger canoe came alongside the ship with the Magistrate of the village, who came aboard. The objectives of our visit to Rongelap were discussed with him, and it was considered advisable to hold a village meeting so that the proposed examinations could be explained to the people and any questions they might have could be answered.

During the village meeting, held in the Council House, the medical examinations to be conducted and the reasons for them were explained. The people expressed continued concern about the effects of fallout on their health, but to no greater extent than at meetings during past surveys. As in the past, they claimed that fish poisoning was related to the fallout, and said that certain fish have a black spot in their abdomens, which they believe to be due to radioactivity and to cause sickness in those eating such fish. It was again explained to them that fish poisoning was in no way related to the fallout. A new complaint was that several families had developed inflammation and blistering of the mouth and upset stomachs from eating arrowroot flour which they felt was affected by fallout. It was explained that this type of effect has been noted in other islands when the flour is not properly prepared. They asked whether coconut crabs could be eaten yet and were told that these were still not safe but that continuous tests would be carried out and that they would be informed when the crabs had reached a low enough level of contamination. This is the only item in their diet which they are forbidden to eat. Another complaint was that the coconuts were small and that certain changes had been noted in the coconut and pandanus trees which they believed resulted from fallout.

Despite the above complaints the people were very friendly and cooperated with the examining team in all aspects of the examination that followed.

EXAMINATION FACILITIES

Whole-body gamma spectroscopy was carried out in the cargo hold of the ship. The air-condi-

Figure 7. Hematology laboratory, Rongelap Island.

tioned wooden room containing .e hundred-channel analyzers and other electronic equipment was located close to the steel room. A shower facility was available on the upper deck. The people were brought to the ship in a motor launch and, after taking a soap and water shower and donning paper coveralls, they went down into the hold to the steel room. Taped music was piped in during the procedure. From past experience the people were accustomed to the procedure and showed no fear, with the possible exception of a few of the younger children. Further description of the gamma spectrographic methods will be presented later.

The other examinations were carried out ashore at Rongelap village. As in the past the dispensary was used as a laboratory for the hematological and other laboratory procedures (Figure 7). The adjacent school building was used for taking histories and performing physical examinations on the people. The council house next door was used for special examinations of the skin, aging studies, urine collections and analyses, and x-ray examinations.

HISTORY AND PHYSICAL EXAMINATIONS

Histories were taken by a Marshallese practitioner and an interpreter, with particular emphasis on the interval history during the past year. A special survey was again conducted by the pediatrician to attempt to ascertain more accurately the birth dates of the Rongelap people, particularly the children. Complete physical examinations were carried out including growth and development studies on the children (anthropometric measurements and x-ray examinations of the left wrist and hand for bone development studies); studies of aging criteria; special examinations of the skin with color photography of selected lesions; a special cancer detection survey; and a dental survey.

CANCER DETECTION SURVEY

In the cancer detection survey, procedures included an evaluation of the history, special physical examination, and certain laboratory tests. The family history did not yield satisfactory informa-

Figure 5. Trust Territory ship *Roque*, at anchor, Rongelap Lagoon.

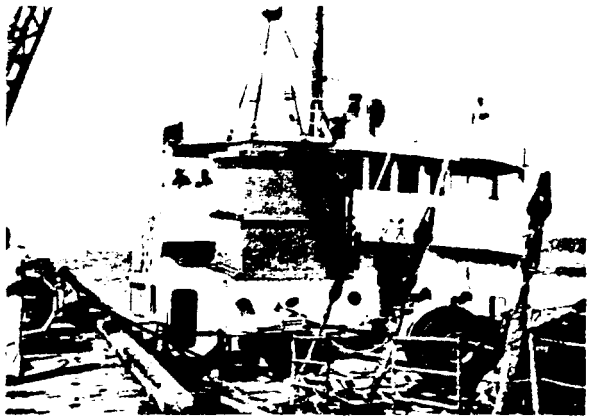


Figure 6. Twenty-one ton steel room being loaded on ship for survey. Used as shielding for gamma spectrographic determination of radionuclides in Rongelap people.

scores into 5-year age groups was done to reduce possible errors due to the uncertainty of the exact age of some individuals.

DENTAL SURVEY

Dental examinations were carried out on most of the people on the island, particularly those requiring dental attention.

LABORATORY PROCEDURES

Hematological Examinations

Complete routine blood counts were carried out with repeat counts on any persons showing abnormalities. White blood counts and red blood counts were obtained with the electronic Coulter, which again proved to be a very satisfactory instrument for examinations of this type in the field. Differential counts were performed in the usual manner after staining with Wright's fluid. Platelet counts were done by phase microscopy. Hemoglobin was determined by the cyan-hemoglobin technique with the Lumitron colorimeter, but only 49 determinations were possible because of breakdown of the Lumitron. Hematocrits were obtained by the microhematocrit method. Reticulocyte counts were carried out in 16 people who had low hemoglobin values. Serum proteins were determined with the Hitachi refractometer. Blood and serum samples for certain studies to be described below were collected and kept under refrigeration and finally shipped back for study.

Urine Analysis

Routine urine analyses were done on almost all people receiving physical examinations. These included determinations of protein and hyperglucosuria by reagent paper strips.*

Serum Examinations for Calcium, Phosphorus, and Protein Bound Iodine

These studies were carried out in six of the children in the exposed group of the growth and development study.**

*Combistix, Ames Company, Inc., Elkhart, Indiana.

**We are grateful to Drs. D.D. Van Slyke and L.V. Hankes of this Laboratory for carrying out these determinations.

Serum Iron

Serum iron determinations were performed in the 16 people who were found to have an anemic tendency to see whether deficiency of this element might be responsible.*

Serum Gamma Globulin Studies

In previous surveys extensive studies have been made on certain genetically inherited characteristics such as blood groupings, haptoglobin and transferrin, hemoglobin types, glucose-6-phosphate dehydrogenase activity of red cells, β-amino-*iso*-butyric acid excretion, etc. One type of gamma globulin had not been previously studied in the Rongelap people, and 149 sera were collected during this survey for determination of the Gm type of gamma globulin. The determinations were carried out by Dr. A.G. Steinberg of Western Reserve University and his collaborators and reported along with the same observations on other populations;[8] the methods used were also reported.

Radionuclide Body Burden Evaluation

The methods used in the radionuclide bod burden evaluation are described later in a separat section.

Results and Discussion

The census of Rongelap people in March 196 was 334, of which 81 were in the exposed gro (including 4 children exposed *in utero* at the time the accident); 33 were children of exposed paren and 220 were unexposed people; Table 1 sho their location. In Table 2 the population is brok down according to percent distribution in vari age groups and compared with that of the Ma shall Islands as a whole for 1948-1950 and of t United States for 1960. The table also shows t median age. The lower median age of the Ma shallese would tend to support the impression th their life span is shorter than that of people in t continental United States, but there has been "population explosion" in those islands whi might account for this discrepancy.

*We are grateful to Mrs. Ruth Saart of South Nassau Co munities Hospital, Rockville Centre, L.I., N.Y., for carrying the serum iron determinations.

Table 1

Location of Rongelap People

	Exposed			Unexposed		
	Adult	Children	Children of exposed parents	Adult	Children	Total
Majuro	2	2	2	9	8	23
Kwajalein	5	2	3	19	20	49
Rongelap	41	25	28	63	84	241
Eniwetok	1	0	0	4	1	6
Other atolls	2	1	0	8	4	15
Total	51	30	33	103	117	334

Table 2

Percent Distribution of Population by Age Groups

Age, yr	Rongelap unexposed (220 people, 1961)	Rongelap exposed (116 people, 1961; includes children of exposed adults)	Marshall Islands (1948-50)	U.S. (1960)
<15	45.4%	50.0%	33.8%	28.7%
15-24	12.3	13.8	18.9	13.8
25-44	23.6	17.2	25.9	27.1
45-64	14.1	10.3	15.5	20.8
>65	4.5	8.6	5.9	9.6
Median age, yr	19.5	14.5	23.6	29.5

Of the 334 Rongelap people, 267 were examined during the survey on Kwajalein, Majuro, and Rongelap. In the exposed group 47 adults, 25 children (age <20), 4 children exposed *in utero*, and 28 children of exposed parents were examined, and in the unexposed comparison population 77 adults and 86 children.

INTERVAL MEDICAL HISTORY

Mortality

No deaths occurred during 1960 in the exposed or comparison population except for two infant deaths. The four deaths that had occurred in the exposed group over the 7-year post-exposure period represent a rate of 7.1 per 1000 population, which is about the same as that reported for the Marshall Islands as a whole (6.8 per 1000). The unexposed population on Rongelap Island has varied, but generally increased, since these people were first examined in 1957, but the death rate in this group appears to be about the same as in the exposed Rongelap group.

Births

Since the number of child-bearing females was not the same in the exposed and unexposed populations, the birth rate for 1960 was calculated on the basis of number of births per woman of child-bearing age, considered to be from 16 through 45 years. There were 20 such women in the exposed group and 29 in the unexposed group (not included in either group were 4 unexposed women whose spouses were exposed males). In the exposed group 7 babies were born, giving an average of 0.35 births per women, and in the unexposed group 11 babies, giving about the same average (0.38). The deliveries were reported to be full term and normal, except that one unexposed women (No. 867) developed severe post-partum hemorrhage and shock and was sent to the Memorial Hospital at Majuro, where her Fallopian tubes were tied.

Congenital Anomalies

One baby, born of unexposed parents, was anencephalic and died several days after birth. The first instance of congenital anomaly in a child of exposed parents was reported during the past year. This was a congenital heart defect in a baby born of an exposed mother (No. 75) but unexposed father. The baby died at four months of age. Unfortunately the diagnosis was not confirmed by autopsy. A low incidence of patent ductus arteriosus, congenital deformity of the hip, and congenital hypoplasia of the middle phalanx of the fifth

finger has been noted previously in children in the unexposed group, but not in children of exposed parents.

Miscarriages and Stillbirths

During the past year two questionable miscarriages occurred in the exposed women, and two miscarriages and one other questionable one occurred in the unexposed women. Thus, during the past two years the incidence of miscarriage appears to be no greater in the exposed than in the unexposed women, and the previously reported suggestive increase in incidence in the exposed women is no longer apparent. Unfortunately it was not possible to have a physician examine the products of miscarriage.

Illnesses

There were few major illnesses reported in the Rongelap population during the past year. Two unexposed people were hospitalized for surgical procedures: No. 867, as mentioned above, had her Fallopian tubes tied following severe post-partum hemorrhage, and No. 855 had a hemorrhoidectomy; recovery in both cases was uneventful. No epidemics of disease occurred in the population, and the dispensary record of the health aide showed the usual number of cases of upper respiratory infections including a notable number of cases of acute bronchitis. Gastroenteritis was frequent as noted in the past. A number of cases of otitis media and skin infections including the prevalent fungus and impetigenous lesions were treated, and also a small number of minor wounds and injuries.

In spite of the complaint at the village meeting of sickness from eating fish, the health aide reported no cases of fish poisoning during the past year. He did report that several members of a few families had developed a sickness 8 to 24 hours after eating arrowroot flour (as mentioned at the village meeting). The affected people developed inflammation and burning of the mucous membranes of the mouth which persisted for about a week. Several also had diarrhea of one-day duration shortly after eating the flour. These illness occurred at two separate times, in June and September 1960. Since most families were not sick from eating the arrowroot flour, it was concluded that in the families in which sickness developed the flour had not been properly prepared. It is known that improper preparation may result in the type of sickness described.

During the interview the health aide admitted that some of the people had at times eaten coconut crabs in spite of the fact that they had been requested not to. Since the individuals involved were not named, it was not possible to attempt to correlate Sr^{90} urinary levels with crab ingestion.

ADULT EXAMINATIONS

Table 3 shows the various abnormalities noted in the exposed and unexposed adult population examined. In Appendix 6 the various clinical findings are enumerated for each individual.

Certain abnormalities such as moderate to severe arteriosclerosis and cataracts showed a higher percentage incidence in the exposed group; however, the increases in both cases were likely related to the larger percentage of older people in the exposed group. In the exposed group 20% of the adults were >65 years of age compared with 7% of the unexposed adults. Taking this into consideration, it does not appear that the abnormalities in the two groups are very different, and no evidence of any increased incidence of degenerative diseases or other diseases is apparent in the exposed group. Several of the older people in the exposed group (No. 57, F, age 107; No. 46, M, age 86; No. 55, M, age 82; No. 56, F, age 78; and No. 28, F, age 75) showed marked infirmities of old age with such findings as arteriosclerotic heart disease, kyphoscoloisis, osteoarthritis, and cataracts with blindness and had to be assisted to the examination room. Only two unexposed people were in this age bracket (No. 862, M, age 88 and No. 946, M, age 85). They showed similar infirmities, but could walk alone.

Cancer Detection Survey

Examinations as thorough as possible under field conditions were carried out for the detection of malignancy. All tumors including presumably benign tumors were recorded. No malignant lesions were detected in either the exposed or unexposed groups.

Pelvic examinations were carried out on the sexually mature females (except when pregnant). Cervical erosions, lacerations, and prolapse were noted with great frequency. Papanicolaou's staining was done on vaginal and cervical smears.

None was positive for malignant cells. It was noted that the secretions were scanty in most of the women, and the smears were consequently somewhat dry. Inflammatory reaction with the presence of blood in the smears was common. Endocervical atypia was noted in a number of cases. The results of these examinations are reported in Appendix 6.

Rectal examinations were carried out on all adults, and proctoscopic examinations when indicated. Several cases of prostatic enlargement were noted, but no evidence of malignancy was apparent. An ulcerating lesion was noted in a 66-year-old female in the exposed group (No. 30) in the upper rectum. Scrapings from this area indicated that it was an inflammatory lesion and not malignant.

PEDIATRIC EXAMINATIONS

During the 1961 survey, 155 children were examined (Table 4). All exposed subjects in the pediatric age group (<20 years) were seen except for two (No. 67 and No. 76) who had moved to other atolls. The increase in number of nonexposed children of exposed parents represents new babies born since the previous survey. New babies also increased the total of nonexposed Rongelap controls, even though a number of children either became old enough to be transferred to the adult study or were lost to the study because of migration. The exposed and nonexposed group at Majuro were all 6 years of age or older. All nonexposed children of exposed parents were <6 years old. Of the 88 nonexposed Rongelap children, 38 were <6 years of age.

The incidence of various physical findings is summarized in Table 5. Adenopathy was defined for the purposes of this tabulation as the presence of (a) nodes 0.5 cm or larger in all areas (cervical, axillary, and inguinal), or (b) nodes 1.0 cm or larger in the axillary regions, or (c) nodes 2.0 cm or larger in either the cervical or inguinal areas. The term "active chronic impetigo" includes several types of superficial skin infections. Many of these lesions probably represented secondary infections of lacerations and abrasions. The incidences of otitis media and adenopathy were related to age, both being more common in younger children. Clinically active chronic impetigo was also found more often in the younger children;

Table 4

Children Examined

	at Rongelap	at Majuro	at Kwajalein	Total
Exposed	22	2	2	26
Exposed *in utero*	2	0	2	4
Nonexposed (exposed parents)	19	2	3	24
Control				
Rongelap series	67	8	13	88
Majuro series	0	13	0	13
Total	110	25	20	155

however, its incidence was higher in the exposed children (although the group was older) than in nonexposed Rongelap children of comparable ages. Its incidence was also higher among the nonexposed Majuro children, who were all in the older group. As in 1959, the pattern of physical findings seemed unrelated to exposure to radiation.

Growth and Development

During the past several surveys considerable effort has been expended to establish as firmly as possible the accuracy of the date of birth for each child. The medical survey records provided reliable written data for children born after March 1954 and for some born immediately before the fallout. For the other children, however, the task of determining birth dates proved to be difficult, frustrating, and unsatisfactory. The reconstruction of the birth chronology was based on an intensive study and evaluation of very frequently contradictory information derived from the following sources:

1. Dates of birth as reported by parents.
2. Dates of birth as recorded occasionally in the village ledgers used for various purposes.
3. Limited number of birth certificates on file at the court house on Majuro.
4. Birth order of children within each family unit.
5. Ranking of childhood population in terms of age by parents.
6. Ranking of childhood population in terms of age by children.
7. Correlation with memorable environmental events.

Table 3

Physical Findings in Rongelap Adults, 1961

	Exposed (47 examined)		Control (77 examined)	
	Subject Nos.	%	Subject Nos.	%
Anemia, anemic tendency	18, 22, 30, 34, 39, 49, 59, 60, 64, 70	21.3	829, 841, 843, 852, 858, 860, 865, 914, 916, 928, 932	14.3
Arteriosclerosis, peripheral, mild	11, 52	4.3	851, 852, 858, 859, 878, 884, 894, 898, 899, 917, 956, 957, 969, 970, 973, 982	20.8
Arteriosclerosis, peripheral, moderate to severe	13, 28, 29, 30, 46, 55, 56, 57, 60	19.1	853, 860, 947, 964	5.2
Asthma	29, 45	4.3		
Auricular fibrillation with myocardial damage	80	2.1		
Blindness	28, 29, 55, 56	8.5		
Cardiac enlargement	30, 60	2.1	853, 859, 942, 964	5.2
Cataracts	28, 29, 46, 55, 56, 58	12.8	853, 860, 984, 964	5.2
Cervical prolapse	12	2.1	893	1.3
Cervical erosion	18, 63, 64, 74	8.5	825, 826, 851, 951	5.2
Cervical lacerations	58, 59, 71, 78	8.5	829, 851, 859, 893, 945, 991	7.8
Congenital defects				
Dislocation of hip	41	2.1		
Prominent head ulna	14, 28, 56	6.4	858, 915	2.6
Bilateral shortening of 5th finger	78	2.1	836	1.3
Polydactylism			938	1.3
Shortened left thumb	57	2.1		
Colloid goiter			853, 858	2.6
Corneal ulcer			859	1.3
Cystocoele	1, 14	4.3		
Cyst, perineal			958	1.3
Dupuytren's contracture			899	1.3
Deafness	1	2.1	853, 916, 964	3.9
Diabetes mellitus	66	2.1	853, 893, 918, 920, 991	6.5
Emphysema			853	1.3
Fungus infection, skin	11, 30, 49	6.4		
Furunculosis	7, 40, 60	6.4	932	1.3
Gynecomastia	46, 55	4.3		
Hydrocoele			961	1.3
Hallux valgus	50	2.1		
Heberden's nodes	52, 57	4.3	928	1.3
Hemiplegia, partial	46	2.1		
Hemorrhoids	27	2.1	849	1.3
Hypertension (>140/90)	1, 28, 30, 60, 73	10.6	858, 859, 878, 947, 982	6.5
Hypotension			865, 914, 932	3.9
Inguinal hernia			1005	1.3
Kyphoscoliosis	13, 52, 56, 57	8.5	859, 860, 964	3.9
Leprosy, arrested	77	2.1		
Myocardial damage or insufficiency (EKG)	46, 56, 60	6.4	844, 851, 858, 878, 884, 893, 917, 947, 956, 957, 969, 970	15.6
Nystagmus			839	1.3
Obesity	1, 28, 49, 50, 60, 71, 74, 78	17.0	838, 849, 851	3.9
Osteoarthritis	13, 29, 46, 52, 55, 56, 57, 60	17.0	858, 859, 860, 884, 894, 898, 899, 915, 922, 928, 964	23.4
Prolapse of vaginal wall	63, 64	4.3	889	2.2
Prostatic hypertrophy	11, 29, 82	6.4	915, 973	4.3
Rheumatoid arthritis (?)			878	2.2
Senility	29, 55, 56	6.4		
Strabismus	11	2.2		
Tonsilar hypertrophy	27, 74	4.3	826, 831, 833, 884, 934	10.6
Tumor, benign	4, 7, 10, 13	6.4	853, 875, 885, 964, 969, 970, 973	14.9
Ulceration, lower colon	30	2.2		
Upper respiratory infection	13, 22, 24	6.4	894	2.2
Varicose veins	13	2.2		
Vitiligo			853	2.2

Table 5

Summary of Physical Findings in Children

	Exposed (26)*	Exposed *in utero* (4)	Nonexposed of exposed parents (24)	Nonexposed Rongelap <6 years (38)	Nonexposed Rongelap >6 years (50)	Nonexposed Majuro (12)
Chronic impetigo (active)	8	1	6	7	3	3
Molluscum contagiosum	0	0	0	2	2	0
Tinea versicolor	0	0	0	0	0	1
Tinea cruris	1	0	0	0	0	0
Chronic otitis media	0	0	2	4	0	0
Acute otitis media	0	1	4	2	2	3
Palpable liver (over 3 cm)	1	0	1	0	0	0
Adenopathy	2	2	4	6	2	0
Cheilosis	0	0	0	2	1	0
Warts	3	0	1	4	3	0
Vitiligo	1	0	0	0	1	0
Furuncle	0	0	0	2	0	0
Rash	0	0	0	2	0	0

*Number examined.

For the Rongelap population a table of most probable birth dates was eventually worked out. Although a few inconsistencies and uncertainties still persisted, these dates of birth were considered to be best estimates and were used in calculating the ages of the children for the analyses. Biologic compatibility of the birth dates within each family group was carefully checked, and physiologic compatibility of status and age for each child was examined.

With the establishment of a presumptive date of birth for each child, analysis of the growth and development data was undertaken. Anthropometric data obtained during 1958, 1959, 1960, and 1961 were used in the initial analysis.* Growth data from examinations prior to 1958 had been collected by several different observers, and this earlier material will be tabulated and analyzed in a subsequent study. Although a number of physical and physiological parameters were measured, the present analysis was limited to stature, weight, and skeletal age. In the very young age groups head circumference data were also evaluated.

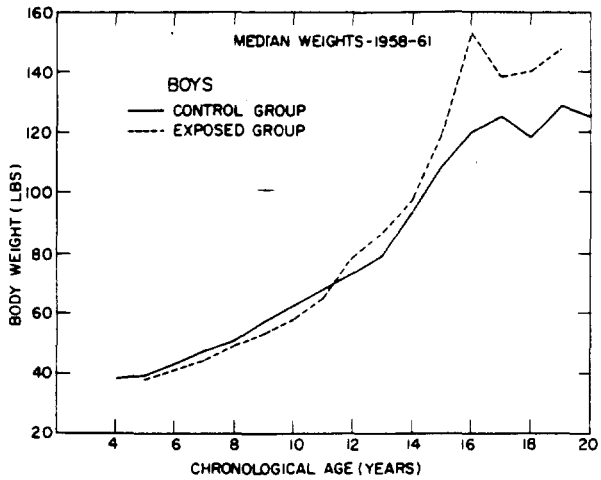
The study population was divided into 5 groups: (1) children born before the fallout and living on Rongelap at the time of fallout (exposed group), (2) children born before the fallout but not living on Rongelap at the time of fallout (control group), (3) children born to mothers who were pregnant when exposed to fallout (exposed *in utero* group), (4) children born subsequent to 1 January 1955 to parents one or both of whom were exposed to fallout (exposed parents group), (5) children born subsequent to 1 January 1955 to parents neither of whom were exposed to fallout (control group for exposed parents group).

Because some of the distributions encountered in these data did not grossly approximate normality or even symmetry of distribution, and because many of the groups were too small to justify making any assumptions about the parameters of the populations from which the samples were drawn (and in many instances too small to permit calculations of any meaningful measure of variability), all analysis of data was done by nonparametric statistical methods.* All measures of central tendency mentioned were medians, and all graphic presentations comparing groups were plotted in terms of medians of the groups. Any descriptive differences between groups mentioned were differences between medians. All tests for significance of differences between groups, unless otherwise specified, utilized the Kruskal-Wallis one-way analysis of variance.[9]

Because the comparisons of skeletal ages and chronological ages involved related distributions, the Walsh test[10] and the Wilcoxon matched-pairs

*The present pediatrician (W.W.S.) actively participated in each of these surveys except the one in 1960.

*We are grateful to Dr. Kenneth Griffith of the M.D. Anderson Hospital, Houston, Texas, for carrying out the statistical analysis.



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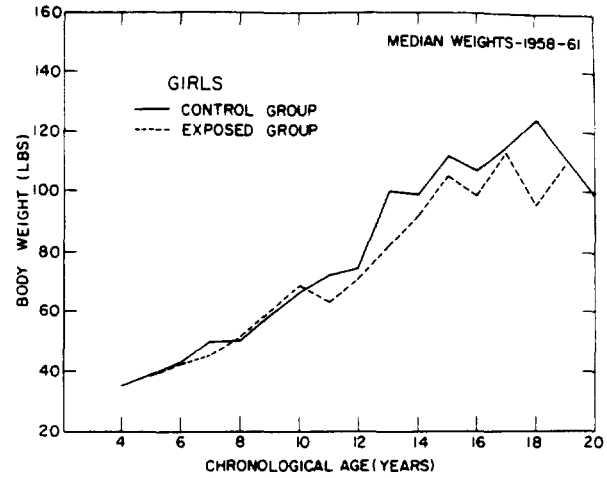

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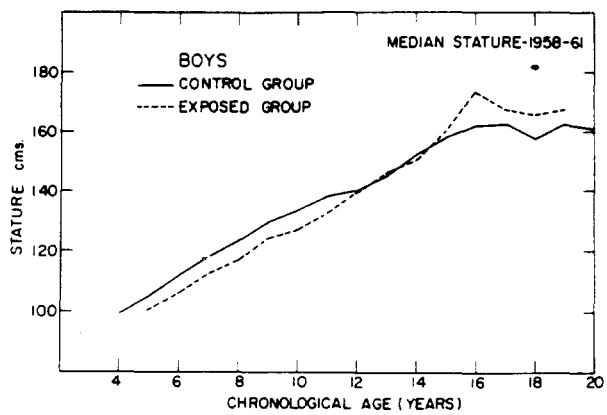

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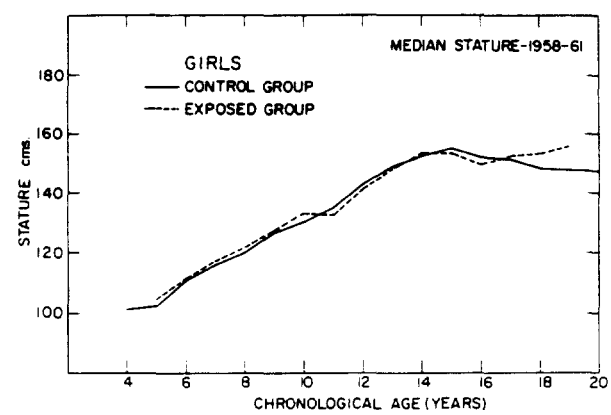

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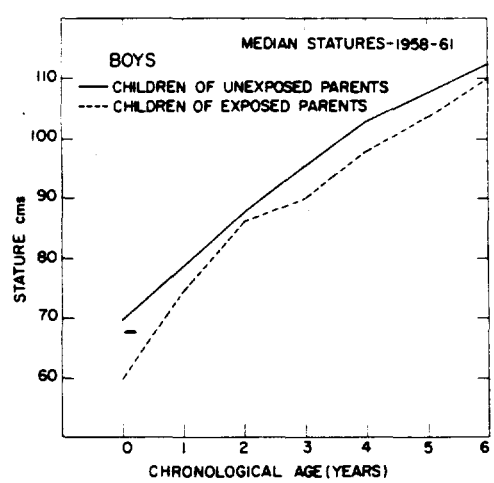

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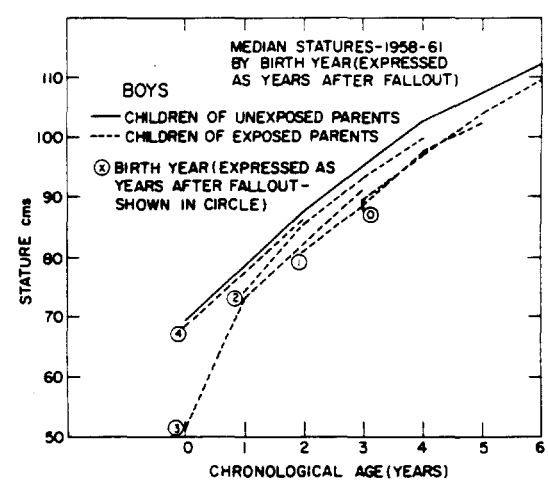

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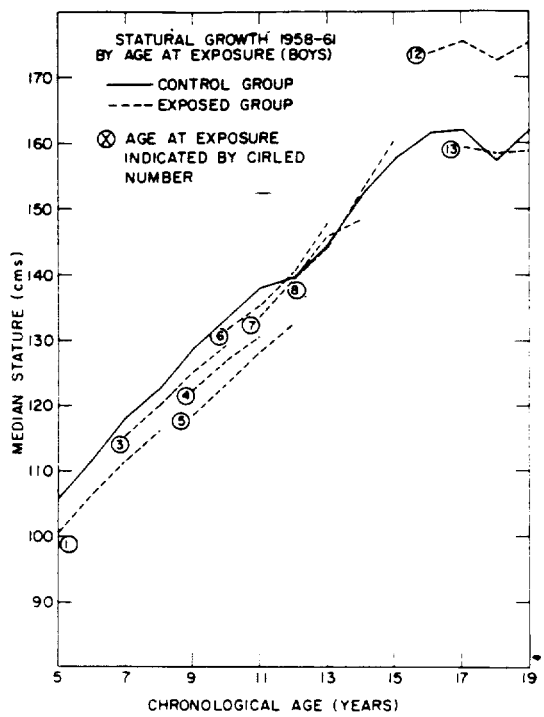

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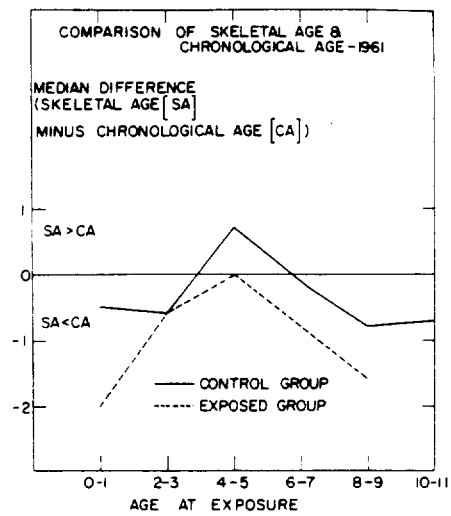

Figure 15.

signed-ranks test[21] were utilized in testing differences in these data. The more powerful Walsh test was used for the comparisons on children exposed at between 12 and 18 months of age because of the very small samples involved. The Wilcoxon test was used for the comparisons on the other children because the larger samples were beyond the functional range of the Walsh test. The Spearman rank correlation coefficient was utilized as the nonparametric measure of correlation.

Comparisons were made (a) between the two sexes for each age separately, (b) between exposed and control groups for each sex and age separately, (c) between exposed and control groups for each age separately with the sexes combined, and (d) between exposed and control groups for each sex, age, and year of birth (or age at exposure) separately. These comparisons have been summarized graphically (Figures 8 to 15), and the results of the comparisons are shown in Tables 6 and 7.

Height and weight data on children born before the fallout showed the expected pattern of pubertal growth spurt occurring earlier in girls than in boys and the eventual superiority in size of boys at maturity. In addition, with respect to stature, there was a distinct tendency, among the boys only, at <12 years of age, for the exposed group to be shorter than the unexposed. This difference was prominent in boys exposed at <6 years and most marked among those exposed at age 1 year, the latter being those aged 12 to 18 months at the time of the fallout. A similar trend, but much less distinct, was also noted in body weight among the boys. No such patterns in growth curve variations between exposed and control groups were noted among the girls.

Among the children born after the fallout, the males with exposed parents were smaller in stature at all ages than those with unexposed parents. The difference was most marked at ages 1, 3, and 4 years. The difference in stature was greatest in boys born <2 years after the fallout. The magnitude of the difference decreased with increasing age. No difference between children with and without history of parental exposure was noted for head circumferences and body weight.

Although the growth data for children exposed *in utero* were examined separately, the small number of individuals in this category prevented adequate statistical analysis. Hereafter the children exposed *in utero* will be included in the group directly exposed to fallout.

Roentgenograms of the left hand and wrist were obtained in 1959 and 1961. In the limited 1960 survey, no x-ray studies were done. The available roentgenograms were assessed by using the inspec-

Table 6

Comparisons of Body Weight and Stature Between Exposed Children and Their Controls
(M = Male; F = Female; E = Exposed; C = Unexposed)

Comparisons	Body weight	Stature
M vs. F (each age separately) E & C combined	F spurt at puberty earlier than M, but M>F after puberty. F>M at age 13. M>F at age 7.	F spurt at puberty earlier than M, but M>F after puberty. F>M at age 13. M>F at age 15, 16, 17, and 19.
E vs. C M only (each age separately)	No significant difference.	Tendency toward E<C before age 12 by 4.4 to 8.5 cm. E<C significant at age 7 and 8.
E vs. C F only (each age separately)	No significant difference.	No significant difference.
E vs. C M & F combined (each age separately)	No significant difference.	No significant difference.
E vs. C* M only (each age separately, each age at exposure separately)	No apparent difference.	Tendency toward E<C in children exposed before age 6, most apparent in those exposed at age 1, who were 4.9 to 6.3 cm shorter with the difference increasing with increasing age, and E<C significant at age 7 and 8.
E vs. C* F only (each age separately, each age at exposure separately)	No apparent difference.	No apparent difference.

Because of small numbers in each group, the groups were compared graphically, except where the graphs suggested certain combinations which could be compared statistically.

Table 7

Comparison of Body Weight, Stature, and Head Circumference Between Children Born to Exposed Parents and Those Born to Unexposed Parents
(M = Male; F = Female; E = Children of exposed parents; C = Children of unexposed parents)

Comparison	Head circumference	Body weight	Stature
M vs. F (each age separately) E & C combined	M>F at age 3. Tendency toward M>F at older ages but not significant.	No significant difference.	No significant difference.
E vs. C M only (each age separately)	No significant difference.	No significant difference.	Tendency toward E<C at all ages by 1.8 to 9.9 cm. Significant at ages 1, 3, and 4.
E vs. C F only (each age separately)	No significant difference.	No significant difference.	No significant difference.
E vs. C M & F combined (each age separately)	No significant difference.	No significant difference.	No significant difference.
E vs. C* M only (each age and each year of birth separately)	No apparent difference.	No apparent difference.	E<C difference greatest in children born 2 years after fallout. In these children, difference is significant at ages 3 and 4. E<C difference diminishes with increasing age.
E vs. C* F only (each age and each year of birth separately)	No apparent difference.	No apparent difference.	No significant difference.

*For these comparisons, most of the groups being compared were so small that it would have been impossible to demonstrate statistically significant differences. These groups were therefore compared graphically, except where the graphs suggested certain combinations which could be compared statistically.

Table 8

Comparison of Stature (1958 Through 1961) of Children With Retarded Osseous Development With That of Their Next Younger Sibs

	Sex	Born	Stature, cm 1958	1959	1960	1961
Subject (#5)	M	10/20/52	95.7	98.8	102.2	104.8
Sib (#85)	M	9/ 7/54	95.5	100.9	108.0	112.5
Subject (#2)	M	10/23/52	103.0	108.3	115.6	119.9
Sib (#91)	M	1/ 3/55	89.8	97.1	104.1	109.9
Subject (#3)	M	9/11/52	98.5	102.2	106.7	108.7
Sib (#83)	M	6/ 8/54	97.6	98.6	113.0	117.0
Subject (#65)	F	12/ 4/52	93.0	98.4	102.9	109.4
Sib (#86)	F	10/17/54	90.6	97.0	103.5	107.5
Subject (#6)	M	10/14/52	100.4	106.3	111.8	116.4
Sib (#84)	M	5/31/54	94.2	98.6	104.8	109.7

Table 9

Skeletal Ages in 8-Year-Old Children

Subject No.	Sex	Age at exposure, mo	Chronological age in 1961, yr	Skeletal age* In 1959	In 1961
2	M	16	8 3/12	4 6/12	6 3/12
3	M	17	8 6/12	2 10/12	3
5	M	16	8 3/12	3 6/12	3 6/12
66	M	16	8 3/12	5 9/12	6 9/12
33	F	20	8 8/12	7 6/12	9 3/12
54	M	12	8 1/12	†	9 6/12
65	F	15	8 3/12	3 6/12	6
814	M	**	8 11/12	5 9/12	8
911	F	**	8	5 6/12	8 1/12
955	F	**	8 10/12	†	10
962	F	**	8 4/12	†	7 4/12
980	F	**	8 3/12	6 10/12	9
996	F	**	8 2/12	†	8 10/12

*Greulich-Pyle standards.
**Control.
†No film.

Table 10

Laboratory Data (1961) on Children Exposed to Fallout at Ages 12 to 18 Months

Subject No.	Serum calcium, mg %	Serum phosphorus, mg %	Protein bound iodine, γ/100 ml
2	9.40	4.61	8.0
3	9.24	4.06	8.8
6	8.36	3.25	10.7
65	8.56	4.34	7.1
83	8.84	4.26	8.1
86	9.80	4.12	12.0

tional method and the standards published by Greulich and Pyle.[12] As shown in Figure 15, the following general trends were noted: (1) Both the exposed and control Marshallese children tended to be less mature skeletally at comparable chronological ages than the norms published by Greulich and Pyle. (2) Boys tended to be consistently less mature skeletally than girls at comparable chronologic ages. (3) Exposed children, both boys and girls, tended to be less mature than control children. (4) When the children were grouped according to age at exposure, the exposed compared to the control group of both boys and girls tended to be less mature. Although these trends suggested that the exposed children may be inferior in skeletal maturation to unexposed children, the differences did not reach the level of statistical significance. The retardation in skeletal development was most prominent in the group of children exposed to fallout at ages 12 to 18 months. As expected, there was a high correlation between retardation in skeletal age and inferiority in statural growth.

Of special interest was the group of children now 8 years old who were exposed to fallout at ages 12 to 18 months. The statural measurements for these 5 children from 1958 through 1961 com-

pared to those of their sibs are shown in Table 8. Median height for controls at the chronological age of 8 years was 122.5 cm for males and 120.0 for females. The skeletal ages for these same children as compared to those of chronological age peers are shown in Table 9. Subjects #3 and #5 continued to show marked retardation in physical and skeletal growth. Subject #65 has shown a spurt in statural growth but continued to lack somewhat in skeletal maturation. Calcium, phosphorus, and protein bound iodine determinations on these children are given in Table 10. Clinically no indication of any disorder involving mineral metabolism or thyroid function was apparent.

LEUKEMIA SURVEY

On physical examination no evidence of lymphadenopathy, spleenomegaly, or other signs of leukemia were detected. Hematological examinations showed no excessive leukocytosis or increased numbers of immature leukocytes in smears. Basophil counts on 4000 leukocytes on each individual showed no elevation of the basophil counts. Alkaline phosphatase studies* on the blood smears showed that, although some people had low levels, this finding was not associated with any other findings suggestive of leukemia.

STUDIES OF AGING CRITERIA

The results of aging criteria studies are plotted in Figures 16 to 32. Individual readings are plotted (circles), and also mean values for each 5-year group combining exposed and unexposed people (squares). The trend of each criterion with increasing age is represented by a line of approximate best fit by eye except in the case of handgrip data for males and females (Figures 30 and 31), which were found to fit the following formulas:**

$$Y = 60 - 0.405A \quad \text{(males)};$$

$$Y = 42 - 0.405A \quad \text{(females)}$$

where Y = handgrip in kilograms and A = age.

The values of the criteria either increase or decrease generally with increasing age. Since the results are similar to those in last year's report,[7] which were given in considerable detail, a lengthy discussion of the various changes will not be included here. Again, little or no difference was apparent between the exposed and comparison groups of the same age, and therefore no discernible effect of radiation on the aging process was noted. Further statistical analysis is needed for more adequate evaluation of the various parameters and better estimates of biological age scores for individuals. It is planned to repeat studies of aging criteria every 2 to 3 years.

RESIDUAL BETA BURNS

Residual skin changes from beta burns sustained in 1954 were observed with certainty in about 10 individuals. Most of these residua consisted of mild changes such as varying degrees of pigment alteration giving a mottled or blotchy appearance, sometimes accompanied by hyperkeratosis (increased rugosity) of the skin. Some showed more pronounced changes than others, such as atrophy and scarring. None of the residual lesions showed any gross tendency to changes associated with the development of chronic radiation dermatitis, nor was there any evidence of malignant change in any of the lesions. As noted last year, in a few cases previously affected areas showed some dark pigmented maculae (lentigo-like). A few appeared as raised moles. It is uncertain whether these lesions are of casual development or related to previous exposure. This type of pigmentation was observed less extensively in the unexposed comparison population. In 10 cases comparison of pictures of these areas taken soon after the appearance of lesions in 1954 and then more recently showed that the maculae had developed subsequent to the lesions. (See Figure 33.) Biopsies were not taken this year but may be considered at a later date for study of the histological appearance of the lesions. In Table 11 are listed descriptions of the residual lesions.

DENTAL SURVEY

A total of 59 school children were examined and treated for caries prevention. Of these, 29 required treatment such as fillings, extractions, and prophylaxis. In the total group 17 showed some evidence of enamel hypoplasia and indication of possible carious lesions in the near future.

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*We are indebted to Dr. W.C. Moloney and Miss Lila Fliegelman of Boston City Hospital for carrying out the alkaline phosphatase analysis of blood smears.

**We are grateful to Dr. R. Hinchcliffe of the State University of Iowa for making this observation.

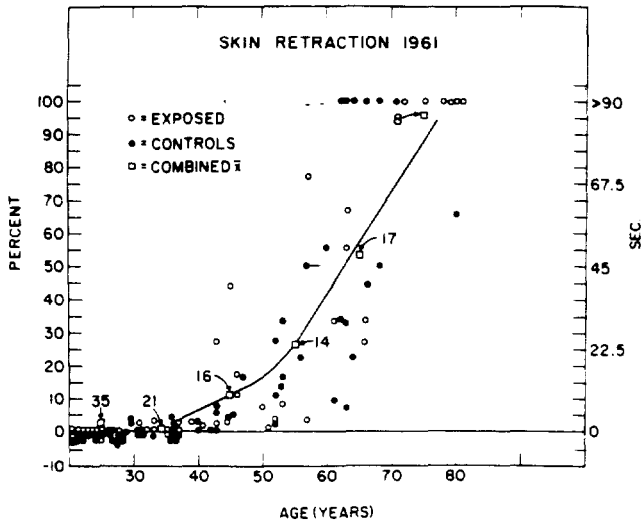


Figure 16.

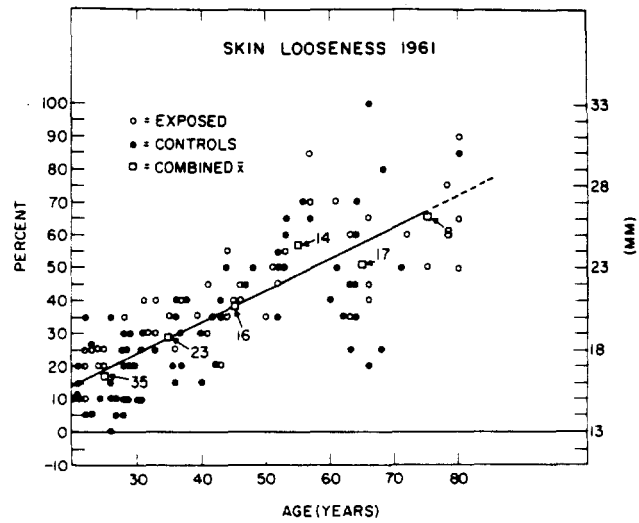
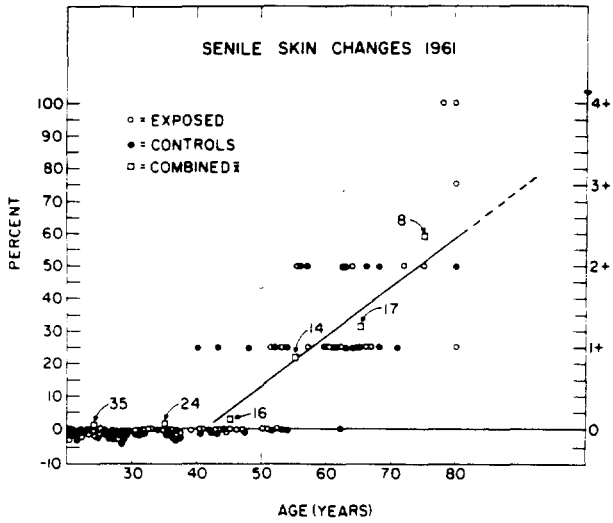


Figure 17.



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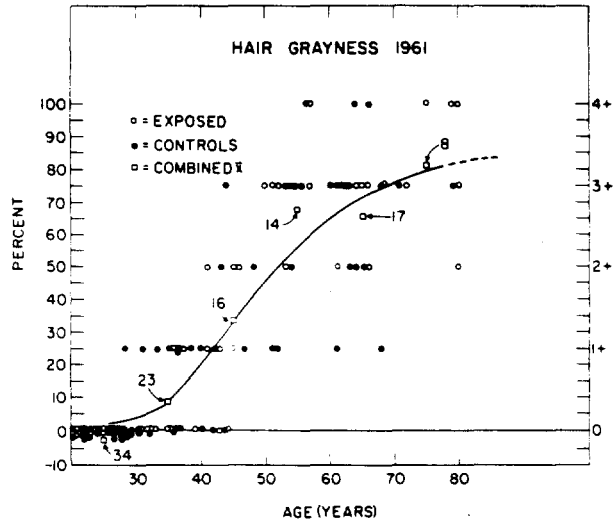

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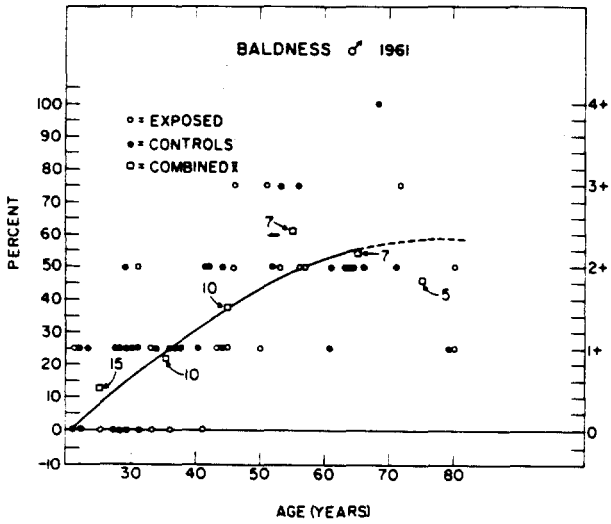

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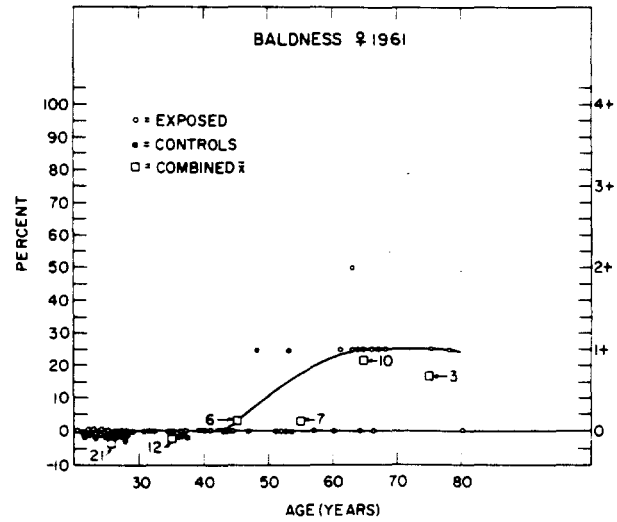

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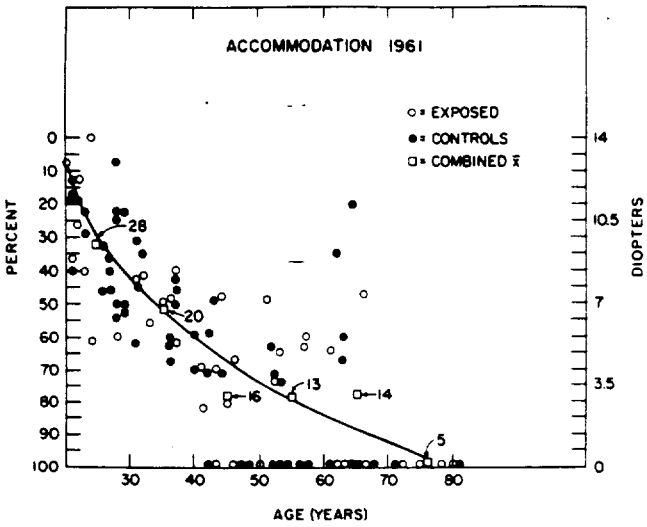

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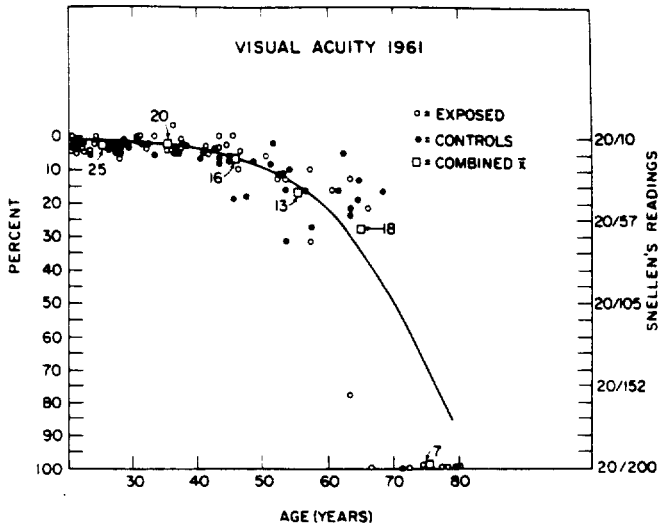

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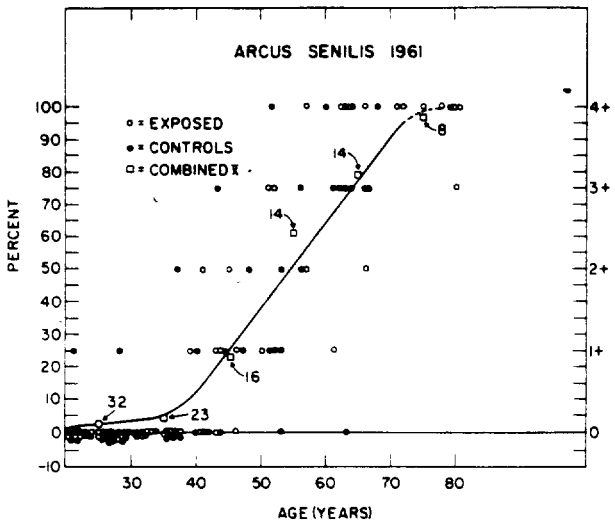

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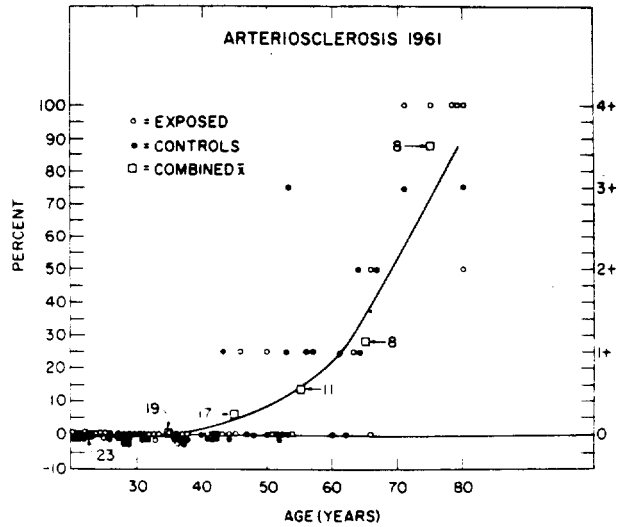

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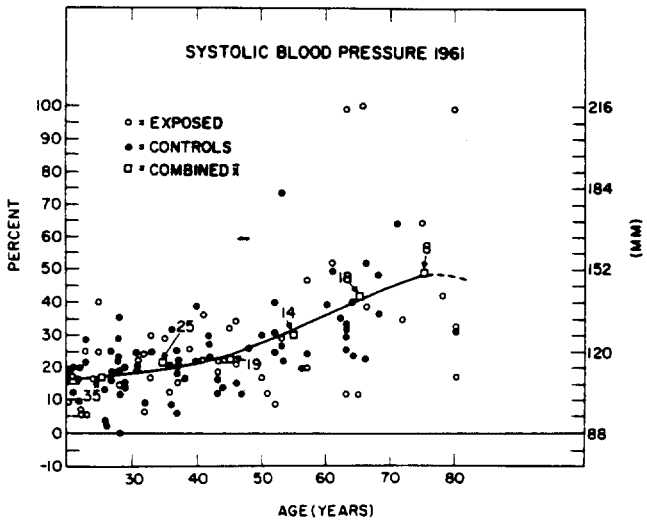


Figure 26.

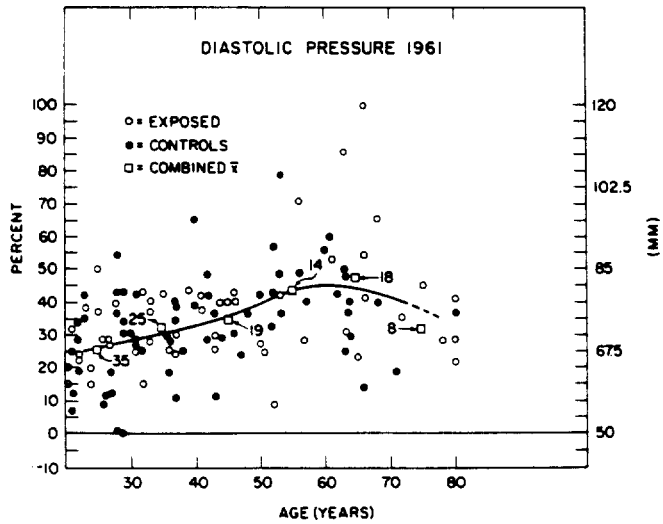


Figure 27.

Figure 33. Pigmented lesions in area of beta burn (Subject #78).

A few adults were also examined and treated. As noted in the past, people of age >40 show a considerable amount of periodontoclasia. A few cases of arrested enamel development involving the occlusal surface of molar teeth were noted, probably due to faulty nutrition during the formative period of the teeth.

No significant differences were found between the Rongelap children and children of the same age group on outlying islands in caries rate, eruption time, or morphology of the teeth. It was estimated that only about 10% of the Rongelap adults practiced good oral hygiene.

Table 11

Residual Beta Burns

Subject No.	Sex	Age	Description
17	F	10	Slight scarring and pigmentation on left antecubital fossa.
24	F	20	Mottled spots of pigmentation and depigmentation on dorsum of feet.
26	M	19	Marked pink-colored depigmented scars on dorsum of right foot, particularly between 1st and 2nd toes. Skin tight in scarred areas – bound to subcutaneous tissues.
39	F	21	Considerable pigment variation and hyperkeratosis on back of neck. Pigmented macular areas on left antecubital fossa and dorsum of feet.
49	F	22	Pigmented maculae (freckle-like) on necklace area of neck, particularly on right side. Increasing in number?
59	F	41	Mottled hyperkeratosis and pigment variation on back of neck.
63	F	43	Considerable pigment variation on back of neck and to a slight degree on right forearm.
67	F	21	Not seen this survey. Previously showed atrophy and scarring on dorsum of feet.
78	F	43	Pigment variation and hyperkeratosis on back of neck. Raised pigmented mole-like lesions on sides of neck, particularly on left side. Appear to be increasing in number (see Figure 33).
79	M	46	White nodular scar and generalized scarring and pigment variation on back of left ear. Area of spotty alopecia on lower occipital region of head.

Laboratory Examinations

HEMATOLOGICAL

Summary tables of the hematological data are presented in tables and graphs in the text, and the raw data on all individuals are presented in the appendices. The more heavily exposed Rongelap group, who received 175 r, are designated as "Rongelap exposed," the Rongelap people who received a smaller exposure of 69 r as "Ailingnae exposed," and the larger unexposed comparison population of Rongelap as "unexposed." Because of the small number of people in the Ailingnae group (15 examined of 18), their data were not treated as fully as those for the larger groups, and are briefly summarized below in a separate para-

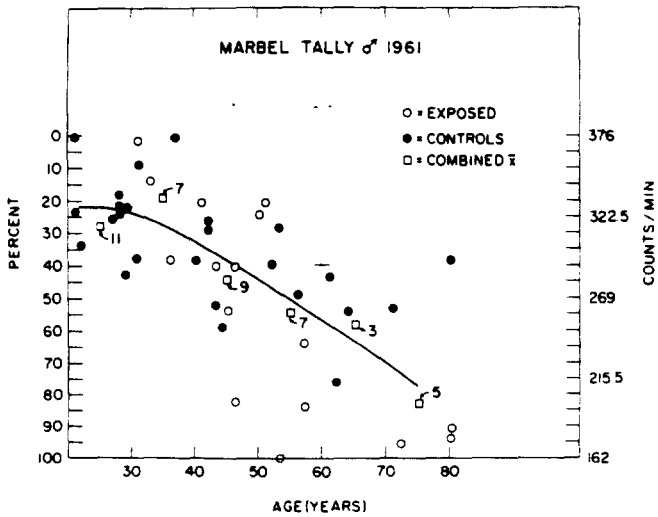


Figure 28.

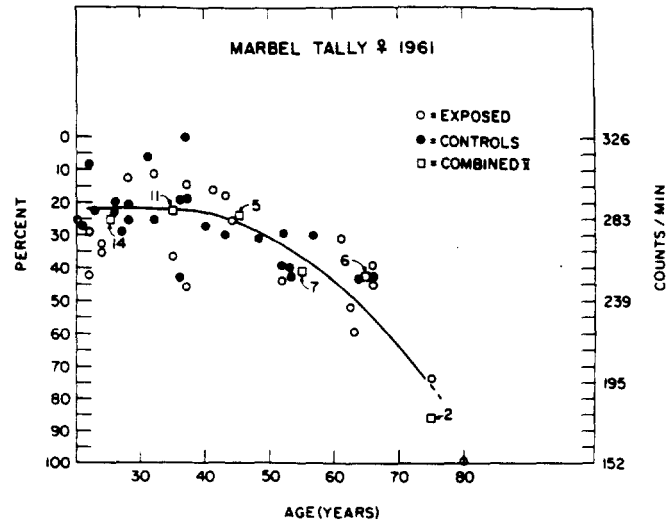


Figure 29.

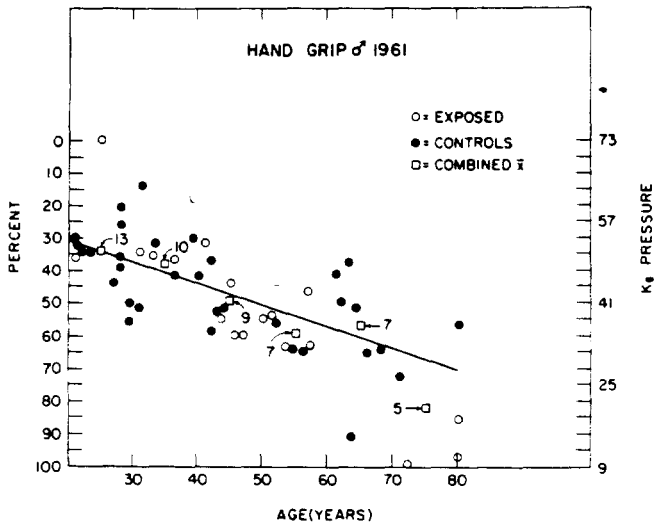
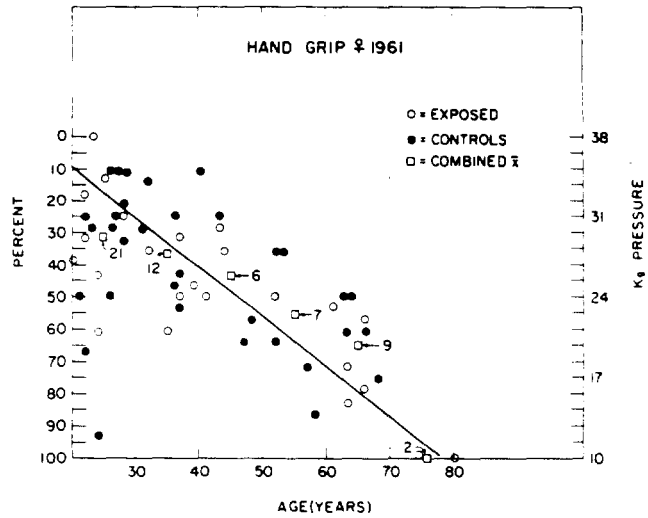


Figure 30.



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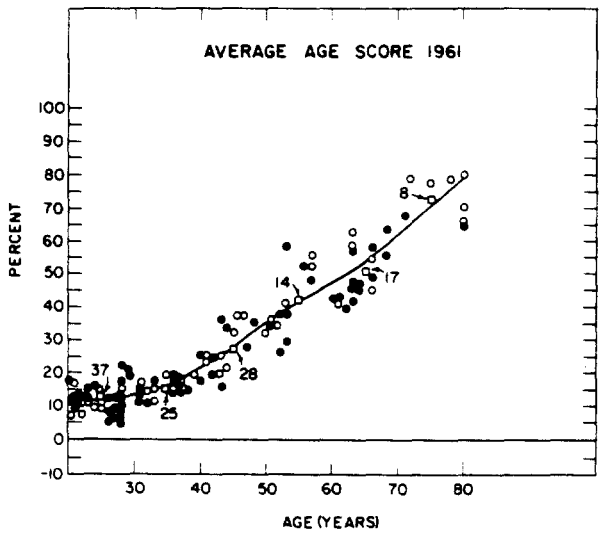

Figure 32.

Table 12

Mean Levels of Peripheral Blood Elements
of Exposed Groups Compared to Those of Unexposed Groups

	Plate. ($\times 10^{-3}$)	WBC ($\times 10^{-3}$)	Neut. ($\times 10^{-3}$)	Lymph. ($\times 10^{-3}$)	Mono. ($\times 10^{-3}$)	Eosin. ($\times 10^{-3}$)	Baso. ($\times 10^{-2}$)
Males, 7–15 yr							
Rongelap exposed**	241±25 (12)*	7.3±1.7	3.6±1.2	3.1±0.8	0.1 ±0.08	0.5±0.1	0.5±0.4
Ailingnae exposed†	315 (3)	9.4	3.5	5.2	0.4	0.4	0.4
Rongelap unexposed	303±70 (16)	8.7±2.9	4.7±1.9	3.3±1.2	0.1 ±0.1	0.5±0.4	0.6±0.3
Females, 7–15 yr							
Rongelap exposed†	299±73 (10)	8.4±1.7	4.5±1.1	3.5±0.8	0.1 ±0.1	0.3±0.1	0.5±0.2
Ailingnae exposed	314±39 (4)	7.5±0.8	3.6±0.7	3.4±0.5	0.04±0.04	0.3±0.3	0.5±0.04
Rongelap unexposed	349±76 (24)	8.4±2.6	4.1±1.6	3.7±1.5	0.1 ±0.1	0.4±0.2	0.5±0.2
Males, >15–40 yr							
Rongelap exposed	282±85 (8)	8.5±1.7	4.7±1.2	3.3±1.3	0.1 ±0.09	0.3±0.2	0.5±0.1
Ailingnae exposed							
Rongelap unexposed	291±83 (24)	7.8±1.9	4.4±1.5	2.8±0.8	0.1 ±0.09	0.4±0.6	0.4±0.2
Females, >15–40 yr							
Rongelap exposed	259±67 (11)	8.1±2.4	5.0±1.5	2.6±1.1	0.1 ±0.08	0.4±0.3	0.5±0.2
Ailingnae exposed	255 (3)	7.0	4.1	2.6	0.09	0.2	0.4
Rongelap unexposed	298±78 (22)	7.7±1.9	4.4±1.6	2.8±0.9	0.1 ±0.08	0.4±0.2	0.5±0.2
Males, >40 yr							
Rongelap exposed	221±74 (9)	5.8±1.5	2.6±0.8	2.8±0.6	0.1 ±0.07	0.3±0.2	0.3±0.1
Ailingnae exposed	213±16 (4)	7.6±3.2	3.9±2.0	3.0±1.0	0.2 ±0.3	0.3±0.2	1.0±0.7
Rongelap unexposed	276±81 (16)	6.7±1.9	3.6±1.1	2.7±0.9	0.06±0.03	0.4±0.2	0.4±0.2
Females, >40 yr							
Rongelap exposed	257±86 (10)	6.4±1.3	3.3±1.2	2.7±0.6	0.08±0.06	0.3±0.2	0.5±0.2
Ailingnae exposed	234 (3)	8.4	5.5	2.5	0.07	0.4	0.5
Rongelap unexposed	295±91 (14)	7.8±2.1	3.7±1.2	3.6±1.3	0.2 ±0.08	0.4±0.4	0.5±0.2

	Hct., %	RBC ($\times 10^{-6}$)	Hgb., g	Serum protein, g
Males, 7–15 yr				
Rongelap exposed**	37.6±2.6	454±49 (10)	11.5±0.9 (10)	7.4±0.2 (11)
Ailingnae exposed†	36.0	456 (1)	10.6 (1)	7.3 (3)
Rongelap unexposed	37.2±2.8	452±39 (11)	11.2±0.5 (7)	7.4±0.5 (16)
Females, 7–15 yr				
Rongelap exposed†	37.7±2.0	453±29 (10)	11.3±0.5 (10)	7.7±0.2 (9)
Ailingnae exposed	37.2±0.8	423 (3)	11.6 (3)	
Rongelap unexposed	37.1±2.0	429±46 (17)	11.2±0.7 (8)	7.7±0.3 (18)
Males, >15–40 yr				
Rongelap exposed	43.2±4.4	475±45 (4)	12.8 (2)	7.8±0.6 (8)
Ailingnae exposed				
Rongelap unexposed	46.3±3.0	491±51 (15)	13.4±1.0 (8)	7.6±0.5 (23)
Females, >15–40 yr				
Rongelap exposed	37.4±4.0	395±33 (9)	10.9±1.4 (8)	7.4±0.3 (11)
Ailingnae exposed	34.6	405 (2)	9.7 (2)	7.5 (3)
Rongelap unexposed	36.4±2.8	391±51 (15)	10.6±1.0 (8)	7.5±0.8 (21)
Males, >40 yr				
Rongelap exposed	40.4±4.2	431±42 (9)	12.7±1.0 (7)	7.4±0.2 (9)
Ailingnae exposed	44.2±2.0	511±48 (4)	13.2 (2)	7.3±0.4 (4)
Rongelap unexposed	41.6±3.7	441±60 (13)	12.4±1.3 (7)	7.6±0.4 (15)
Females, >40 yr				
Rongelap exposed	35.9±2.0	386±27 (10)	10.9±0.9 (8)	7.4±0.2 (9)
Ailingnae exposed	39.3	425 (3)	12.3 (3)	7.5 (3)
Rongelap unexposed	37.7±2.7	413±50 (10)	11.4±1.6 (4)	7.8±0.4 (14)

*Standard deviation and number of people in group.
**Includes 2 children exposed *in utero*.
†Includes 1 child exposed *in utero*.

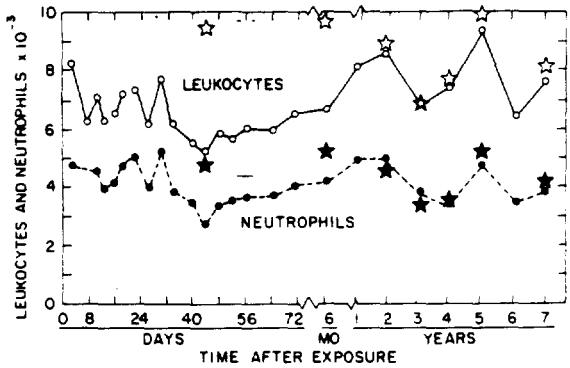

Figure 34. Mean neutrophil and white blood counts of exposed Rongelap people from time of exposure through 7 years post exposure. Stars represent mean values of comparison population.

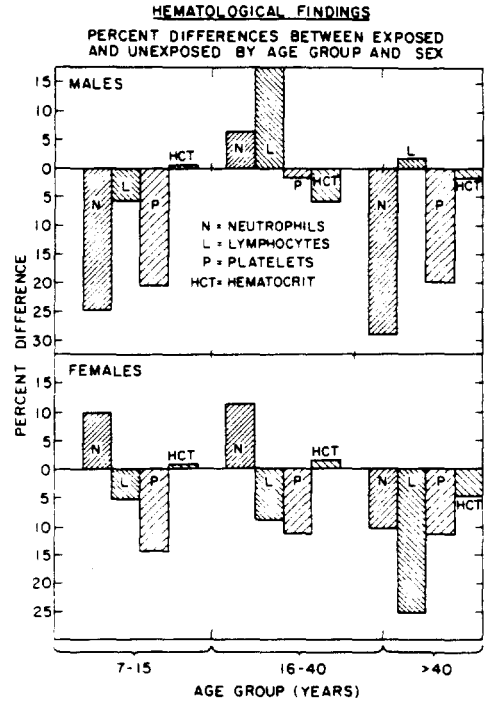

Figure 35. Sex and age distribution of percent difference of peripheral blood elements in exposed compared with unexposed groups.

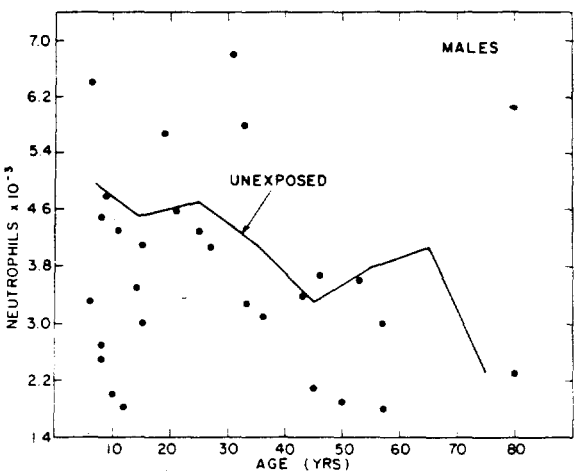

Figure 36. Neutrophil counts of exposed Rongelap males plotted against age. Solid line represents mean level of unexposed male population.

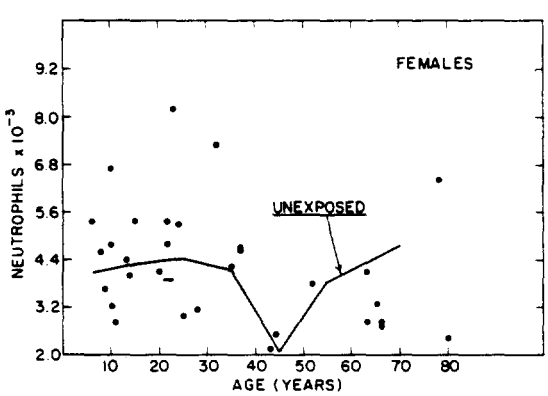

Figure 37. Neutrophil counts of exposed Rongelap females plotted against age. Solid line represents mean level of unexposed female population.

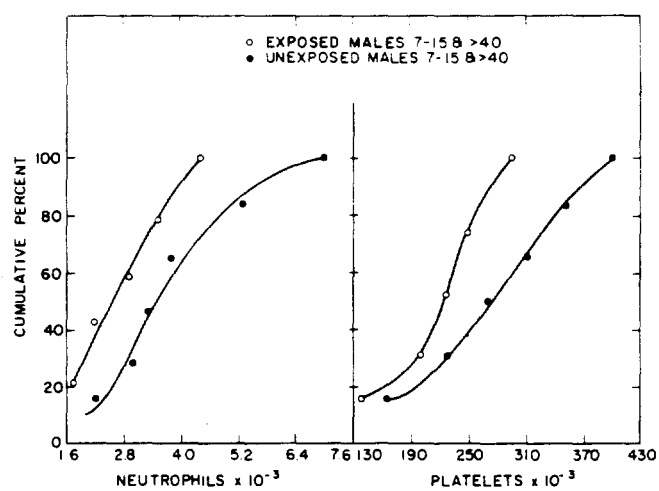

Figure 38. Neutrophil and platelet cumulative percent distribution curves for exposed males <15 and >40 years of age compared with unexposed males of the same age groups.

graph. The data are handled somewhat differently this year: because of certain differences noted in age and sex groups between the exposed and the unexposed, in addition to the comparisons of mean levels for entire groups, comparisons are also made for the age and sex groups of age 7 to 15, 16 to 40, and >40 years.

The data are summarized in Tables 12 and 13 and in Figures 34 to 50. In Appendices 1 and 2 are presented summaries of the mean blood counts of the exposed population and the various comparison populations since exposure in March 1954. In Appendix 3 are listed the individual blood counts for 1961. It should be noted that in 1960 only the exposed group were examined; therefore, the last complete data on both exposed and unexposed groups are for 1959. In Appendix 4 the individual alkaline phosphatase and basophil counts are presented.

Leukocytes

The mean leukocyte levels in 1961 were somewhat below the 1959 levels in the population at large, while both exposed groups (the only groups examined in 1960) showed a slight increase over the 1960 level. Changing neutrophil and lymphocyte mean levels were generally parallel. As will be brought out, a slight deficit in both neutrophils and lymphocytes in certain age groups of the more heavily exposed Rongelap people largely accounted for the lower leukocyte levels. The Ailingnae group (69 r) showed about the same levels of the various leukocytes as the unexposed group. The mean level of leukocytes in the exposed Rongelap people (175 r) was about 6% below that in the unexposed group. The percentage distribution of the various white cell levels in all groups was not very different from that previously reported. Table 12 and Figure 34 show leukocyte levels.

Neutrophils

The mean neutrophil count for the exposed population was slightly (7.3%) below the mean count of the unexposed comparison population. (See Table 12 and Figure 34.) However, examination of the mean counts by age and sex groups made it apparent that the deficit was largely accounted for in the young (age 7 to 15) and older males (age >40 years). This is demonstrated in the histogram (Figure 35) showing the percentage differences in the exposed and unexposed groups classified according to age and sex, and in the scattergram (Figure 36) showing the larger number of individual counts below rather than above the mean line of the unexposed. A cumulative percentage distribution curve of counts combining the younger and older males (Figure 38) shows the curve for exposed males definitely displaced to the left of the curve for unexposed males of the same age groups. Among the females, only in the older group were the neutrophils slightly depressed (see Figures 35 and 37). In groups aged 15 to 40 years, both sexes showed slightly higher neutrophil levels than unexposed people of the same age.

Lymphocytes

As with neutrophils, the exposed population at large showed a slight (6.3%) deficit of lymphocytes compared with the unexposed population (see Table 12 and Figure 39). Among the males, only the younger age group showed slightly lower levels, while the three female groups all showed lower levels. This may be seen in the histogram (Figure 35), the scattergrams (Figures 40 and 41), and the cumulative percentage distribution curve (Figure 42).

Eosinophils, Monocytes, and Basophils

These all showed slightly lower levels than in 1959. The levels of monocytes and eosinophils in the exposed group were somewhat lower than in the unexposed group. As has been noted previously, eosinophil counts >5% of the total white count were common in both groups.

Platelets

The mean levels of platelets in the various age groups in both exposed and unexposed populations were about the same as two years previously (see Table 12 and Figure 43). However, the level in the exposed group as a whole, as in the past, remained below the unexposed level by about 12%. Age distribution scattergrams for the individual platelet counts in both males and females of the exposed population showed more counts below than above the unexposed mean curves (see Figures 44 and 45). This was also borne out by comparison of the cumulative distribution curves of the exposed and unexposed populations, the former showing a continued displacement to the left (Figure 46). The platelet levels showed most depression in the exposed male groups of ages 7 to 15

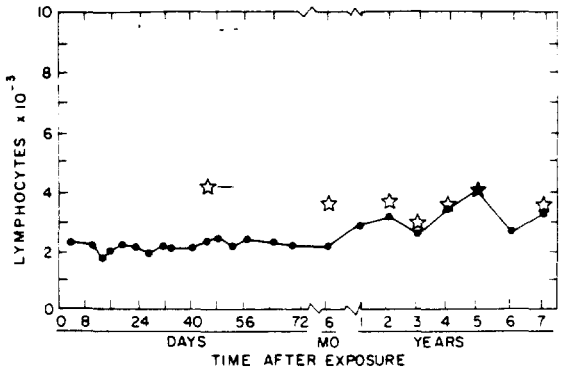

Figure 39. Mean lymphocyte counts of exposed Rongelap people from time of exposure through 7 years post exposure. Stars represent mean values of comparison population.

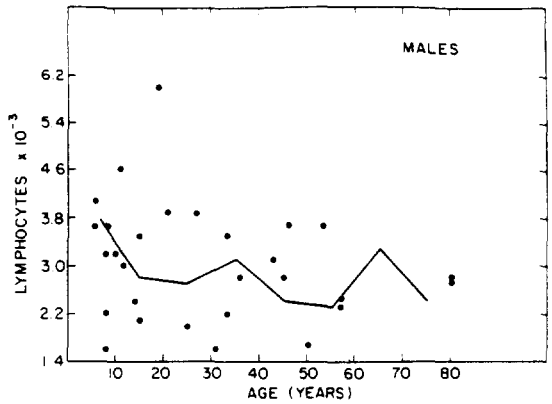

Figure 40. Lymphocyte counts of exposed Rongelap males plotted against age. Solid line represents mean level of unexposed male population.

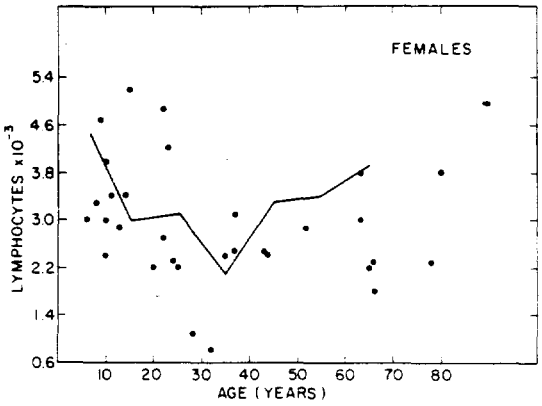

Figure 41. Lymphocyte counts of exposed Rongelap females plotted against age. Solid line represents mean level of unexposed female population.

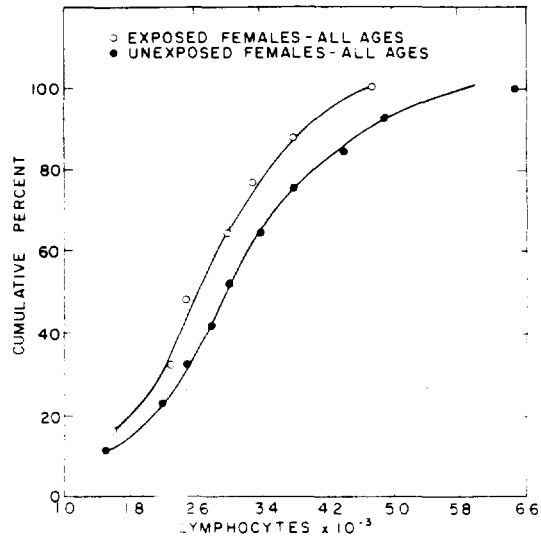

Figure 42. Lymphocyte cumulative percent distribution curves for exposed compared with unexposed population.

and >40 years (see Figures 35 and 38). The continued depression is also indicated by the finding of levels of <250,000 in 44% of the exposed group (including Ailingnae) but in only 25% of the unexposed group.

Erythrocytes

The mean levels of hematocrit, red blood count, and hemoglobin by sex and age group (Table 12) showed generally lower levels in the exposed males of age >15 and females of age >40 years. These differences were not marked as in the case of some of the other peripheral blood elements. The scattergrams (Figures 47 and 48), cumulative percentage distribution curves (Figures 49 and 50) and histogram (Figure 35) demonstrate this.

Morphology

No very unusual morphological changes in blood cells were noted. A few bilobed lymphocytes were seen in differential smears of both exposed and unexposed people, but no counts were made. Price-Jones curves in the past have shown a slight microcytic tendency of the red cells.

Ailingnae Blood Counts

Of the 17 Ailingnae people, 15 were available for examination. The levels of peripheral blood elements in this group were generally very nearly the same as in the unexposed population, except that the platelets were still somewhat below the unexposed mean levels for most of the age and sex groups (see Table 12 and Appendix 3).

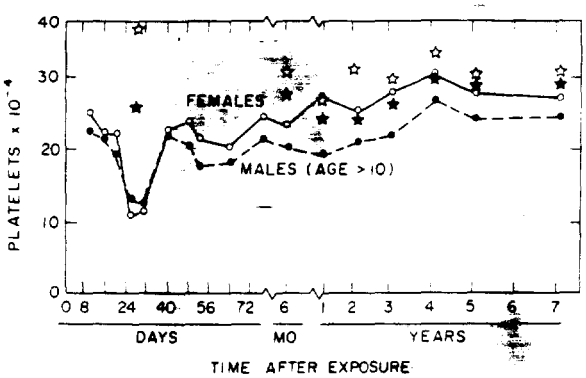

Figure 43. Mean platelet values of exposed Rongelap people from time of exposure through 7 years post exposure. Stars represent mean values of unexposed comparison population.

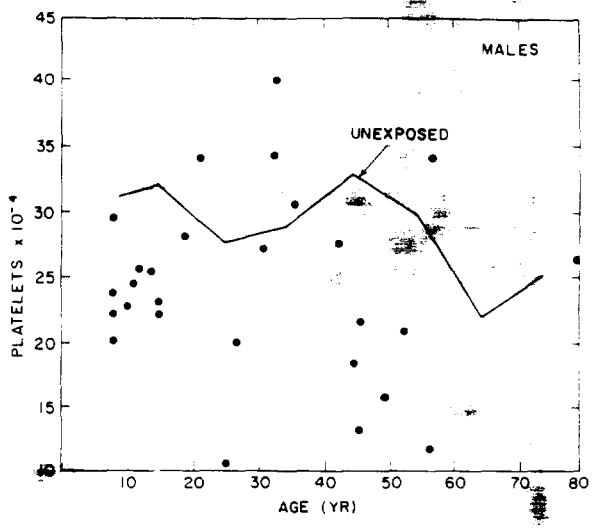

Figure 44. Platelet counts of exposed Rongelap males plotted against age. Solid line represents mean level of unexposed male population.

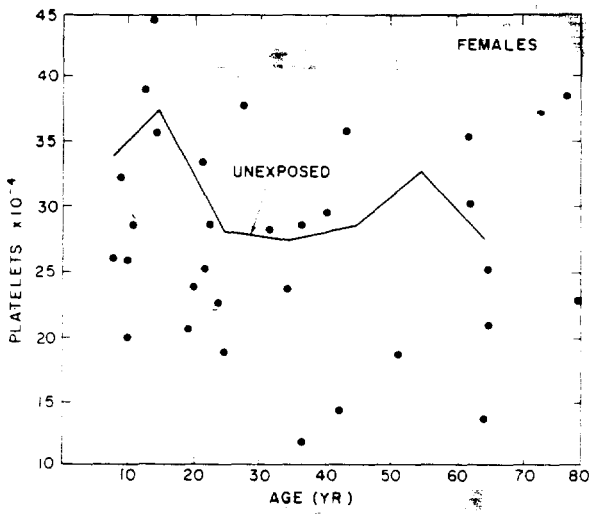

Figure 45. Platelet counts of exposed Rongelap females plotted against age. Solid line represents mean level of unexposed female population.

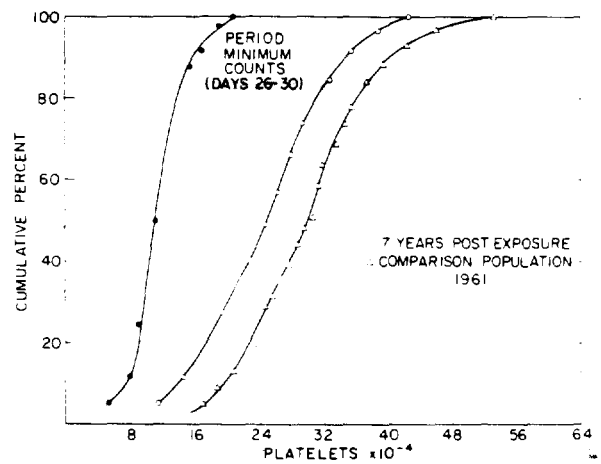

Figure 46. Platelet cumulative percent distribution curves for exposed compared with unexposed population.

Children of Exposed Parents

Peripheral blood elements of children with parents of whom one or both had been exposed were compared with those of children with unexposed parents. These children were <7 years old. The results are presented in Table 13 and Figure 51. The male children of exposed parents showed lower levels of leukocytes and platelets, but not the female children.

Hematological Leukemia Survey

No evidence of leukemia was detected in any of the population studied based on leukocyte counts, differential counts, alkaline phosphatase staining of leukocytes, and counts of basophils in 400 white cells. No marked leukocytosis or immature forms of white cells were observed. Relatively low scoring alkaline phosphatase was recorded in some people, but in no case was it associated with other findings suggestive of leukemia. Basophil counts were elevated slightly (to 2.48%) in a 7-year-old exposed male (No. 3) who had showed a similar elevation in past surveys but no other sign suggestive of leukemia. The individual values for

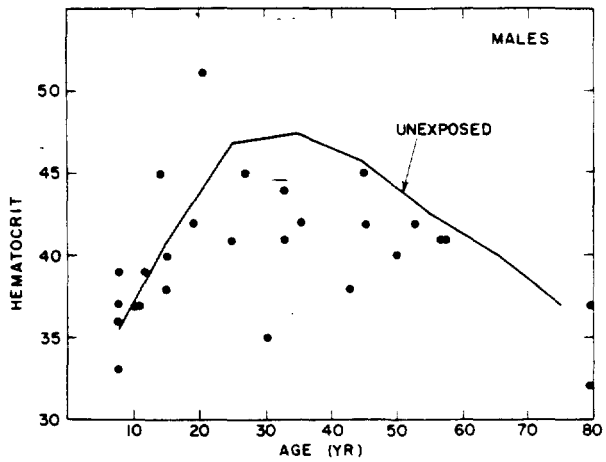

Figure 47. Hematocrit values of exposed males plotted against age. Solid line represents mean level of unexposed male population.

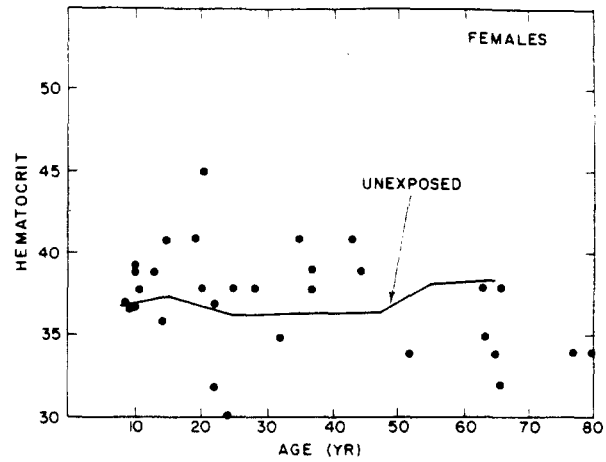

Figure 48. Hematocrit values of exposed females plotted against age. Solid line represents mean level of unexposed female population.

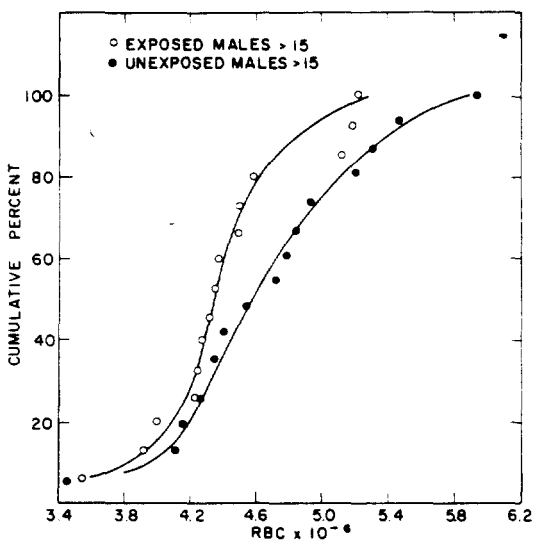

Figure 49. Red blood count cumulative percent distribution of exposed males >15 years of age compared with unexposed males of the same age group.

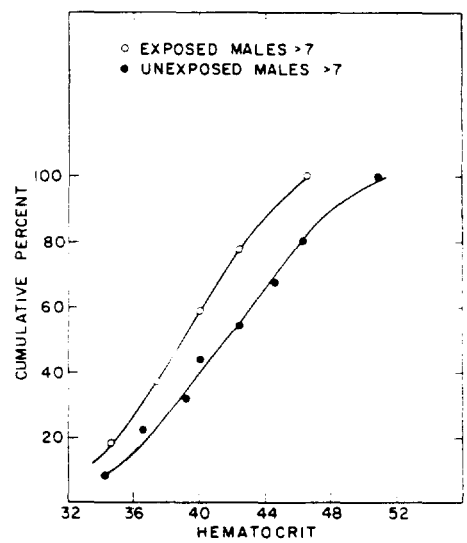

Figure 50. Cumulative percent distribution curves for hematocrit values in exposed males compared with unexposed males.

the basophil and alkaline phosphatase counts are presented in Appendix 4.

Comments on Hematological Data

The 1961 leukocyte and red blood counts remained slightly below the 1959 levels in the population at large, while the platelets were at about the same level. Such fluctuations have been noted before and have not been explained. The exposed group continued to show slight depression of certain blood elements compared with the unexposed group. Though the mean levels were only slightly below those of the controls, more pronounced differences were brought out by comparison of sex and age groups. Thus, it appeared that the exposed male children (age 7 to 15 years) and the exposed older people of both sexes (age >40) showed lower levels, particularly of neutrophils and platelets, than did the other age-sex groups (see Figure 35). It is interesting that these blood

Table 13

Mean Levels of Peripheral Blood Elements of Children of Exposed Parent(s) Compared to Those of Children of Unexposed Parents

	Plate. ($\times 10^{-3}$)	WBC ($\times 10^{-3}$)	Neut. ($\times 10^{-3}$)	Lymph. ($\times 10^{-3}$)	Mono. ($\times 10^{-3}$)	Eosin. ($\times 10^{-3}$)	Baso. ($\times 10^{-2}$)
Males, <7 yr							
Of exposed parent(s)	351± 94 (11)*	8.3±1.9	3.6±1.3	4.3±0.8	0.1±0.08	0.4±0.2	0.6±0.3
Of unexposed parents	374±104 (20)	10.3±2.9	4.5±2.0	5.0±1.2	0.2±0.1	0.6±0.5	0.5±0.2
Females, <7 yr							
Of exposed parent(s)	426± 90 (10)	11.3±2.5	4.7±1.2	5.6±1.9	0.2±0.1	0.8±0.4	0.6±0.3
Of unexposed parents	413± 68 (17)	10.7±2.8	4.6±1.8	5.3±1.6	0.2±0.1	0.6±0.5	0.7±0.3

	Hct., %	RBC ($\times 10^{-4}$)	Hgb., g	Serum protein, g
Males, <7 yr				
Of exposed parent(s)	36.4±1.0	429±92 (11)	10.4±0.4 (4)	
Of unexposed parents	36.1±3.4	439±41 (18)	10.8±0.6 (7)	7.2 (1)
Females, <7 yr				
Of exposed parent(s)	35.5±2.0	433±40 (6)	10.4 (3)	
Of unexposed parents	37.0±2.0	448±42 (16)	10.9±0.2 (9)	

*Standard deviation and number of people in group.

elements in the male children of exposed parents (age <7 years) also are lower than levels in male children of unexposed parents (Table 13), and that these same children appeared to show suggestive evidence of retardation of growth and development (see section on Growth and Development).

The slight anemic tendency in the Rongelap people, noted in the past, was still evident, though the explanation was not clear. Serum iron levels were generally not depressed. It is possible that another type of nutritional deficiency exists. However, their serum protein (particularly gamma globulin) and serum vitamin B_{12} levels tended to be high. The high incidence of eosinophilia may be related to chronic fungus infection of the skin, intestinal parasites, or other causes.

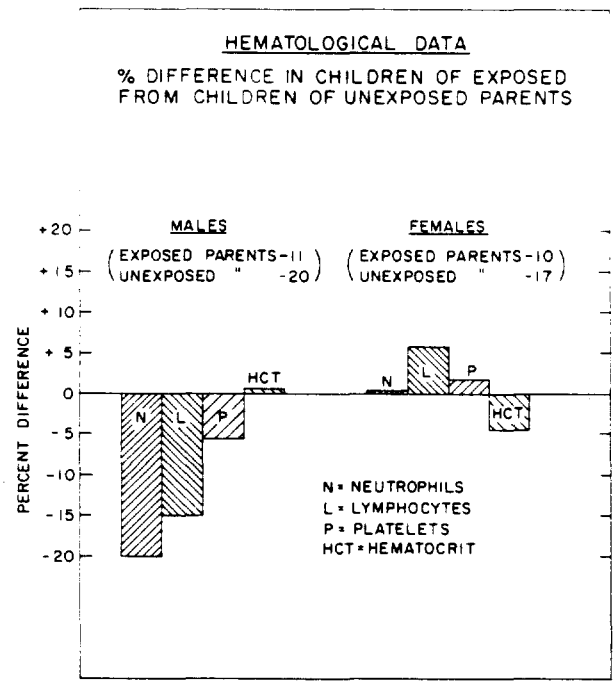

Figure 51. Sex and age distribution of percent differences of peripheral blood elements in children of exposed parents (one or both exposed) compared with children of unexposed parents.

OTHER LABORATORY STUDIES

Serum Iron

Serum iron levels were determined in 16 people (11 exposed and 5 control unexposed) who had an anemic tendency. The results are presented in Table 14 along with the hematocrit. With 83 μg considered as the lower limit of normal, only Nos.

Table 14

Serum Iron Levels (With Hematocrit)

Subject No.	Hematocrit	Serum iron, μg
2	36	122
3	37	108
6	36	78
13	34	83
22	30	139
30	32	104
34	34	104
65	37	100
70	26	69
83	35	96
86	34	83
843	36	135
860	37	91
865	35	96
912	31	131
929	37	96

69 and 78 (both with low hematocrits) were subnormal.

Glucosuria

Routine urine analysis carried out on 87 individuals showed positive sugar reactions in 8 persons, one exposed (No. 66, age 36) and 7 unexposed (No. 853, age 56; No. 893, age 40; No. 916, age 39; No. 920, age 29; No. 956, age 53; No. 961, age 68; and No. 991, age 53). Unfortunately, blood sugar determinations were not possible. As noted before, the incidence of diabetes in the Marshallese appears to be higher than in the United States population. These cases were of the chronic type with no acute manifestations. In 1959, as reported previously, however, an unexposed woman died of uterine infection complicating diabetes.

Protein Bound Iodine; Serum Phosphorus and Calcium

These levels were determined in 6 children, 4 of whom showed evidence of retarded growth and development (Nos. 2, 3, 6, and 65; see Table 10). The PBI values found varied from 8.0 to 12.0 γ/100 ml. Since particular care had been taken and specially cleaned glassware used, the slightly high values found appeared to be valid and substantiated the earlier findings of slightly increased levels, particularly since the samples were analysed at a different laboratory than previously. No explanation is apparent for this finding. The calcium levels were a little on the high side of normal and the phosphorus levels, normal. Thus there was no indication that the impairment in growth and development noted in these children was related to these findings.

STUDIES OF GENETICALLY INHERITED TRAITS

Gm Phenotypes

These determinations were run on sera that had been obtained in 1959. No further studies of genetically inherited traits were carried out during the 1961 survey. The results on Gm phenotypes on each individual are presented in Appendix 5. The Micronesians were found to be 100% $Gm^{(a-)}$ and nearly 100% $Gm^{(b-)}$. There was a complete absence of Gm^{x} and a high frequency of Gm-like (Gm^{c}). Table 15 shows the frequencies in percent of Gm factors in the Marshallese (Micronesians) and various other populations.

The following is taken from a summary of the paper by Steinberg et al.[5]

"Whites have a relatively low frequency of Gm^{a}, high frequency of Gm^{x} and Gm^{b}, and have no Gm-like. The African and Micronesian populations have a high frequency of Gm^{a}, Gm^{b}, and Gm-like, and have no Gm^{x}. North American Indians have a high frequency of Gm^{a} and Gm^{x} (higher than whites), a low frequency of Gm^{b}, and have no Gm-like. Eskimos have a similar pattern, but may have a lower frequency of Gm^{x}.

"The genetic aspects of the Gm factors are discussed. Whites appear to have alleles Gm^{a}, Gm^{ax}, and Gm^{b}. It is tentatively suggested that among colored populations, alleles Gm^{ab}, Gm^{bc}, and Gm^{abc} may occur, where c represents Gm-like."

Estimation of Internal Body Burden of Radionuclides

BACKGROUND

For large scale surveys, such as the Marshallese surveys, measurement with whole-body gamma counters (both liquid scintillation detectors and crystal detectors) has proved to be the most satisfactory technique. Early evaluation of the body burdens of the people of Rongelap was carried out by radiochemical urinalysis, which is time con-

suming and difficult. In 1957, several Rongelap people were brought to Argonne National Laboratory, where it was demonstrated that their body burdens of gamma emitters could be measured in the whole-body counter. Cs^{137} and Zn^{65} were shown to be the prominent isotopes in the Marshallese at that time.

The Rongelap people, originally evacuated to another island in 1954, were returned to their home island in July 1957. Since the island and the indigenous food sources still had a low level of persisting radionuclides, continued evaluation of the body burdens in these people was of considerable interest.

A portable whole-body counter was designed and constructed at Brookhaven National Laboratory and transported to the Marshall Islands, where it has since been employed during the annual medical surveys to ascertain the level of internally-deposited gamma-emitting isotopes. In 1958, 100 Marshallese people were counted, and the procedure was repeated one year later to obtain gamma spectra on 227 people. Details of the exposure and findings of these medical studies have been reported.[1-7]

During the 1961 survey the portable shield was used in conjunction with an improved detection and data-recording apparatus. The gamma-ray spectra of 110 people were obtained. Half the people measured had been exposed to the fallout in 1954; the other half were unexposed.

METHOD

Shielding was provided by a 21-ton room with walls constructed of laminated ¼-in.-thick steel panels bolted together, which can be disassembled. The inside dimensions of the room are 5×5 ft and 6 ft high. Figure 52 shows the steel room (as set up for the 1959 survey). In 1961 the room was set up in the cargo hold of the *Roque* (Figure 6). Along with the steel room, an air-conditioned prefabricated wooden room 7×8×10 ft was set up to house the pulse-height analyzer and other elec-

Table 15

Frequencies (in Percent) of Gm Factors in Various Populations

Population	No.	Gm Factor			
		a	b	x	like
United States					
White	303	50.8	91.8	17.5	0
Negro	364	98.1	97.5	3.0	27.7
Athabascan Indians (Alaska)					
Artic Village	58	100.0	0	41.4	0
Fort Yukon	51	100.0	3.9	31.4	0
Total	109	100.0 Av	1.8 Av	36.7 Av	0 Av
Eskimos (Alaska)					
Wainwright	50	100.0	20.0	6.0	0
Africa					
Yoruba	35	100.0	100.0	0	100.0
Fulani	35	100.0	100.0	0	94.3
Bashi	37	100.0	94.6	0	35.1
Pygmies	120	100.0	100.0	0	100.0
Micronesia					
Rongelap Atoll	149	100.0	97.3	0	43.6
Surinam					
Djuka Negroes	35	100.0	100.0	0	62.9
Javanese	20	100.0	100.0	15.0	50.0
Oyana Indians	15	100.0	86.7	26.7	0
Carib Indians	19	100.0	79.0	52.6	0

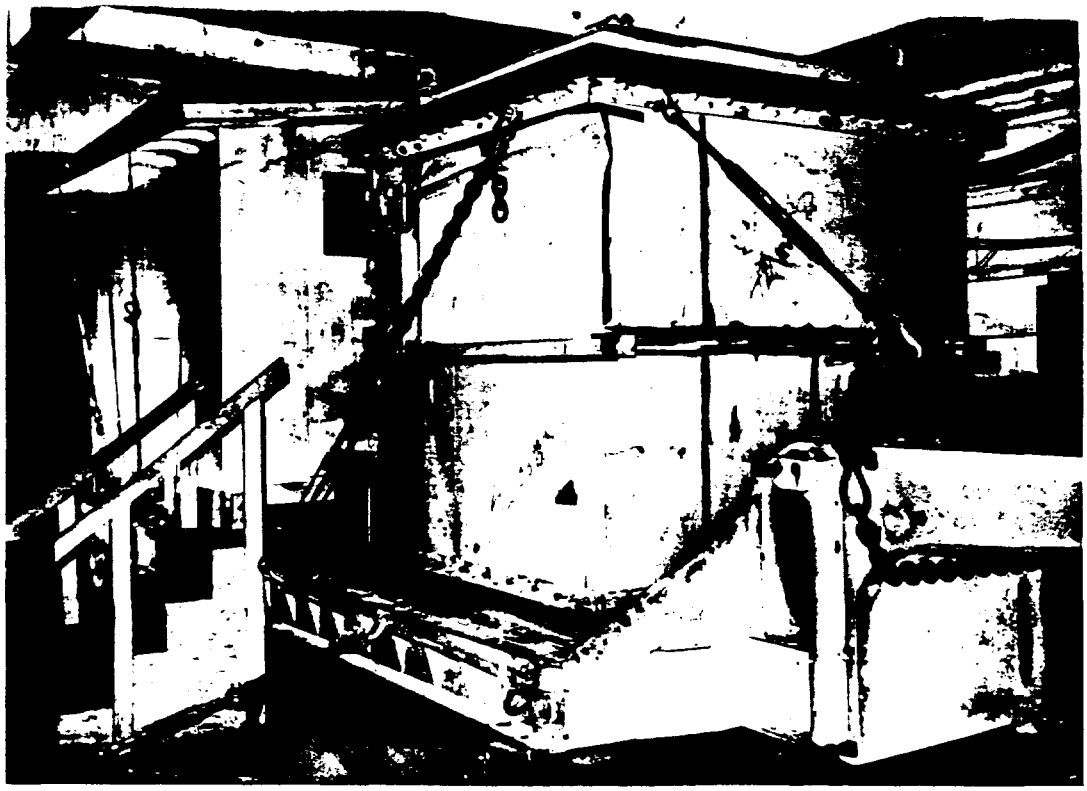

Figure 52. Portable steel-shielded room and air-conditioned instrument room as used on the 1959 survey.

Figure 53. Marshallese subject in the standard counting position in steel room under the NaI (Tl) crystal detector.

Figure 54. Marshallese subject leaving the counting room through the pneumatically-driven sliding door.

PRIVACY ACT MATERIAL REMOVED

tronic equipment (Figure 52). The air conditioning and dehumidification were of considerable value in maintaining the stability of the instruments in the tropical climate.

The subjects were ferried out to the ship, and, before being counted, showered and donned paper coveralls and slippers in order to minimize the possibility of counting external contamination from the island environment on their bodies and clothing. The subjects were seated on a folded hospital cot and placed in a standard, fixed position under the detector, as shown in Figure 53. Figure 54 shows a Marshallese subject leaving the counting room through the pneumatically-driven sliding door.

The counting geometry employed in the portable counter is identical to that used in the permanent whole-body counter at BNL, which makes possible the cross calibration of the two units.[13] The efficiency and precision of the portable counter for the various isotopes are presented in Table 16. The values obtained are very similar to those for the whole-body counter at BNL.

The background observed in the Marshall Islands counting room in the range 100 kev to 2 Mev was 1796 cpm (counts per minute), a value somewhat higher than the average background level observed at BNL (1400 cpm). The increased background count in the Marshall Islands was chiefly in the very low energy range and probably can be explained in terms of the thinner shielding in the portable counter and the omission of Pb lining for the ceiling of the counting room. To some extent the thinner shielding was compensated for by the shielding provided by the water under and around the ship.

Since difficulties had been experienced in identifying small photopeaks of various isotopes deposited in the Marshallese in the presence of relatively large amounts of Cs^{137} and Zn^{65}, the counting time was increased for a number of subjects over that used in previous years. In addition, a larger crystal detector was substituted for the 5-in. detector formerly used. The majority of the subjects were counted for 10 min, and a large number were counted for 30 min.

An 8×4-in. NaI (Tl) crystal (Harshaw) detector was placed above the patient at a distance of 19 in. (see Figure 53). Pulses from three 3-in. photomultiplier tubes were fed into a Nuclear Data 256-channel transistorized pulse-height analyzer (Model 120). The analyzer fed the data directly to an IBM typewriter and simultaneously to a Tally paper punch unit, Model 420. Provision is made in this analyzer for transferring spectra recorded on paper tapes into the memory of the analyzer so that calibration spectra can be compared with the incoming data when desired.

The data recorded on the punched paper tapes were transferred to IBM cards and thence to the

Table 16

Properties of Portable Whole-Body Counter for Measuring Specific Radionuclides

	Cs^{137}	Co^{60}	Zn^{65}	K^{40}
Photopeak energy (Mev)	0.66	1.17	1.12	1.46
Energy band measured (Mev)	0.61–0.71	1.12–1.22	1.07–1.17	1.41–1.51
Background (cpm)	75.5	29.6	33.7	40.2
Calibration factor (C.F.) (cpm/μC)	6114	4320	1733	0.86 (cpm/kg)
Precision (P) of counter* (mμC)	0.360	0.324	0.866	3.8 (g)
Percent standard deviation of count rate**	±0.23	±10.0	±1.73	±3.58
Integrated background (cpm) at 0.2 to 2 Mev	1796			

*For 70-kg phantom in standard counting geometry,

$$P=\frac{\sqrt{(R_c/t_c)+(R_b/t_b)}}{\text{C.F.}}.$$

where R_c = Sample plus background counting rate (cpm), R_b = background counting rate (cpm), t_c = sample plus background counting time (min), and t_b = background counting time (min).

**Average Marshallese adult male.

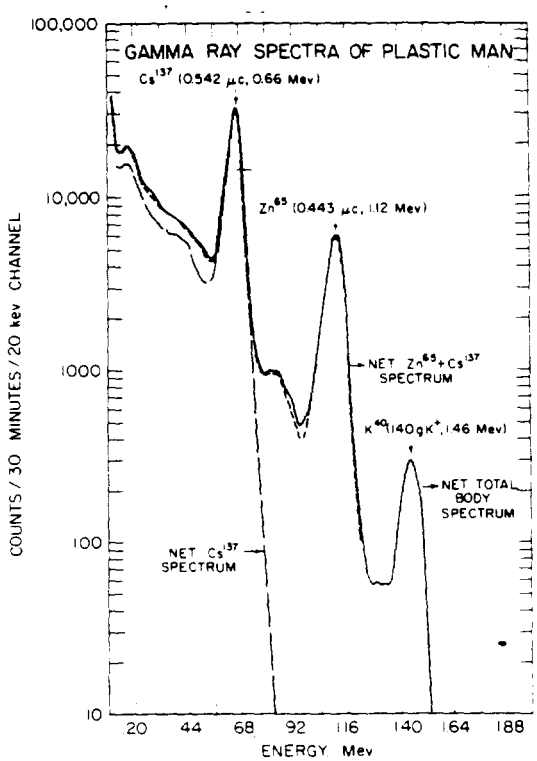

Figure 55. Gamma spectrum of the calibration phantom containing Cs^{137}, Zn^{65}, and K^{40} in the approximate range found in Marshallese subjects. The dotted lines indicate the results of spectral stripping of the higher energy photopeaks.

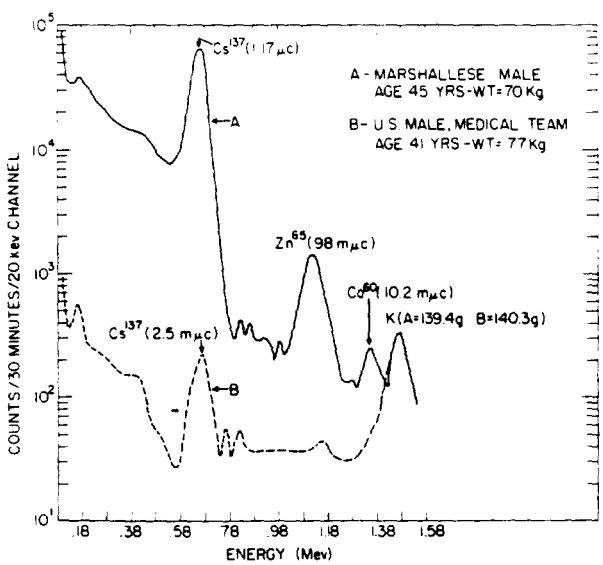

Figure 56. Spectrum of typical Marshallese adult male compared with that of member of the U.S. medical team.

magnetic tape of a 704 computer. The task of "spectral stripping" was carried out on the computer with a FORTRAN program. In this operation the spectrum of each individual isotope is removed from the total spectrum obtained for the subject, which represents the combination of the contributions from all the isotopes deposited in that subject.

Spectra for each of the individual isotopes quantitated in this study were obtained with the use of an Alderson phantom (REMCAL). Solutions of known concentration of each radionuclide were placed in the phantom to approximate the effects of tissue absorption and scatter. The spectrum of the phantom for each of the isotopes was obtained under conditions of counting geometry identical with that used in counting the subjects. By this technique it was possible to simulate quite closely with the phantom the multicomponent spectra of the Marshallese. A representative Marshallese spectrum obtained by adding K, Cs^{137}, and Zn^{65} at average levels (as determined in the medical study of 1959) to the phantom is shown in Figure 55. The K, Cs^{137}, and Zn^{65} were distributed homogeneously throughout the phantom, while Co^{60} was placed in the liver only.

Analyses of the complex spectra were performed by subtracting the calibrated pulse-height spectrum for each gamma emitter to be quantified. Although these spectra are obtained ideally from a subject of identical build, an approximation is obtained with the use of the plastic phantom. Computation was carried out by an IBM-704 computer. Starting with the highest energy photopeak, that of K^{40} (after correction for background and normalization of the K^{40} photopeak to that of the subject), the computer performs a channel-by-channel subtraction of the normalized K^{40} spectrum. In a similar manner, the normalized spectra for Zn^{65}, Co^{60}, and Cs^{137} were subtracted from the total spectrum (see Figure 55).

Individual 24-hr urine specimens were collected and one pooled urine sample of 20 liters. The radiochemical procedure for the Sr^{90} analysis has been described previously.[4]

RESULTS

A spectrum for an average Marshallese adult male, obtained in the 1961 study, is shown in Figure 56, with the spectrum of a member of the U.S. medical team of about the same body weight

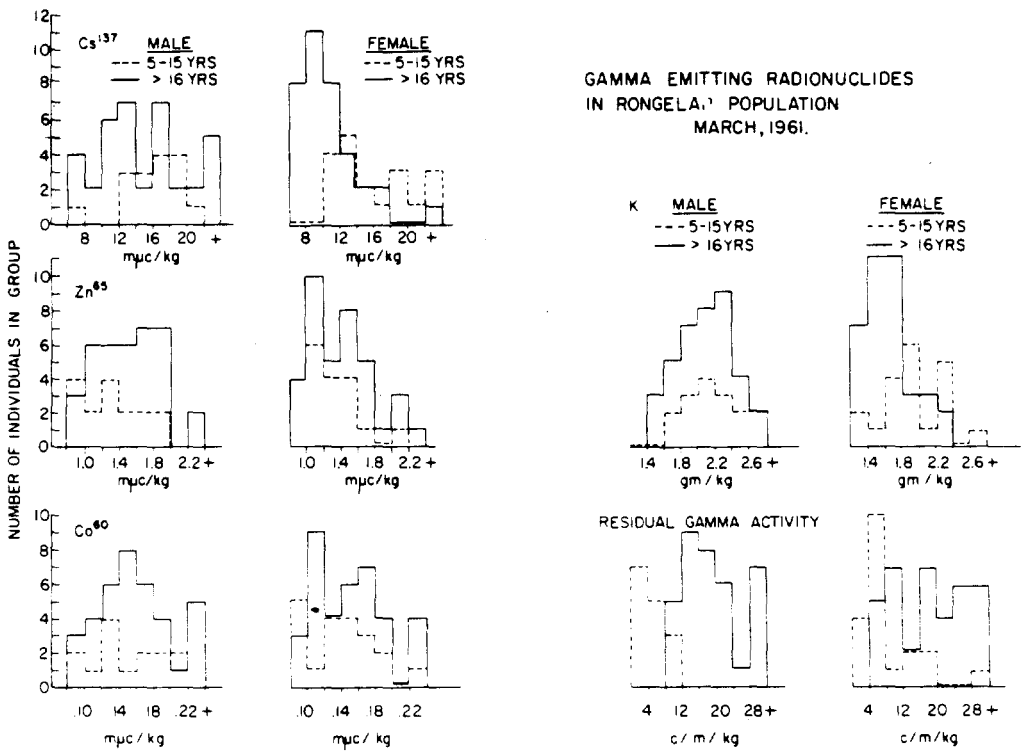

Figure 57. Frequency distribution of levels of radionuclides in various Marshallese groups.

cally significant. Also, females tend to have values lower than those found in the corresponding male group (Figure 57). No significant difference between the Zn^{65} concentrations in the exposed and unexposed groups could be determined. The percent variation within each group was approximately the same as in the case of Cs^{137}.

Potassium-40

The mean K concentrations for the younger groups were consistently higher than those for the corresponding adult groups. The male adult group averaged 2.13 g/kg body weight and the female adult group 1.63.

Cobalt-60

Analyses of the residual spectra obtained after subtraction of the three above-mentioned photopeaks revealed the presence of a fourth photopeak, Co^{60}. The levels of Co^{60} were quite low (mean $=0.148$ mμC/kg), but were nevertheless readily detectible. No significant differences between the Co^{60} levels in the exposed and unexposed groups, or on the basis of age or sex, were observed.

Total Residual Gamma Activity

The total residual gamma activity levels present after subtraction of the above four spectra are also shown in Table 17. The activity in cpm/kg has no absolute significance, but is of value in indicating, first, that there is some activity left, and, second, the nature of its distribution by groups. Members of the adult group have considerably higher levels of residual activity per unit body weight than do members of the juvenile group. No significant differences were observed, however, on the basis of sex, or between the exposed and nonexposed groups.

Radiochemical Analysis for Strontium-90

The estimated 1961 Sr^{90} mean urinary excretion in the Rongelap inhabitants was 7.2 ± 0.4 μμC/l; this represents a 14% increase over the 1959 value (6.3 μμC/l). This value is based on the results of analysis of a pooled 20.8-liter sample collected from the adult population (see Table 18). This Sr^{90} urinary excretion expressed in terms of urinary calcium is 50.6 ± 3.9 μμC/g Ca. These values give

Table 17

Summary of Radionuclide Body Burdens of Marshallese, 1961, Obtained by Whole Body Counting

	Rongelap				
	Male		Female		
	Age 5–15	Age >15	Age 5–15	Age >15	U.S. medical team
Cs^{137} (mμC/kg)					
Exposed	15.8±1.28*	16.0±2.12	16.3±1.55	11.1±0.95	
Unexposed	15.7±1.42	14.2±1.27	15.9±1.51	9.84±0.90	
Av	15.8	14.7	16.2	10.4	0.048±0.012
Zn^{65} (mμC/kg)					
Exposed	1.31±0.13	1.70±0.15	1.20±0.08	1.58±0.09	
Unexposed	1.30±0.02	1.43±0.07	1.32±0.15	1.28±0.07	
Av	1.31	1.51	1.24	1.42	0.015±0.003
Co^{60} (μμC/kg)					
Exposed	159±14.5	161±11.1	127±14.4	170±18.6	
Unexposed	152±22.6	159±11.8	155±27.3	137±8.83	
Av	156	159	137	139	6.43 ±1.39
K^{40} (g/kg)					
Exposed	2.11±0.10	2.07±0.07	2.03±0.05	1.53±0.07	
Unexposed	2.26±0.09	2.13±0.07	1.77±0.11	1.71±0.06	
Av	2.17	2.11	1.94	1.63	1.72 ±0.05
Total residual gamma activity (cpm/kg)					
Exposed	5.40±0.89	20.5±2.72	9.30±2.53	19.8±2.06	
Unexposed	4.50±1.42	20.3±1.91	8.50±2.00	18.0±1.80	2.00 ±0.59
No. of People					
Exposed	10	11	13	17	
Unexposed	6	26	7	20	
Total	16	37	20	37	7

*Standard error of mean.

and age included for comparison. Almost identical amounts of the radionuclide K^{40} are noted along with large differences in the Cs^{137} and Zn^{65} levels between the two subjects. The summary of the average levels of Cs^{137}, Zn^{65}, K^{40}, and Co^{60} determined in 1961 for both the exposed and the non-exposed Marshallese, according to age and sex, is presented in Table 17. The frequency distribution of values in various groups is shown in Figure 57. Appendix 7 contains the individual values for each subject.

Cesium-137

The mean value for all groups for body burden of Cs^{137} was 13.7 mμC/kg body weight. Although the mean levels of the exposed are slightly higher than those of the unexposed groups, the two do not differ significantly. Variation within any group is large, as can be seen from Figure 57 and from the value of the standard error. As expressed here in terms of body weight, Cs^{137} body burdens in the groups aged >16 and <16 years do not differ significantly. The mean levels of Cs^{137} in females tend to be somewhat lower than in males, but the difference does not appear significant (Figure 57).

Zinc-65

Zn^{65} body burdens per kg body weight appear to be somewhat lower in the younger population group, but again the differences are not statisti-

an estimated body-burden of 6.9 mμC as compared to 6.0 mμC estimated in 1959.

Wide variation was found in the individual "24-hour" urine samples collected and analyzed individually (Table 18). The average Sr^{90} excreted was 159 μμC Sr^{90}/g Ca for the children as compared to 30.9 for the mean adult population. Adult women appeared to have higher Sr^{90}/Ca ratios than men. However, in terms of urinary Sr^{90} (μμC/l), the value was 2.3 for women compared to 4.5 for men. This difference was similar to that observed in 1959. With the small sample size and the wide spread in values in any single group, it is not possible to detect any significant difference between the exposed and unexposed Rongelap groups.

Two coconut crabs were brought back from the survey and analyzed for Sr^{90}. One from Rongelap Island had a level of 1140 (±12) μμC Sr^{90}/g Ca, and one from the more heavily exposed island of Eniaetok, 8 miles north, had a level of 3900 (±23) μμC Sr^{90}/g Ca. These values were about half those found in these crabs in the early years after the fallout.

One 3-month fetus, the product of a miscarriage in a Rongelap woman, was also analyzed and showed a level of 25 (±20) μμC Sr^{90}/g Ca. However, the sample was so small that the results are of little significance.*

Figure 58 shows the levels of radionuclides in the Rongelap people at various times after exposure, based on radiochemical urine analyses and spectrographic determinations.

DISCUSSION

It is interesting that no significant differences were observed in the body burdens of the four gamma-emitting radioisotopes measured in the 1961 whole-body spectrographic study between the groups of Marshallese exposed to fallout in the 1954 accident and unexposed groups who have been living in the same environment for the last 4 years. The original contamination of these gamma emitters in the exposed population has already been eliminated, and what is observed in this study is the radioactivity derived from the slightly contaminated environment.

*We wish to thank Mr. E.P. Hardy, Jr., Health and Safety Laboratory, New York Operations Office, AEC, for carrying out these Sr^{90} analyses.

Table 18

Urinary Sr^{90} Values in Marshallese, 1961

Subject No.	Sr^{90}, μμC/l	Ca, g/l	Sr^{90}, μμC/g Ca
Rongelap Exposed			
Males, Age 1-15 (No Females)			
23	32.3	0.150	216
20	0.6	0.028	23
19	6.8	0.054	127
47	3.5	0.012	302
36	5.6	0.044	127
Av	9.8	0.058	159
Males, Age >15			
40	11.6	0.440	26.4
27	4.0	0.149	26.8
4	5.8	0.193	30.1
7	1.4	0.208	6.7
40	0.3	0.429	7.0
41	3.7	0.098	37.8
Av	4.5	0.253	22.5
Females, Age >15			
45	1.8	0.028	64.3
59	2.0	0.101	19.8
66	4.7	0.063	74.6
12	0.7	0.043	16.3
Av	2.3	0.059	43.8
Rongelap Unexposed			
Males, Age 1-15 (No Females)			
822	2.6	0.030	86.7
Males, Age >15 (No Females)			
944	1.1	0.182	6.0
838	3.8	0.210	18.1
838	3.1	0.215	14.7
Av	2.7	0.202	12.9

Analyses of Pooled Specimen (20.8 l)

Sample	Sr^{90}, μμC/l	Ca, mg/l	Sr^{90}, μμC/g Ca
1	7.2	145	49.3
2	6.4	149	43.2
3	6.8	142	47.6
4	7.2	141	50.7
5	7.0	131	53.3
6	8.3	139	59.7
Av	7.2±0.4	141±4	50.6±3.9

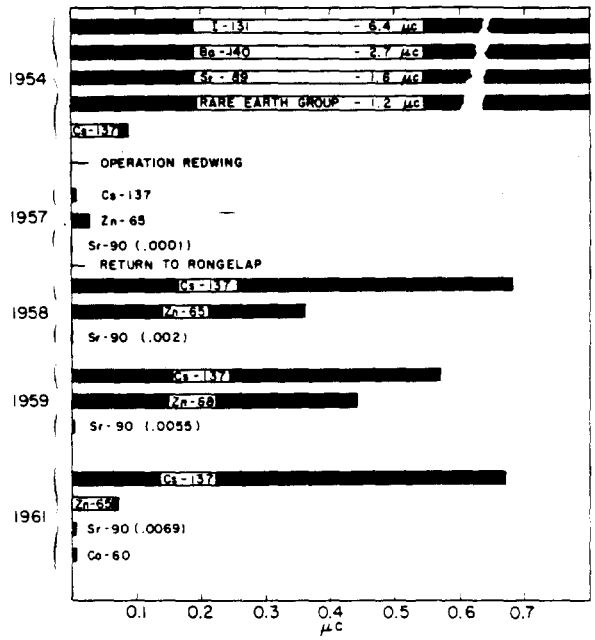

Figure 58. Estimated body burden of isotopes in Rongelap people at various times since 1954. Values obtained either by gamma spectroscopy or by radiochemical urine analyses.

The levels of radioactive contamination appeared to be about the same for juveniles as for adults, for men as for women. Factors of age and sex apparently do not influence significantly the deposition of these radionuclides. Wide variations in the level of contamination appear among individuals of all groups, as might be expected when the source of the contamination is via ingestion. Even with a fairly limited diet, individual tastes may dictate great differences in the food consumed.

As for the time course of the contamination in the population as a whole, it is necessary to consider each radioisotope individually, as to source of supply and discrimination of the soil, plant and animal life, and the human body for each isotope.

The mean Cs^{137} body burden of the Rongelap adult males is 14.7 mμC/kg as compared to 14.1 in 1959. Thus, no significant change has occurred in the past 2 years. These Cs^{137} body burdens of the Marshallese result from the relatively high dietary intake through foodstuffs produced in the Marshallese environment. They reflect the level of residual fallout on the island, and also the higher uptake and retention of Cs^{137} by food plants grown in a K-deficient soil. It appears that the body burden of Cs^{137} has reached an approximate equilibrium with the levels of Cs^{137} in the diet (although this is a difficult point to ascertain). The uniformity of the Cs^{137}/g K in the entire Marshallese population suggests that the Cs^{137} level, like the K^{40} level, is proportional to the lean body weight.

The mean Cs^{137} body burden of the Rongelap adult males (14.7 mμC/kg) was 300 times the mean of that of the medical team carrying out the study (0.048). The level of Cs^{137} in the world-wide population in July 1961 appears to have declined from the maximum level observed in 1959. The average value for Cs^{137} measured in BNL personnel, for example, declined from 59 μμC/g K in June 1960 to 30 in December 1961. The body burdens of the Marshallese will, of course, also be affected by this world-wide fallout as well as by the fallout released in the series of tests carried out by the USSR in October 1961. As the accumulation is gradual both in the environment, as world-wide fallout settles out, and in the human body, via the ingestive route, it will be a number of months before a clear picture emerges. However, after the moratorium of 3 years, during which time the Cs^{137} levels had an opportunity to stabilize, it will be clear what the direct results are of that particular series of tests.

The mean Zn^{65} concentration per kg body weight of the Marshallese does not differ significantly between the exposed and unexposed groups, nor does it differ on the basis of sex or age. However, the mean was generally slightly higher among adults than in juveniles, and higher among adult males than adult females. In the period 1959-1961 the value of Zn^{65} body burden dropped by a factor of 6. The value of Zn^{65} in male adult Marshallese dropped from 9.9 mμC/kg body weight in 1959 to 1.51 in 1961. No clear reason emerges as the basis for this drop, although it is possible that dietary variation may be responsible. It is known that the source of Zn^{65} in these people is from fish, which selectively concentrate this element. Therefore, it is possible that the decrease in Zn^{65} levels noted may be due to the fact that the people are eating more canned meats in place of fresh fish. If the Zn^{65} intake in the diet had decreased radically during that period, the observed fall in the level of internally-deposited Zn^{65} would be consistent with the effective half-life of Zn^{65}, which is $\simeq$120 days.[14] The evidence that could be obtained on this subject is insufficient to make any conclusive statement, however.

Although levels of Zn^{65} body burdens equivalent to those found in the Marshallese (1.51 mμC/kg body weight) have been observed in certain BNL reactor workers,[15] the values in the Rongelap population were about 100 times those measured in the members of the medical team. The mean level of Zn^{65} in the medical team was 0.015 mμC/kg body weight, just slightly higher than the precision of the counter, and therefore not statistically significant (Table 17).

The K content of the adult Marshallese male averages 2.12 g/kg, compared to the mean for the medical team of 1.72. The average K of a large group of male employees at BNL is 1.84 g/kg body weight.[15] The higher value for the Marshallese males reflects their well-developed musculature and low fat content, as the value of K is proportional to the lean body mass. Thus, the K values for the Marshallese females are generally lower, in the range 1.52 to 1.71 g/kg body weight. As expected, the K concentrations measured in the children are somewhat higher than those of the adults, particularly among females.

A new and very interesting finding of this study was the determination of the presence of Co^{60} in the Marshallese population. The mean level is fairly uniform, about 1/9 of the Zn^{65} level, but the spread in values in any one group is large. There is a fairly consistent correlation between the Zn^{65} and the Co^{60} values, which could very well reflect their common origin in the fish, snails, and clams of the Marshallese diet. The induced activity, Co^{60}, had not been detected previously in this population because its very low level was masked by the relatively large peaks of the other radionuclides in the relatively short counting periods hitherto used. It was to reveal just such secondary levels that the longer counting period and larger detector were adopted for the 1961 study.

The residual gamma activity in the spectra remaining after the subtraction of the major components indicates that there are some residual radionuclides present in quantities not sufficient for identification via their photopeaks. The adult groups all had approximately the same level of residual activity, while the juveniles had 25 to 50% of the adult levels. The residual activity values in the adult groups were at least 10 times the mean observed in the U.S. medical team. Unfortunately whole-body counting techniques have not been developed for external beta counting of the very important fission product Sr^{90}. Its levels can only be estimated from data obtained by radiochemical analysis of urine. Undoubtedly the bremsstrahlung resulting from the Sr^{90} body burdens in the Marshallese contributes to the residual activity noted above.

A study of the levels of body burdens of the several gamma-emitting radionuclides in the Marshallese people indicates how the fission products move through the environment and accumulate in man. Further, the biological turnover rate of these radionuclides in human beings can be estimated. The survey made with the portable whole-body counter has been invaluable in monitoring the levels of internal contamination of gamma emitters in this population. The experience gained in this study should be of value in future surveys among other populations.

Summary and Conclusions

In March 1961, a medical survey was carried out on the Marshallese people of Rongelap Atoll who had been accidentally exposed to fallout radiation 7 years previously. The medical team consisted of 10 medical specialists from the United States and 8 from the Trust Territory of the Pacific Islands. A total of 267 Rongelap people were examined, most of them on Rongelap Island and some at Kwajalein and Majuro Atolls. Of this number, 76 were in the exposed group; 28 were children of exposed parents; and 163 were adults and children from the comparison population not exposed to fallout. The Trust Territory ship *Roque* was used to transport and house the team and in addition housed, in its cargo hold, the 21-ton steel room with its electronic equipment for carrying out spectrographic analyses.

Prior to the survey, at Rongelap a village meeting held with representatives of the medical team revealed that the people were still concerned and had some misconceptions about certain conditions that they felt were related to fallout such as fish poisoning and a sickness from eating arrowroot flour. The true nature of these maladies was explained to them. Some dissatisfaction was also expressed about the continued ban on eating coconut crabs, but the people were assured that regular analyses for radioactivity would be run on the crabs and they would be notified as soon as the crabs were considered safe for consumption.

A review of the medical status of the people during the previous year revealed that few major illnesses had occurred and no epidemics of infectious diseases. No illnesses were reported in the exposed group which might be related to radiation exposure. No deaths had occurred except in two infants. One baby born of unexposed parents died of a congenital malformation (anencephaly). Only a few miscarriages had occurred and the incidence was no greater in the exposed women than in the unexposed. Thus, the suggestive trend of increased number of miscarriages and stillbirths in the exposed women reported for previous years was not apparent during the past two years.

Physical examinations showed no acute illnesses present nor any diseases which could be directly associated with radiation effects. The incidence of various diseases was generally the same in the exposed and unexposed groups. It was noted that several of the older people, particularly in the exposed group, were becoming quite feeble and helpless.

Residual skin changes in areas previously showing lesions from fallout were present with certainty in about 10 people. The changes noted were principally atrophy, scarring, and pigment aberrations. None of these cases showed any evidence of chronic radiation dermatitis or malignant change. The appearance of pigmented maculae and moles in previously irradiated areas of the skin was more common this year than noted in the past, it is believed. However, these pigmented lesions appeared quite benign. They will be scrutinized closely on future surveys. The incidence of diabetes in both exposed and unexposed people appeared to be higher than found in the United States.

Dental examinations revealed a high incidence of caries in the younger people and periodontoclasia in the older people. No difference was noted between the exposed and unexposed group in regard to dental findings.

Careful examinations for the detection of such late effects of radiation as cancer and leukemia revealed no evidence of these malignancies in any of the people, exposed or unexposed.

Growth and development data from 1958 through 1960 were reviewed for the children. These data revealed a distinct tendency for the exposed boys of ages <12 to be shorter than the unexposed males of comparable chronological ages. This was prominent in the boys exposed before 6 years of age, and most marked in those exposed at 12 to 18 months. A similar but less distinct trend was noted for body weight among the exposed boys. These trends were not noted among the exposed girls. However, skeletal maturation studies (roentgenograms of left hand and wrist) showed trends suggestive of inferior skeletal maturation in both male and female exposed children compared with unexposed children, although the differences did not reach the level of statistical significance. A high degree of correlation was found between retardation in skeletal age and inferiority in statural growth. Of specific interest was the group of children now 8 years old who had been exposed to fallout at ages 12 to 18 months; this group showed the most marked retardation in physical and skeletal growth.

Similar studies comparing growth and development in children of exposed parents and in children of unexposed parents showed that the male offspring of exposed parents were smaller in stature at all ages then those of unexposed parents. The most marked differences occurred at 1, 3, and 4 years of age, and in those born <2 years after the fallout.

In order to investigate the possible effect of radiation in inducing premature aging, various criteria were measured or estimated during physical examinations (visual acuity, accommodation, arcus senilis, graying of hair, balding, skin looseness, skin elasticity, senile skin changes, blood pressure, peripheral arteriosclerosis, and neuromuscular function). No apparent effect of radiation was noted, since measurements were not greatly different in the exposed and unexposed age groups and were similar to the measurements previously reported.[7] However, the study is of interest with regard to the measurement of biological aging, since the various criteria showed definite age dependent trends, and, by combining the scores on a percentage basis, an over-all biological age score was determined.

Slit-lamp observations for cataracts were not carried out on this survey, but previous surveys had shown no evidence of induced opacities of the lens.

Genetic studies have not been specifically conducted. Examinations of children of exposed parents have revealed little in the way of congenital anomalies. One baby born of an exposed

mother was diagnosed as having congenital heart disease and died several months after birth. The suggestive evidence of increased miscarriages and stillbirths in the exposed women during the first few years after exposure might be related to irradiation of the germ plasm. The finding that male children of exposed parents were shorter in stature than children of the same age of unexposed parents may have some genetic significance, but further studies would be necessary to substantiate such a conclusion.

Hematological studies showed that certain peripheral blood elements in the more heavily exposed Rongelap group continued to show slightly lower levels than in the unexposed comparison population, suggesting a persistent irradiation effect on hemopoietic function. Examination of the data by age and sex groups showed that neutrophil and platelet levels among the exposed were consistently below the unexposed levels, and that exposed males aged 7 to 15 years and exposed persons, both males and females, aged >40 showed the most difference from their controls. The exposed males and females of ages 15 to 40 showed less difference, with even higher levels of some elements than the unexposed groups of the same ages. The platelet levels, however, were consistently lower in all age groups in both sexes. It was noted also that male children of exposed parents had somewhat lower levels of neutrophils, lymphocytes, and platelets than children of unexposed parents.

The Rongelap population as a whole continued to show slightly lower erythropoietic function than found generally in Americans, the explanation for which is not clear. Though iron deficiency does not appear to be a problem in view of generally normal serum iron levels, some other type of nutritional deficiency may be responsible. The high incidence of eosinophilia may be partly related to chronic fungus infection of the skin and intestinal parasitism, but other causes, not immediately obvious, may be involved.

The "portable" whole-body counter was used to obtain the gamma spectra of 110 Marshallese, half of whom had been exposed to the fallout in 1954. No significant difference in body burdens of the gamma-emitting radioisotopes was observed between the Marshallese exposed to fallout in 1954 and those of the comparison population living in the same environment for the past 4 years.

The levels of internal contamination per unit weight appeared to be about the same for juveniles as for adults, male and female. Wide variations in levels of contamination in any group were found, apparently due to differences in diet and metabolism. The mean Cs^{137} body burden in adult males was 14.7 mμC/kg, which is not significantly different from the mean value of a similar group obtained 2 years ago; it was 300 times that of the medical team, who were measured at the same time for comparison. The Zn^{65} level in adult males (1.51 mμC/kg) dropped to 17% of the mean value measured 2 years ago. With a larger detector and a longer counting time than previously employed, it was possible to identify and quantify Co^{60} for the first time in these people; the mean level of Co^{60} was about 11% of the Zn^{65} level. A small amount of residual activity was still present after the subtraction of K^{40} and the above radionuclides from the total spectrum. The mean level of urinary excretion of Sr^{90} was 7.2 $\mu\mu$C/l or 14% higher than measured in the 1959 medical survey.

Though the acute effects of radiation exposure in the Marshallese have largely subsided, it is possible that certain late or delayed effects may occur. Some late effects have already been observed in the Japanese, such as an increased incidence of leukemia and possibly other malignancies and also development of cataracts. Still other late effects have been observed in irradiated animals. It is unlikely that such abnormalities will develop in the relatively small Rongelap population and, should they develop, the incidence should be quite low. Nevertheless, it is extremely important that further surveys be conducted to detect subtle changes associated with the development of such abnormalities so that they can be documented and therapeutic procedures instituted whenever possible. The examinations show some findings which persist in the exposed people, and these need to be carefully followed. These include incomplete recovery of certain blood elements to levels found in the unexposed people, retardation of growth and development in some of the irradiated children, and pigmented changes at the sites of radiation burns of the skin.

Recognizing the importance of these surveys, the Trust Territory of the Pacific Islands and the U.S. Atomic Energy Commission have agreed that annual medical surveys of the Marshallese people exposed to fallout in 1954 should continue indefinitely.

Appendices

APPENDIX 1

Rongelap Group and Control Mean Blood Counts at Various Times After Exposure

	WBC ($\times 10^{-3}$)		Neutrophils ($\times 10^{-3}$)		Lymphocytes ($\times 10^{-3}$)		Platelets ($\times 10^{-4}$)				Monocytes ($\times 10^{-2}$)		Eosinophils ($\times 10^{-2}$)	
Postexposure day	<5	>5	<5	>5	<5	>5	Male <10	Male >10	Female all ages	Total group	<5	>5	<5	>5
3	9.0	8.2	6.4	4.7	1.8	2.2	—	—	—	—	0.8	0.3	0.1	0.7
7	4.9	6.2	—	—	—	—	—	—	—	—	—	—	—	—
10	6.6	7.1	3.5	4.5	2.6	2.1	28.2	22.7	24.9	24.8	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	27.1	21.3	21.7	22.5	3.0	2.3	1.1	1.3
18	6.7	7.2	3.4	4.7	2.4	2.1	21.8	19.1	21.8	21.0	2.7	1.7	3.5	1.6
22	7.0	7.4	4.3	5.0	2.6	2.1	16.8	14.6	15.2	15.3	1.9	2.0	2.3	1.8
26	5.7	6.1	3.0	3.9	2.3	1.8	13.2	12.9	10.9	11.9	1.9	1.6	1.8	1.3
30	7.6	7.8	4.0	5.3	3.2	2.1	14.1	12.3	11.8	12.3	1.5	0.9	3.4	2.2
33	6.5	6.2	3.1	3.8	3.2	2.0	17.9	16.6	15.1	16.0	1.7	1.6	2.6	2.2
39	5.7	5.5	3.0	3.3	2.6	2.0	25.5	22.0	22.4	22.8	0.9	0.9	0.5	1.0
43	5.2	5.2	2.0	2.6	2.9	2.3	26.8	20.9	23.2	23.2	1.1	1.1	1.4	0.8
47	5.9	5.8	2.6	3.3	3.1	2.4	24.6	20.6	23.9	23.1	1.0	1.0	1.1	0.5
51	6.7	5.6	2.6	3.5	3.4	2.1	22.1	17.5	21.2	20.3	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	23.1	18.2	20.2	20.1	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	—	—	—	—	—	—	26.2	21.7	24.7	24.1	—	—	—	—
6-mo survey	8.5	6.6	4.6	4.2	3.6	2.2	24.4	20.3	23.2	22.6	1.4	1.1	2.5	1.6
1-yr survey	10.1	8.1	4.7	4.8	4.6	2.8	26.6	19.5	27.6	24.9	0.7	1.3	6.7	2.8
2-yr survey	11.8	8.6	5.9	4.8	4.7	3.1	30.0	21.4	25.5	24.7	2.7	1.5	9.6	5.3
3-yr survey	8.6	6.9	4.1	3.7	3.7	2.7	32.0	22.1	28.1	—	1.2	0.7	6.4	4.5
4-yr survey	8.9	7.5	3.3	3.4	4.6	3.6	32.5	27.1	30.8	—	1.5	1.1	7.9	4.0
5-yr survey	13.5	9.5	6.9	4.8	6.0	4.0	32.3	24.4	27.6	—	2.7	2.0	7.0	5.0
6-yr survey	—	6.5	—	3.5	—	2.7	—	—	—	—	—	0.6	—	2.7
7-yr survey	—	7.4	—	3.9	—	2.9	—	21.6*	27.3	—	—	1.0	—	3.8
Majuro controls	13.2	9.7	4.8	4.8	7.4	4.1	41.2	25.8	36.5	33.4	2.0	2.0	9.5	4.7
Rita controls, 6 mo	10.7	7.6	5.4	5.2	4.7	3.7	35.0	27.3	30.9	30.4	1.9	1.7	4.2	4.8
Rita controls, 1 yr	—	—	—	—	—	—	37.5	24.5	29.4*	27.6	—	—	—	—
Rita controls, 2 yr	14.0	8.9	7.0	4.4	5.6	3.6	35.5	24.2	31.2	29.5	1.4	1.5	12.8	6.6
Rongelap controls, 3 yr	9.8	6.9	4.0	3.4	4.7	2.9	32.6	26.9	30.0	—	1.4	0.7	6.2	4.0
Rongelap controls, 4 yr	11.2	8.0	4.0	3.6	6.2	3.7	38.8	30.7	34.0	—	2.3	1.1	7.0	4.5
Rongelap controls, 5 yr	13.7	10.1	6.2	5.2	6.2	4.1	35.8	28.0	33.6	—	3.7	2.4	6.2	6.0
Rongelap controls, 7 yr	—	7.8	—	4.2	—	3.1	—	28.5*	31.4	—	—	1.2	—	4.1

*Includes all males >7.

APPENDIX 2

Ailingnae Group and Control Mean Blood Counts at Various Times After Exposure

	WBC ($\times 10^{-3}$)		Neutrophils ($\times 10^{-3}$)		Lymphocytes ($\times 10^{-3}$)		Platelets ($\times 10^{-4}$)				Monocytes ($\times 10^{-2}$)		Eosinophils ($\times 10^{-2}$)	
Postexposure day	<5	>5	<5	>5	<5	>5	Male <10	Male >10	Female all ages	Total group	<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	22.5	22.6	20.9	21.5	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	29.0	20.2	24.6	23.9	3.7	2.6	2.3	1.4
18	6.8	7.8	2.9	5.0	3.5	2.4	27.5	21.7	24.9	24.3	2.3	1.5	3.2	2.3
22	8.9	8.7	5.3	5.4	2.7	2.9	23.5	17.0	22.9	21.3	1.5	2.4	5.8	2.4
26	8.4	7.0	4.8	4.4	3.2	2.2	20.0	13.8	17.4	16.7	2.3	2.4	0.6	1.6
30	9.6	8.6	5.3	6.2	3.7	2.0	19.5	12.8	18.2	16.8	1.9	1.9	4.1	2.0
33	7.7	7.8	3.3	5.2	3.5	2.2	24.0	15.8	22.7	17.6	2.8	2.2	6.0	1.9
39	7.5	6.2	2.9	4.2	4.7	1.9	26.5	20.8	27.0	25.2	1.1	1.7	2.7	1.6
43	6.9	6.5	2.7	3.6	3.9	2.7	28.0	19.6	25.3	24.0	0.6	1.4	2.8	0.6
47	7.3	6.7	3.5	3.8	3.4	2.7	27.0	20.0	26.1	24.5	2.2	1.9	1.5	0.7
51	8.4	6.3	3.8	3.6	4.0	2.2	32.0	18.2	25.0	23.9	2.7	2.8	2.2	1.0
54	4.6	6.3	2.8	3.5	3.2	2.5	37.0	19.8	23.8	24.2	1.5	1.9	1.8	0.8
6-mo survey	7.7	6.5	4.8	3.9	2.7	2.2	25.2	19.2	23.9	22.7	1.1	1.4	1.5	2.2
1-yr survey	11.1	7.8	4.2	4.7	6.5	5.6	38.7	21.4	28.3	27.5	1.0	1.1	1.7	2.2
2-yr survey	11.0	9.1	4.9	5.1	4.8	3.2	51.2	17.4	26.4	26.7	3.6	1.4	9.6	6.4
3-yr survey	12.1	7.0	5.5	3.9	5.6	2.6	40.8	22.4	31.2	—	3.0	0.7	5.3	3.7
4-yr survey	11.5	7.5	2.8	3.7	7.0	3.3	33.2	24.7	33.6	—	2.2	1.1	12.6	4.2
5-yr survey	—	9.7	—	5.1	—	3.7	40.9	26.3	26.8	—	—	3.2	—	6.0
6-yr survey	—	7.3	—	3.6	—	3.0	—	—	—	—	—	0.6	—	4.0
7-yr survey	—	7.7	—	4.1	—	3.1	—	25.6*	28.1	—	—	1.2	—	2.9
Majuro controls	13.2	9.7	4.8	4.8	7.4	4.1	41.2	25.8	36.5	33.4	2.0	2.0	9.5	4.7
Rita controls, 2 yr	14.1	8.9	7.0	4.4	5.6	3.6	35.5	24.2	33.2	29.5	1.4	1.5	12.8	6.6
Rongelap controls, 3 yr	9.8	6.9	4.0	3.4	4.7	2.9	32.6	26.9	3[illegible].0	—	1.4	0.7	6.2	4.0
Rongelap controls, 4 yr	11.2	8.0	4.0	3.6	6.2	3.7	38.8	30.7	34.0	—	2.3	1.1	7.0	4.5
Rongelap controls, 5 yr	13.7	10.1	6.2	5.2	6.2	4.1	35.8	28.0	33.6	—	3.7	2.4	6.2	6.0
Rongelap controls, 7 yr	—	7.8	—	4.2	—	3.1	—	28.5*	31.4	—	—	1.2	—	4.1

*Includes all males >7.

APPENDIX 3

Individual Hematological Findings, 1961

Subject No.	Age	Plate. ($\times 10^{-3}$)	WBC ($\times 10^{-3}$)	Neut. ($\times 10^{-3}$)	Lymph. ($\times 10^{-3}$)	Mono. ($\times 10^{-3}$)	Eosin. ($\times 10^{-3}$)	Baso. ($\times 10^{-2}$)	Hct., %	RBC ($\times 10^{-4}$)	Hgb., g	Serum protein, g
					Exposed Males, Age 7–15							
2	8	223 –	5.45	2.67	2.23	0.11	0.38	0.27	36	419	11.5	7.2
3	8	202	6.30	2.46	3.15	0.19	0.38	1.56	37	411	11.2	8.0
5	8	240	8.92	4.80	3.57	0.18	0.27	0.67	33			7.0
19	12	258	5.83	1.80	2.97	0.17	0.82	0.29	39	562	11.5	7.2
20	14	257	6.27	3.51	2.38	0.13	0.25	0.24	45	514	13.2	7.4
23	11	247	9.87	4.34	4.64	0	0.89	0.28	37	486	10.6	7.6
32	10	227	5.79	2.03	3.24	0.12	0.35	0.49	37	416	10.9	
36	15	221	6.91	2.97	3.52	0.07	0.35	0.24	38	429	10.6	7.8
47	15	231	6.27	4.07	2.13	0	0.06	0.16	40	423	13.2	7.4
54	8	297	6.82	4.50	1.64	0	0.61	0.31	39	432	11.2	7.4
83	6	279	10.70	6.42	3.75	0.21	0.32	0.80	35	449	11.5	7.6
85	6	208	8.57	3.34	4.10	0.09	0.94	0.41	35			7.0
	Mean	241	7.32	3.57	3.10	0.11	0.47	0.48	37.60	454	11.5	7.4
					Exposed Females, Age 7–15							
15	14	455	7.61	3.96	3.35	0.08	0.23	0.37	36	434	11.2	7.8
17	10	281	9.13	4.75	3.92	0.09	0.27	0.41	37	519	11.8	
21	10	200	5.97	3.22	2.89	0.06	0.24	0.39	39	484	11.5	7.4
33	9	325	8.79	3.69	4.66	0.18	0.26	0.53	37	435	11.2	7.6
42	10	261	9.89	6.72	2.97	0	0.20	0.82	39	427	12.1	7.6
61	15	360	11.11	5.44	5.22	0.33	0.11	0.70	41	474	11.8	8.2
65	8	262	8.79	4.57	3.34	0.26	0.53	0.55	37	447	10.9	7.6
69	11	293	6.73	2.83	3.43	0	0.47	0.45	38	425	10.6	7.2
72	13	393	7.44	4.39	2.90	0	0.07	0.44	39	451	11.5	8.2
86	6	156	8.96	5.37	2.96	0.09	0.45	0.67	34	434	10.3	7.4
	Mean	299	8.40	4.50	3.52	0.11	0.28	0.53	37.7	453	11.3	7.7
					Exposed Males, Age 16–40							
10	31	274	8.68	6.77	1.64	0.17	0.09	0.30	35	521	13.2	7.4
26	19	282	12.28	5.66	6.01	0	0.49	0.61	42			8.0
27	33	345	7.24	3.28	3.49	0.07	0.21	0.68	41	425		7.6
35	21	343	9.15	4.58	3.94	0.09	0.46	0.67	51			8.4
37	27	200	8.95	4.12	3.94	0.27	0.63	0.38	45			7.4
40	36	307	6.35	3.05	2.79	0.19	0.32	0.57	42	436	12.4	7.8
73	25	105	6.58	4.27	2.04	0.07	0.13	0.49	46			6.9
77	33	402	8.60	6.02	2.24	0	0.34	0.43	44	519		8.6
	Mean	282	8.47	4.71	3.26	0.10	0.33	0.52	43.2	475	12.8	7.8
					Exposed Females, Age 16–40							
12	25	189	5.57	3.00	2.22	0.11	0.22	0.24	38			7.4
14	32	285	8.45	7.26	0.76	0.08	0.25	0.41	35	361		7.0
18	28	381	4.48	3.14	1.12	0	0.18	0.35	38	434	10.9	7.0
22	24	228	8.53	5.29	2.30	0.09	0.68	0.42	30	331	9.4	6.4
24	20	240	6.76	4.12	2.16	0.14	0.27	0.34	38	434	11.2	7.6
39	22	254	8.78	5.36	2.72	0.26	0.44	0.64	32	385	10.0	7.2
49	22	337	10.09	4.84	4.94	0.20	0	0.55	37	385	10.3	7.4
64	37	289	7.59	4.63	2.51	0.08	0.38	0.33	39	393	11.8	7.4
66	37	118	7.89	4.66	3.08	0.08	0.0	0.29	38	396	11.2	7.0
71	35	238	7.31	4.17	2.41	0.07	0.66	0.39	41	437	12.4	7.8
74	23	289	13.97	8.24	4.19	0.28	1.26	0.98	45			8.9
	Mean	259	8.13	4.97	2.58	0.13	0.39	0.45	37.4	395	10.9	7.4

Individual Hematological Findings, 1961

Subject No.	Age	Plate. ($\times 10^{-3}$)	WBC ($\times 10^{-3}$)	Neut. ($\times 10^{-3}$)	Lymph. ($\times 10^{-3}$)	Mono. ($\times 10^{-3}$)	Eosin. ($\times 10^{-3}$)	Baso. ($\times 10^{-2}$)	Hct., %	RBC ($\times 10^{-4}$)	Hgb., g	Serum protein, g
					Exposed Males, Age >40							
4	45	184	5.09	2.14	2.80	0	0.15	0.19	45	513	14.0	7.6
7	43	278	7.12	3.35	3.13	0.14	0.50	0.39	38	431	12.1	7.4
11	57	119	4.22	1.77	2.28	0.04	0.13	0.22	41	449	11.8	7.2
46	83	265	5.81	2.27	2.79	0.12	0.58	0.17	37	392		7.6
55	82	296	4.41	1.37	2.83	0.04	0.18	0.26	32	355		7.4
68	50	159	3.84	1.88	1.73	0.04	0.19	0.30	40	434	11.8	6.8
79	46	133	7.86	3.70	3.70	0.08	0.31	0.65	47	459	14.4	7.6
80	53	210	7.74	3.64	3.72	0.15	0.23	0.27	43	450	13.2	7.6
82	57	343	6.23	2.99	2.49	0.25	0.44	0.45	41	400	11.8	7.4
	Mean	221	5.81	2.56	2.83	0.10	0.30	0.32	40.4	431	12.7	7.4
					Exposed Females, Age >40							
13	65	137	6.27	3.32	2.19	0.13	0.44	0.39	34	351	10.6	7.4
30	66	254	4.71	2.68	1.79	0.05	0.14	0.34	32	348	9.1	7.8
34	52	186	7.29	3.79	2.92	0.0	0.58	0.85	34	348	10.0	7.6
52	63	355	6.59	2.77	2.97	0.07	0.72	0.53	38	400	11.2	7.6
56	78	388	8.77	6.40	2.28	0.0	0.09	0.66	34	410		7.6
57	107	227	6.48	2.40	3.83	0.13	0.06	0.43	34	395		7.0
58	66	219	5.55	2.72	2.33	0.17	0.28	0.49	38	392	12.1	7.2
60	63	304	8.22	4.10	3.78	0.16	0.16	0.52	35	385	10.6	
63	43	143	5.12	2.15	2.46	0.05	0.46	0.13	41	431	12.1	7.2
78	44	360	5.15	2.52	2.42	0.05	0.10	0.33	39	402	11.5	7.2
	Mean	257	6.42	3.29	2.70	0.08	0.30	0.47	35.9	386	10.9	7.4
					Male Children of Exposed Parents, Age <7							
88	5	344	10.73	5.36	4.72	0.0	0.54	0.72	35	409	10.6	
89	5	384	6.02	2.35	3.21	0.12	0.18	1.14	40	437		
90	5	319	6.73	3.70	2.56	0.07	0.40	0.49	35	435	10.6	
91	5	353	5.63	1.86	3.43	0.06	0.23	0.30	34	421		
93	5	286	10.64	5.42	4.89	0.11	0.21	0.88	36	419	9.7	
96	3	462	9.13	2.65	5.57	0.09	0.82	0.23	34	403		
97	3	186	7.53	2.56	4.67	0.08	0.23	0.38	35	446		
98	3	242	10.87	5.00	5.10	0.33	0.33	0.82	35	384		
102	3	545	9.24	4.35	4.16	0.09	0.65	0.40	39	462		
104	2	337	8.82	3.79	4.76	0.0	0.26	0.66	38	475	10.6	
110	1	404	5.97	2.21	3.47	0.18	0.12	0.21	39	521		
	Mean	351	8.28	3.56	4.25	0.10	0.36	0.57	36.4	429	10.4	
					Female Children of Exposed Parents, Age <7							
87	5	293	9.57	4.12	4.60	0.10	0.77	0.41	38			
92	4	359	10.86	6.41	3.48	0.23	0.65	0.79	33	449	10.0	
94	4	450	8.50	3.74	3.82	0.09	0.77	0.43	37	446		
95	5	436	9.20	3.50	5.15	0.18	0.27	0.69	38	486	10.6	
100	5	456	8.87	5.23	3.19	0.18	0.27	0.18	35	366	10.6	
101	3	451	16.25	4.55	10.08	0.32	1.30	0.94	35			
103	2	279	10.83	4.28	6.28	0.11	0.22	0.27	35	416		
105	2	562	11.02	3.86	5.62	0.11	1.21	0.86	37	437		
107	2	420	12.60	3.66	7.44	0.38	1.13	0.44	32			
108	2	555	15.50	7.44	6.51	0.16	1.40	1.08	35			
	Mean	426	11.32	4.68	5.61	0.18	0.80	0.61	35.5	433		

Individual Hematological Findings, 1961

Subject No.	Age	Plate. ($\times 10^{-3}$)	WBC ($\times 10^{-3}$)	Neut. ($\times 10^{-3}$)	Lymph. ($\times 10^{-3}$)	Mono. ($\times 10^{-3}$)	Eosin. ($\times 10^{-3}$)	Baso. ($\times 10^{-2}$)	Hct., %	RBC ($\times 10^{-4}$)	Hgb., g	Serum protein, g
					Ailingnae Males, Age 7–15							
6	8	317	6.36	2.10	3.31	0.38	0.51	0.48	36	456	10.6	7.4
44	11	366	7.65	2.98	4.44	0	0.23	0.23	36			7.4
84	6	263	14.03	5.33	7.85	0.84	0.42	0.35				7.0
	Mean	315	9.36	3.46	5.16	0.41	0.39	0.35	36	456	10.6	7.3
					Ailingnae Females, Age 7–15							
8	8	294	6.8	2.60	3.30	0	0.83	0.54	37	403	12.1	
48	13	267	7.20	3.31	3.60	0	0.29	0.50	38	450	11.8	
53	15	326	8.85	4.51	3.98	0.09	0.18	0.40	38	417	10.9	
81	15	370	85	3.97	2.74	0.07	0.07	0.48	36			
	Mean	314	7.45	3.60	3.40	0.04	0.34	0.48	37.2	423	11.6	
					Ailingnae Females, Age 16–40 (No Males)							
45	39	169	10.27	6.78	3.08	0.21	0.21	0.46	37			7.6
51	32	390	6.38	3.00	3.19	0.06	0.06	0.37	41	414	12.4	7.4
70	24	206	4.31	2.41	1.47	0.0	0.39	0.30	26	396	7.0	7.6
	Mean	255	6.98	4.06	2.58	0.09	0.22	0.38	34.6	405	9.7	7.5
					Ailingnae Males, Age >40							
16	46	216	4.68	2.29	2.15	0.0	0.23	0.33	45	593	13.2	7.2
29	72	239	8.66	3.81	4.16	0.0	0.61	1.10	44	490		8.0
41	51	194	4.76	2.38	1.95	0.0	0.38	0.36	41	467	13.2	6.8
50	41	204	12.31	7.27	3.82	0.74	0.12	2.22	47	492		7.2
	Mean	213	7.60	3.93	3.02	0.19	0.34	1.00	44.2	511	13.2	7.3
					Ailingnae Females, Age >40							
1	61	211	6.56	2.82	3.21	0.07	0.39	0.56	42	466	13.2	8.0
41	51	194	4.76	2.38	1.95	0.0	0.38	0.36	41	467	13.2	6.8
59	41	298	13.90	11.40	2.36	0.14	0.28	0.58	35	341	10.6	7.6
	Mean	234	8.40	5.53	2.51	0.07	0.35	0.50	39.3	425	12.3	7.5
					Unexposed Males, Age 7–15							
813	7	320	4.90	1.81	2.89	0.05	0.15	0.47	35	373	10.9	6.8
814	9	256	7.72	4.79	2.39	0.23	0.31	1.20	36	453	10.6	7.2
815	11	399	5.93	3.20	2.02	0.18	0.53	0.18	36	452	10.9	7.4
818	10	384	9.17	5.32	3.39	0.18	0.28	0.58	37	447	11.2	6.8
822	15	363	9.10	7.00	1.82	0.09	0.18	0.61	41	463	11.8	7.0
869	15	374	8.35	4.34	2.92	0.17	0.92	0.44	40			8.0
887	15	251	6.52	2.74	3.33	0.07	0.37	0.41	40			7.8
892	15	314	7.79	4.13	3.35	0.08	0.23	0.34	40	415		8.0
912	8	305	6.53	2.81	2.55	0.07	1.11	0.36	31	399		7.4
913	10	272	6.19	3.28	2.54	0.06	0.31	0.34	35	481		7.4
919	13	288	6.87	3.44	2.96	0.07	0.41	0.45	35			8.2
921	7	311	11.61	6.62	4.18	0.0	0.70	1.14	37	485		7.6
931	7	344	12.92	5.69	5.17	0.13	1.94	0.65	35			6.7
939	15	198	7.81	5.07	2.19	0.16	0.31	0.43	42	484	11.5	7.0
940	12	353	12.06	6.39	4.95	0.24	0.36	1.09	39	519	11.8	8.4
981	7	116	15.75	9.13	5.99	0.16	0.47	0.91	36			7.0
	Mean	303	8.70	4.74	3.29	0.12	0.54	0.60	37.2	452	11.2	7.4

Individual Hematological Findings, 1961

Subject No.	Age	Plate. ($\times 10^{-3}$)	WBC ($\times 10^{-3}$)	Neut. ($\times 10^{-3}$)	Lymph. ($\times 10^{-3}$)	Mono. ($\times 10^{-3}$)	Eosin. ($\times 10^{-3}$)	Baso. ($\times 10^{-2}$)	Hct., %	RBC ($\times 10^{-4}$)	Hgb., g	Serum protein, g
					Unexposed Females, Age 7–15							
805	7	447	5.46	1.69	3.28	0.11	0.38	0.32	40	473	11.2	7.8
811	7	376	7.61	3.65	3.27	0.23	0.46	0.63	36	428	11.5	
812	7	465	14.20	8.23	5.11	0.28	0.57	0.75	36			7.6
816	11	379	8.90	5.97	2.32	0.27	0.36	0.43	37	387	10.6	
821	13	299	10.16	6.10	3.45	0.20	0.41	0.71	39	372	11.5	
891	13	306	8.49	5.69	2.21	0.17	0.42	0.25	39	425	12.1	7.0
909	11	392	8.92	3.30	4.28	0.27	0.98	0.56	37			7.0
911	8	294	7.35	2.94	3.68	0.29	0.44	0.51	36			7.6
912	8	305	6.53	2.81	2.55	0.07	1.11	0.36	31	399		7.4
925	11	453	6.61	3.11	3.31	0.07	0.14	0.40	37	464		8.2
926	10	248	14.17	4.82	9.21	0.0	0.14	1.04	36			8.0
937	8	176	10.50	4.94	4.62	0.21	0.74	0.61	38			8.0
946	10	353	12.15	6.32	5.22	0.24	0.24	0.89	40			
955	9	419	7.88	4.65	2.99	0.0	0.24	0.38	37	423		
959	13	343	5.16	2.12	2.53	0.10	0.41	0.27	37	387	10.6	7.6
960	9	346	7.49	2.62	4.65	0.07	0.15	0.32	35	414	10.0	8.0
962	8	398	12.44	5.97	5.35	0.37	0.75	0.53	36	452		8.0
977	15	466	6.42	3.73	2.31	0.06	0.32	0.45	35			
978	10	240	6.32	1.58	4.29	0.13	0.25	0.46	39	491		7.6
980	8	264	8.65	4.84	3.37	0.09	0.35	0.46	39	481	12.1	7.8
988	7	278	6.29	3.77	2.14	0.0	0.25	0.62	35	335		6.8
993	14	391	8.23	3.21	4.36	0.16	0.49	0.39	42	513		8.0
996	8	418	6.48	3.04	3.04	0.0	0.39	0.31	35	392		7.6
998	14	320	5.74	3.27	2.27	0.06	0.06	0.42	38	449		8.2
	Mean	349	8.42	4.10	3.74	0.14	0.42	0.50	37.1	429	11.2	7.7
					Unexposed Males, Age 15–40							
823	18	173	8.46	6.43	1.35	0.0	0.68	0.31	43	434	12.8	7.2
824	17	539	5.92	2.61	2.84	0.0	0.41	0.43	43			
828	20	206	8.95	4.56	3.94	0.18	0.27	0.33	47			7.6
830	22	277	5.32	3.30	1.75	0.11	0.16	0.27	45	481		7.0
831	21	255	7.47	3.81	3.21	0.15	0.30	0.40	47	526	13.2	7.4
833	28	204	4.17	1.42	2.59	0.04	0.08	0.22	45	550	13.6	7.2
834	27	303	8.91	4.90	0.03	0.18	0.80	0.36	48	482	14.0	7.8
838	28	298	6.85	3.22	3.22	0.07	0.34	0.21	52	536		7.0
840	31	292	6.76	3.58	2.77	0.14	0.27	0.32	50	595	14.8	8.0
842	37	279	10.37	3.63	3.63	0.21	2.91	0.50	47			7.8
845	31	279	5.92	2.37	3.25	0.12	0.18	0.28	46	477	14.0	7.0
1005	28	210	7.68	5.07	2.15	0.08	0.31	0.38	52			7.8
868	38	251	6.56	3.48	2.67	0.0	0.39	0.38	48	424		7.6
877	23	318	7.12	5.27	1.50	0.0	0.36	0.36	47			8.4
880	40	244	11.37	6.14	4.44	0.34	0.34	0.43	46			7.2
881	29	276	8.09	5.33	2.59	0.0	0.0	0.89	44	436		7.4
882	28	357	8.14	5.86	2.20	0.0	0.08	0.61	45	544		7.2
885	21	282	8.83	4.24	4.24	0.09	0.26	0.25	43	446		7.5
920	29	265	9.18	5.69	3.03	0.18	0.18	0.67	51			8.8
943	33	470	8.38	5.19	2.68	0.17	0.25	0.65	49			8.6
944	36	338	10.30	5.87	3.50	0.10	0.82	0.39	46			7.9
958	29	292	11.86	8.06	2.97	0.24	0.59	0.89	40	416	11.2	7.2
963	37	186	4.99	2.55	2.15	0.05	0.25	0.35	44	500		6.8
971	18	405	6.04	3.86	1.99	0.06	0.12	0.32	45	512	14.0	7.8
	Mean	291	7.81	4.43	2.82	0.10	0.43	0.43	46.3	491	13.4	7.6

Individual Hematological Findings, 1961

Subject No.	Age	Plate. ($\times 10^{-3}$)	WBC ($\times 10^{-3}$)	Neut. ($\times 10^{-3}$)	Lymph. ($\times 10^{-3}$)	Mono. ($\times 10^{-3}$)	Eosin. ($\times 10^{-3}$)	Baso. ($\times 10^{-2}$)	Hct., %	RBC ($\times 10^{-4}$)	Hgb., g	Serum protein, g
					Unexposed Females, Age 15–40							
825	19	355	7.30	3.50	3.58	0.15	0.07	0.35	39	413	12.1	8.4
826	24	253	8.65	4.41	3.81	0.09	0.26	0.56	40			8.6
829	22	443	5.27	3.26	1.74	0.05	0.21	0.41	33	337	9.1	8.4
832	23	241	11.46	6.99	4.01	0.0	0.46	0.29	38	456		7.6
835	27	287	6.60	2.77	3.30	0.07	0.46	0.26	40	431		7.8
841	28	317	6.95	4.10	2.43	0.0	0.35	0.59	35	387	9.7	7.6
843	32	242	5.23	4.23	0.73	0.05	0.16	0.34	36	336	10.6	6.2
865	28	277	5.14	2.72	2.06	0.10	0.21	0.40	35	342	10.9	6.6
889	36	314	7.00	4.90	1.68	0.21	0.21	0.37	36	391	11.8	8.0
895	31	311	7.41	2.74	3.48	0.22	0.96	0.48	42	451		7.0
896	21	259	6.16	2.71	2.89	0.06	0.49	0.43	35	373		7.6
914	26	276	9.20	5.70	2.94	0.09	0.46	0.40	34			6.4
916	37	244	5.48	3.67	1.59	0.05	0.16	0.30	30	294	9.4	6.2
922	37	330	5.63	3.04	2.37	0.17	0.06	0.42	36	439		8.0
932	26	370	6.88	3.10	3.03	0.14	0.55	0.61	33	343		7.6
934	26	236	6.90	3.18	3.52	0.0	0.21	0.48	37	425		7.8
938	22	167	10.15	5.38	4.47	0.10	0.20	0.68	41			8.4
950	16	529	8.65	4.07	3.81	0.09	0.61	0.55	36			
951	28	312	8.98	5.12	3.68	0.09	0.09	0.72	37			8.0
965	17	342	10.75	7.96	1.72	0.32	0.75	0.65	35			7.3
982	40	212	9.59	6.14	2.88	0.19	0.38	0.34	39	449	11.5	7.2
1001	27	236	10.83	7.58	2.60	0.11	0.43	1.01	35			6.4
	Mean	289	7.73	4.42	2.83	0.11	0.35	0.48	36.4	391	10.6	7.5
					Unexposed Males, Age >40							
849	42	314	7.02	3.30	3.09	0.14	0.49	0.41	44	485	14.0	7.4
853	56	258	4.97	2.54	2.29	0.05	0.10	0.15	44	427	12.8	
856	62	260	7.22	3.47	3.25	0.14	0.36	0.40	40	414	11.2	7.6
860	71	277	6.05	3.15	2.54	0.06	0.30	0.15	37	341	10.9	7.6
875	44	360	5.58	2.90	2.29	0.0	0.39	0.44	49	438		7.2
878	61	182	6.28	3.27	2.83	0.0	0.19	0.31	40	472		8.0
884	66	348	11.03	5.73	4.52	0.22	0.55	0.69	39			8.4
897	63	189	11.58	6.13	4.98	0.0	0.35	0.75	40			8.3
915	64	167	5.82	3.02	2.38	0.0	0.35	0.45	40	434		7.4
917	42	206	5.10	3.47	1.48	0.05	0.10	0.38	50	593	14.8	7.8
947	53	342	7.91	4.98	2.45	0.08	0.32	0.58	39	407		7.0
961	68	176	5.60	2.69	1.96	0.0	0.95	0.28	42			7.8
964	85	229	4.04	1.49	2.26	0.04	0.20	0.27	37	348		7.0
969	43	418	6.80	3.67	2.79	0.07	0.27	0.30	40	471	11.5	7.2
973	52	427	6.79	3.80	2.04	0.0	0.88	0.33	45	442		7.4
1007	50	264	6.15	3.32	2.58	0.06	0.18	0.39	39	460	11.5	8.0
	Mean	276	6.74	3.56	2.73	0.06	0.37	0.39	41.6	441	12.4	7.6
					Unexposed Females, Age >40							
851	52	245	7.56	4.31	2.72	0.15	0.38	0.26	36	387	11.2	7.2
852	57	319	10.45	3.97	4.49	0.21	1.67	0.76	37	419	10.3	7.8
858	66	227	6.97	3.90	2.79	0.07	0.21	0.40	37	410	10.0	8.2
859	68	356	9.77	3.91	5.47	0.10	0.29	0.34	40			7.8
893	43	237	5.98	2.75	2.45	0.30	0.42	0.54	36	385		7.2
894	64	304	4.67	2.99	1.45	0.09	0.14	0.37	40	399		7.8
898	63	153	11.10	5.55	4.44	0.22	0.78	1.05	38			8.0
928	48	246	4.41	1.41	2.86	0.04	0.09	0.11	33	344		7.8

Individual Hematological Findings, 1961

Subject No.	Age	Plate. ($\times 10^{-3}$)	WBC ($\times 10^{-3}$)	Neut. ($\times 10^{-3}$)	Lymph. ($\times 10^{-3}$)	Mono. ($\times 10^{-3}$)	Eosin. ($\times 10^{-3}$)	Baso. ($\times 10^{-2}$)	Hct., %	RBC ($\times 10^{-4}$)	Hgb., g	Serum protein, g
				Unexposed Females, Age >40 (continued)								
929	63	351	11.03	4.52	5.95	0.22	0.33	0.47	37			8.0
941	60	371	9.38	5.72	3.38	0.09	0.09	0.68	38			7.6
956	52	326	6.66	3.86	2.46	0.27	0.07	0.55	35	349		7.0
957	53	532	7.42	2.45	4.68	0.22	0.0	0.54	41	471		8.4
970	47	283	7.28	2.18	4.44	0.22	0.36	0.47	37	452		7.8
991	53	182	7.02	4.21	2.60	0.0	0.14	0.49	43	511	14.0	8.0
	Mean	295	7.83	3.70	3.58	0.16	0.36	0.50	37.7	413	11.4	7.8
				Male Children of Unexposed Parents, Age <7								
801	5	191	12.84	5.26	6.29	0.27	1.03	0.64	37	424	11.2	
802	5	318	8.79	3.96	4.40	0.18	0.26	0.16	41	512		
803	5	498	7.53	2.11	5.12	0.15	0.08	0.38	37	438	10.6	
806	6	586	9.78	4.50	5.08	0.0	0.10	0.62	35	449	11.2	
807	6	414	9.12	3.38	4.65	0.09	1.00	0.26	35	405	10.6	
809	5	353	8.44	4.05	3.46	0.08	0.76	0.59	37	451	10.6	
870	5	383	7.44	3.50	3.72	0.0	0.15	0.71	38	408	10.9	
904	4	384	9.41	4.61	4.42	0.38	0.0	0.71	36	419		
905	3	412	10.43	4.38	5.21	0.52	0.31	0.45	40	502		
952	4	506	16.05	9.95	5.78	0.0	0.32	0.69	31	469		
972	6	496	8.93	5.63	2.59	0.18	0.54	0.27	38	479	10.6	
1004	3	323	11.91	4.05	6.67	0.24	0.95	0.60	31	355		
1006	3	407	9.63	2.50	6.07	0.29	0.77	0.56	38	411		
1009	2	427	7.24	2.39	4.71	0.07	0.07	0.35	39	479		
1010	2	405	7.78	3.27	3.89	0.31	0.31	0.57	37	465		
1014	5	193	14.55	4.66	7.28	0.29	2.33	0.78	31			
1002	5	263	17.55	9.83	6.49	0.18	1.05	0.88	31			7.2
1017	3	341	9.06	3.25	5.25	0.18	0.36	0.45	37	435		
1020	1	384	12.04	5.06	6.38	0.0	0.60	0.30	38	435		
1024	2	195	6.82	3.27	3.14	0.07	0.34	0.33	34	368		
	Mean	374	10.26	4.48	5.03	0.17	0.57	0.52	36.1	439	10.8	7.2
				Female Children of Unexposed Parents, Age <7								
808	6	441	7.42	3.34	3.27	0.15	0.67	0.36	36	415	10.3	
810	6	351	8.05	3.38	4.35	0.0	0.32	0.52	37	481	10.6	
866	6	417	12.88	4.90	6.57	0.39	1.03	0.60	36	447		
901	4	489	15.85	9.04	6.02	0.0	0.79	1.24	34	421		
902	3	450	9.08	3.09	5.18	0.45	0.36	0.21	32	408		
903	3	339	9.21	3.68	5.16	0.09	0.28	0.32	39	446	10.6	
906	3	310	8.99	4.86	3.78	0.18	0.18	0.40	37	377	10.6	
923	6	592	8.82	4.23	3.88	0.09	0.61	0.69	37	472		
930	6	495	14.09	7.04	6.20	0.14	0.56	0.99	39	439		
954	4	359	12.12	3.52	6.06	0.24	2.18	1.15	35			
979	6	444	7.15	2.00	4.72	0.14	0.29	0.31	38	456	11.8	
992	4	393	8.59	3.78	3.70	0.26	0.77	0.73	37	473		
995	4	422	13.16	7.89	4.87	0.13	0.26	0.92	38	472	11.2	
1011	2	354	9.48	3.13	5.98	0.28	0.09	0.27	39	495	11.2	
1012	3	354	10.33	5.68	3.93	0.21	0.52	0.65	34	408	10.6	
1021	2	431	10.63	3.40	6.48	0.32	0.43	0.64	39	427		
1025	2	380	16.17	5.17	9.86	0.49	0.65	1.05	37	536		
	Mean	413	10.70	4.60	5.29	0.21	0.59	0.65	37.0	448	10.9	

APPENDIX 4

Individual WBC, Basophil, and Alkaline Phosphatase Determinations, 1961

Subject No.	WBC ($\times 10^{-3}$)	A.P., % neg.	% Baso. per 4000 cell count	Subject No.	WBC ($\times 10^{-3}$)	A.P., % neg.	% Baso. per 4000 cell count
1	6.6	74	0.85	60	8.2	90	0.63
2	5.5	74	0.50	61	11.1	63	0.63
3	6.3	73	2.48	63	5.1	80	0.25
4	5.1	85	0.37	64	7.6	85	0.43
5	8.9	0	0.75	65	8.8	45	0.63
6	6.4	95	0.75	66	7.9	1	0.37
7	7.1	84	0.55	68	3.8	92	0.78
8	6.9	88	0.78	69	6.7	78	0.67
10	8.7	88	0.35	70	4.3	86	0.70
11	4.2	91	0.53	71	7.3	91	0.53
12	5.6	94	0.43	72	7.5	86	0.60
13	6.3	61	0.63	73	6.6	77	0.75
14	8.4	10	0.48	74	14.0	51	0.70
15	7.6	93	0.48	75	10.2	48	0.48
16	4.7	67	0.70	77	8.6	67	0.50
17	9.1	94	0.45	78	5.1	84	0.65
18	4.5	82	0.78	79	7.9	60	0.83
19	5.8	89	0.50	80	7.7	69	0.35
20	6.3	79	0.38	81	6.9	50	0.70
21	6.0	59	0.65	82	6.2	59	0.73
22	8.5	58	0.50	83	10.7	56	0.75
23	9.9	78	0.28	84	14.0	52	0.30
24	6.8	63	0.50	85	8.6	87	0.48
26	12.3	88	0.50	86	9.0	0	0.75
27	7.1	86	0.95	87	9.6	15	0.43
29	8.7	90	1.27	88	10.7	42	0.67
30	4.7	90	0.73	89	6.0	75	1.90
32	5.8	91	0.85	90	6.7	64	0.73
33	8.8	70	0.60	91	5.6	59	0.53
34	7.3	91	1.17	92	10.9	6	0.73
35	9.2	68	0.73	93	10.6	10	0.83
36	6.9	91	0.35	94	8.5	39	0.50
37	8.9	71	0.43	95	9.2	67	0.75
39	8.8	45	0.73	96	9.1	67	0.25
40	6.3	87	0.90	97	7.5	73	0.50
41	4.8	88	0.75	98	10.9	0	0.75
42	9.9	84	0.83	100	8.9	60	0.20
44	7.7	82	0.30	101	16.3	38	0.58
45	10.3	0	0.45	102	9.3	49	0.43
46	5.8	79	0.30	103	10.8	50	0.25
47	6.3	89	0.25	104	8.8	24	0.75
48	7.2	89	0.70	105	11.0	54	0.78
49	10.1	31	0.55	107	12.6	87	0.35
50	12.3	65	1.80	108	15.5	69	0.70
51	6.4	62	0.58	110	6.0	34	0.35
52	6.6	74	0.80	801	12.9	21	0.50
53	8.8	0	0.45	802	8.8		0.18
54	6.8	63	0.45	803	7.5	63	0.50
55	4.4	88	0.60	805	5.5	60	0.58
56	8.8	79	0.75	806	9.8	63	0.63
57	6.5	93	0.67	807	9.1	72	0.28
58	5.6	84	0.88	808	7.4	88	0.48
59	13.9	16	0.42	809	8.4	80	0.70

Individual WBC, Basophil, and Alkaline Phosphatase Determinations, 1961

Subject No.	WBC ($\times 10^{-3}$)	A.P., % neg.	% Baso. per 4000 cell count	Subject No.	WBC ($\times 10^{-3}$)	A.P., % neg.	% Baso. per 4000 cell count
810	8.1	82	0.65	889	7.0	85	0.53
811	7.6	84	0.83	891	8.5	45	0.30
812	14.2	78	0.53	892	7.8	95	0.43
813	4.9	86	0.95	893	6.0	80	0.90
814	7.7	72	1.55	894	4.7	82	0.80
815	5.9	91	0.30	895	7.4	81	0.65
816	8.9	89	0.48	896	6.2	87	0.70
818	9.2	84	0.63	897	11.6	91	0.65
819	6.7	56	0.55	898	11.1	80	0.95
820	5.7	90	0.48	901	15.9	38	0.78
821	10.2	76	0.70	902	9.1	57	0.23
822	9.1	79	0.67	903	9.2	2	0.35
823	8.5	81	0.37	904	9.4	81	0.75
824	5.9	80	0.73	905	10.4	60	0.43
825	7.3	91	0.48	906	9.0	80	0.45
826	8.7	75	0.65	909	8.9	96	0.63
828	9.0	66	0.37	911	7.3	57	0.70
829	5.3	67	0.78	912	6.5	73	0.55
830	5.3	87	0.50	913	6.2	89	0.55
831	7.5	68	0.53	914	9.2	79	0.43
832	11.5	83	0.25	915	5.8	47	0.78
833	4.1	87	0.53	916	5.5	72	0.55
834	8.9	50	0.40	917	5.1	93	0.75
835	6.6	80	0.40	919	6.9	70	0.65
838	6.9	62	0.30	920	9.2	49	0.73
840	6.8	78	0.48	921	11.6	31	0.98
841	6.9	64	0.75	922	5.6	77	0.75
842	10.4	75	0.48	923	8.8	58	0.78
843	5.2	0	0.65	925	6.6	89	0.60
844	8.7	16	0.38	926	14.2	60	0.73
845	5.9	92	0.48	928	4.4	68	0.25
849	7.0	74	0.58	929	11.0	83	0.43
851	7.6	85	0.35	930	14.1		0.70
852	10.4	82	0.73	931	12.9	87	0.50
853	5.0	76	0.30	932	6.9	60	0.88
856	7.2	86	0.55	934	6.9	74	0.70
858	7.0	7	0.58	937	10.5	52	0.58
859	9.8	93	0.35	938	10.2	59	0.67
860	6.0	81	0.25	939	7.8	91	0.55
865	5.1	65	0.78	940	12.1	0	0.90
866	12.9	74	0.48	941	9.4	91	0.73
868	6.6	87	0.58	943	8.4	79	0.78
869	8.4	97	0.53	944	10.3	89	0.38
870	7.4	64	0.95	946	12.2	81	0.73
875	5.6	92	0.78	947	7.9	46	0.73
877	7.1	66	0.50	949			0.67
878	6.3	99	0.50	950	8.7	93	0.63
880	11.4	89	0.38	951	9.0	71	0.80
881	8.1	67	1.10	952	16.0	63	0.43
882	8.1	34	0.75	954	12.1	0	0.95
884	11.0	93	0.63	955	7.9	59	0.48
885	8.8	74	0.28	956	6.7	88	0.83
887	6.5	98	0.63	957	7.4	91	0.73

Individual WBC, Basophil, and Alkaline Phosphatase Determinations, 1961

Subject No.	WBC ($\times 10^{-3}$)	A.P., % neg.	% Baso. per 4000 cell count	Subject No.	WBC ($\times 10^{-3}$)	A.P., % neg.	% Baso. per 4000 cell count
958	11.9	31	0.75	992	8.6	53	0.85
959	5.2	94	0.53	993	8.2	86	0.48
960	7.5	83	0.43	995	13.2	52	0.70
961	5.6	93	0.50	996	6.5	80	0.48
962	12.4	98	0.43	998	5.7	92	0.73
963	5.0		0.70	1001	10.8	13	0.93
964	4.0	86	0.67	1004	11.9	81	0.50
965	10.8	46	0.60	1005	7.7	74	0.50
967	6.5	91	0.50	1006	9.6	83	0.58
969	6.8	84	0.58	1007	6.1	93	0.63
970	7.3	82	0.65	1009	7.2	82	0.48
971	6.0	93	0.53	1010	7.8	51	0.73
972	8.9	67	0.30	1011	9.5	0	0.28
973	6.7	77	0.48	1012	10.3	51	0.63
977	6.4	75	0.70	1014	14.6	23	0.53
978	6.3	91	0.73	1015	17.6	71	0.50
979	7.2	65	0.43	1017	9.1	90	0.50
980	8.7	5	0.53	1020	12.0	40	0.25
981	15.8	69	0.58	1021	10.6	46	0.60
982	9.6	90	0.35	1024	6.8	55	0.48
988	6.3	63	0.98	1025	16.2	65	0.65
991	7.0	89	0.70				

APPENDIX 5

Gamma-Globulin Groups of Rongelap Islanders
(Blood Collected 1959)

A.G. Steinberg and B.S. Blumberg

Subject No.	Gm groups	Subject No.	Gm groups	Subject No.	Gm groups	Subject No.	Gm groups
1	a+b+x−c+	52	a+b+x−c−	834		890	
	a+b+x−c+	53	a+b+x−c−	835		891	a+b−x−c−
2	a+b+x−c+	54		836		892	
3		55	a+b+x−c+	837		893	a+b+x−c+
4		56	a+b+x−c+	838	a+b+x−c+	894	a+b+x−c+
5	a+b+x−c−	57	a+b+x−c−	839	a+b+x−c−	895	a+b+x−c+
6		58	a+b+x−c−	840	a+b+x−c−	896	a+b+x−c+
7	a+b+x−c+	59		841	a+b+x−c+	898	
8		60		842		899	
9	a+b+x−c−	61	a+b+x−c−	843	a+b+x−c−	908	a+b+x−c−
10		62		844	a+b+x−c+	911	
11	a+b+x−c−	63	a+b+x−c+	845	a+b+x−c−	912	a+b+x−c+
12		64		846	a+b+x−c+	913	a+b+x−c−
13	a+b+x−c+	65	a+b+x−c−	849	a+b+x−c+	914	a+b+x−c+
14	a+b+x−c+	66	a+b+x−c+	850	a+b+x−c−	916	a+b+x−c+
15	a+b+x−c+	67		851	a+b+x−c+	921	a+b+x−c−
16	a+b+x−c+	68	a+b+x−c−	852	a+b+x−c−	922	a+b+x−c+
17	a+b+x−c−	69	a+b+x−c−	853		925	a+b+x−c−
18	a+b+x−c+	70	a+b+x−c−	854	a+b+x−c+	927	
19		71	a+b+x−c+	855	a+b+x−c−	928	a+b+x−c+
20		72		856	a+b+x−c−	932	a+b+x−c+
21		73		857		933	a+b+x−c+
22		74		858	a+b+x−c+	934	a+b+x−c+
23	a+b+x−c+	75		859	a+b+x−c−	935	a+b+x−c+
24		76	a+b+x−c−	860	a+b+x−c+	939	a+b+x−c−
25		77	a+b+x−c+	861	a+b+x−c−	940	a+b+x−c+
26		78	a+b+x−c−	862	a+b+x−c−	944	a+b+x−c−
27		79	a+b+x−c−	863	a+b+x−c+	947	a+b+x−c−
28	a+b+x−c+	80		864	a+b+x−c−	953	a+b+x−c+
29	a+b+x−c−	81	a+b+x−c−	865		955	a+b+x−c−
30	a+b+x−c−	82	a+b+x−c−	867		956	a+b+x−c+
31			a+b+x−c−	868	a+b+x−c−	958	a+b+x−c+
32		814		869	a+b+x−c+	959	a+b+x−c−
33	a+b+x−c−	815	a+b+x−c+	871	a+b+x−c+	960	a+b+x−c−
34		816		872	a+b+x−c−	963	a+b+x−c−
35		817		873	a+b−x−c−	966	a+b+x−c−
36	a+b+x−c−	818	a+b+x−c−	874	a+b+x−c−	967	
37		819		875	a+b+x−c−	968	
38		820	a+b+x−c+	876	a+b+x−c−	969	a+b+x−c−
39	a+b+x−c−	821		877		971	a+b+x−c−
40	a+b+x−c−	822		878	a+b+x−c−	973	
41	a+b+x−c+	823	a+b+x−c+	879		976	
42	a+b+x−c+	824		880	a+b+x−c−	977	a+b+x−c−
43	a+b+x−c+	825		881	a+b+x−c+	978	a+b+x−c−
44		826		882	a+b+x−c+	980	a+b+x−c−
45	a+b+x−c−	827	a+b+x−c+	883		989	
46	a+b+x−c−	828		884		991	a+b+x−c+
47	a+b+x−c−	829	a+b+x−c−	885		993	a+b−x−c−
48	a+b+x−c−	830	a+b+x−c−	886		996	a+b+x−c−
49	a+b+x−c+	831	a+b+x−c+	887	a+b+x−c−	1003	a+b+x−c−
50	a+b+x−c+	832	a+b+x−c−	888		1005	a+b+x−c−
51		833	a+b+x−c+	889	a+b+x−c−		

APPENDIX 6

Individual Physical Examination Findings, 1961

NO. AGE, SEX	PAST HISTORY	INJURIES	WEIGHT POUNDS	BLOOD PRESSURE HEART AND LUNGS	EENT	ABDOMEN GU OR GYN
1 61 F	Obesity, Deafness, Mild hypertension	Fixation, left elbow, trauma	163 mod. obesity	156/90 hypertension		mild cystocele;
4 45 M	Substernal pain, 1 year; slight hypertension	Traumatic Amputation, distal phalanx, lt. 2nd. finger	154	130/80		
7 43 M			126	116/68		
10 31 M	Chronic lower abdominal pain		133	118/70		
11 57 M	Kahn 3+	Scarring due to burn, rt. shoulder, lt. chest; fracture rt. tibial head(old)	113 thin	150/100 mild hypertension	3+4 retinal arteriosclerosis, strabismus	
12 25 F	Hydatiform mole, bleeding, 1957		136	120/76		Cervical prolapse
13 65 F	Cough, sputum, backache, "broke back" '54		75 losing weight	100/66 exp. rales rt. chest, difficult resp.		2X2 cm. hard mass, left labia. kraurosis vaginae
14 32 F		Scar, rt. antecubital space	142 preg.	96/60		Liver down 1 cm. pregnant 7 mo. Hist. of cystocoele
16 46 M			127	118/80		
18 28 F			103	106/78		3 mo. post-partum left ovary palp, erosion cervix

SKIN	TUMORS	CONGENITAL ABNORMALITIES	MISCELLANEOUS	BLOOD COUNT	OTHER LAB. DATA. URINE PAP. ETC.	COMMENTS
				WBC 6.6 Hct. 42 Hgb. 13.2 Plat. 211	Pap. superficial squamous. Dry smear; Neg. for Ca	acc. lung marking rt. mid. chest.
Bl. macule rt. neck	Fibroma mid-line D_6			WBC 5.1 Hct. 45 Hgb. 14.0 Plat. 164		R_x trial of nitroglycerine
	small tumor lt. buttock ? lipoma		abcess left thigh; tender inguinal nodes	WBC 7.2 Hct. 38 Hgb. 12.1 Plat. 278		
Bl. macule rt. ant. axil.	Lipoma 5X5 cm. left shoulder			WBC 8.7 Hct. 35 Hgb. 13.2 Plat. 274		R_x antipar
Depig. scalp (fungus); raised moles axil.			Prostate sl. enlarged	WBC 4.2 Hct. 41 Hgb. 11.8 Plat. 119		Chest film taken
				WBC 5.6 Hct. 38 Hgb. Plat. 189	Pap: good estrogen level; Neg. Ca; Trichomonas	
				WBC 6.3 Hct. 34 Hgb. 10.6 Plat. 137	Pap: scanty smear, Neg. Ca	R_x Vitamins. chest film taken. Resp. difficulty due to osteoarthritis
		Wrist, abn. wide distal epiphyses		WBC 8.5 Hct. 35 Hgb. — Plat. 285		R_x Iron and Calcium
			Vib. sense absent, lower extremities	WBC 4.7 Hct. 45 Hgb. 13.2 Plat. 216		
Biopsy scar back neck, sl. scarring rt. neck				WBC 4.5 Hct. 38 Hgb. 10.9 Plat. 381	Pap: Neg. Ca, severe inflammation, Trichomonas	R_x Iron

NO. AGE, SEX	PAST HISTORY	INJURIES	WEIGHT POUNDS	BLOOD PRESSURE HEART AND LUNGS	EENT	ABDOMEN GU OR GYN
22 24 F	Cough, sputum		98	96/60		Preg. 4 mo.
24 20 F	Cough sputum		97	100/60		Lactating
27 33 M			139	110/70	Tonsilar	External
28 75 F			111 slight obesity	170/84 sys M transmitted aortic area	4± retinal arteriosclerosis, bilateral lens opacities	
29 72 M	Occ. asthmatic attack. History stroke, 1956		121	130/75 wheezes both bases	4± retinal arteriosclerosis, bilateral lens opacities	
30 66 F	Scratching continuously		92	216/120 hypertension		Liver down 2 cm. Area of ulceration 10 cm. above anus on procto., bleeds easily.
34 52 F	Pan-hysterectomy 1954		121	100/86		
35 21 M			129	100/74		
39 22 F		9 mo. pregnant	116 1/2	96/66		Preg. 9 mo. del. 3/26/61

SKIN	TUMORS	CONGENITAL ABNORMALITIES	MISCELLANEOUS	BLOOD COUNT	OTHER LAB. DATA. URINE PAP. ETC.	COMMENTS
				WBC 8.5 Hct. 30 Hgb. 9.4 Plat. 228		R_x Iron
Bl. macules rt. & lt. neck, also two macs.rt.forearm. Depig.spot dorsom, rt. foot				WBC 6.8 Hct. 38 Hgb. 11.1 Plat. 240		
2 bl. macule upper back near nec,				WBC 7.1 Hct. 41 Hgb. — Plat. 345		
		Prominent heads of ulnae	Sl. fever, Tachycardia		Pap: some endocervical atypia REPEAT	Light perception only due to cataracts. Almost blind, Rec. cat. removal
			Prostate 2X normal	WBC 8.7 Hct. 44 Plat. 239		Light perception only due to cataracts. Senile cerebral arteriosclerosis
Roughening back neck (Fungus Inf.?)				WBC 4.7 Hct. 32 Hgb. 91 Plat.254	Pap: Neg. for Ca. Rectal lesion smear showed inflammation	Cerebral arteriosclerosis. Redo proctoscopic
				WBC 7.3 Hct. 34 Hgb. 10.0 Plat. 186	Pap: Neg. for Ca. Dry smear	
				WBC 9.2 Hct. 51 Hgb. — Plat. 343		
Pig. Var. Roughening back neck. macule 1 ea. hand lt. ant. cub. fossa. sl. pig. var. rt. ft. and mac dors.lt. ft.				WBC 8.8 Hct. 22 Hgb. 10.0 Plat. 254		

AGE,	NO.	SEX	PAST HISTORY	INJURIES	WEIGHT POUNDS	BLOOD PRESSURE HEART AND LUNGS	EENT	ABDOMEN GU OR GYN
40	36	M	–	Deformity rt. forefinger	---	104/68		
41	51	M		Scar, left inguinal region	120	104/66 Tachycardia		
45	39	F	Asthma		99 1/2	120/80		Cervical laceration endoc.
46	86	M	Residual rt. hemeplegia, heart. block	1-2 toe rt. foot missing	129	220/66 Arteriosclerosic heart disease. aortic stenosis insufficiency bradycardia	Bilateral cataracts.4+ retinal arteriosclerosis	Gynecomastia
49	22	F	3 previous abortions	scar rt. su. .- clavical area	156 obese	96/66		2 mo. preg.
50	51	M	Bilateral hallux valgus		182 obese	116/80		
51	32	F	Episode abd.pain? Mittelschmerz		94	118/60		
52	63	F	Pain rt. shoulder		107	104/72	2+ ret. arteriosclerosis	
55	82	M			134	114/70	Bilateral cataracts light perception only	Moderate gynecomastia
56	78	F				140/70 aortic insufficiency	Bilateral cataracts	
57	107	F		Subcutaneous masses both hips		130/70		

SKIN	TUMORS	CONGENITAL ABNORMALITIES	MISCELLANEOUS	BLOOD COUNT	OTHER LAB. DATA. URINE PAP. ETC.	COMMENTS
Raised mole back neck			Lt. Ing. node 1 cm. Inf. lt. hand, buttock	WBC 6.4 Hct 42 Hgb 12.4 Plat 303		
2 macule rt. neck, 1 lt. neck, 1 foot	Lump, lateral aspect left arm, ? infection site	Congenital dislocation hips		WBC 4.8 Hct 41 Hgb 13.2 Plat. 194		Does not look well
				WBC 10.7 Hct. 37 Plat. 169	Pap: Neg. Ca. scanty smear	
				WBC 5.8 Hct 37 Plat. 265		
macule neck lace area, part. rt. neck; fungus leg				WBC 10.1 Hct 37 Hgb 10.3 Plat. 337		Check Cholesterol
				WBC 12.3 Hct 47 Plat. 204		
Macule lt. neck				WBC 6.4 Hct 41 Hgb. 12.4 Plat. 390	Pap: neg., inflammation	
Raised moles side neck			Osteoarthritis, Scoliosis to right	WBC 6.6 Hct. 38 Hgb. 11.2 Plat. 355	Pap: Neg dry smear	
			Osteoarthritis, Positive Romberg, ? tabes dorsalis	WBC 4.4 Hct 32 Plat. 296		R_x Poor vision Penicillin Vitamins - Do bone marrow
Sebaceous cysts, face		large heads ulnae	Severe Osteoarthritis, kyphoscoliosis	WBC 8.8 Hct 34 Plat. 388		Unable to stand, cerebral arterioscleros is
Hyperkerotic areas rt. chest	Subcutaneous masses both hips		Kyphoscoliosis to right. Severe osteoarthritis	WBC 6.5 Hct 34 Plat. 227		Light perception only, x-ray of hip masses not diagnostic

AGE,	NO.	SEX	PAST HISTORY	INJURIES	WEIGHT POUNDS	BLOOD PRESSURE HEART AND LUNGS	EENT	ABDOMEN GU OR GYN
58	66	F			105	140/80	Early cataracts	Lacerated cervix blood in smear
59	41	F			88	140/76		Lacerated cervix blood in smear
60	63	F			160 obesity	220/110 hypertension; sys M,art. heart disease	2+ retinal arterio-sclerosis	
63	43	F			110	120/72		Cerv. erosion, rt. ovary palp., vaginal prolapse
64	37	F			132	120/70		cerv. erosion, vag. prolapse
66	37	F			140	108/72		
68	51	M			127	112/70		
70	24	F	Anemia in past		120	110/64 sys M		Indurated rt. adnexa
71	35	F			131 slightly obese	126/80		Abd. rigid, Leukorrhea laceration cerv 4 o' clock
73	25	M			161	138/86 slight hypertension		
74	23	F			173 obese	120/80	Lt. tonsilar hypertrophy	Cervical erosion

SKIN	TUMORS	CONGENITAL ABNORMALITIES	MISCELLANEOUS	BLOOD COUNT	OTHER LAB. DATA. URINE PAP. ETC.	COMMENTS
Few raised moles around neck				WBC 5.6 Hct 38 Hgb. 12.1 Plat. 219	Pap:? Neg, Trichomonas, marked inflammation	Old pleuritic thickening diaphragmatic area.
Hyperker. Back neck				WBC 13.9 Hct 35 Hgb 10.6 Plat. 298	Pap: Neg Ca, marked inflammation, sl. atrophia	
Chronic Pyoderma			mild osteo-arthritis	WBC 8.2 Hct 35 Hgb 10.6 Plat. 304	Pap: blood inflammation	
Pig. var. back lt. neck; pig. rt. forearm 5 cm. from ant. cub. fossa				WBC 5.1 Hct 41 Hgb. 12.1 Plat. 143	Pap: Neg. Ca, blood inflammation	
raised mole back neck				WBC 7.6 Hct. 39 Hgb. 11.8 Plat. 289	Pap: Neg. Ca., marked inflammatory changes. Dry smear	
2 macule back neck				WBC 7.9 Hct. 38 Hgb. 11.2 Plat. 118	Pap: Neg. Urine sugar 2+	Rx Fasting blood sugar
Depig. area dorsum lt. ft. (?)			small rt. axillary node	WBC 3.8 Hct. 40 Hgb. 11.8 Plat. 159		
				WBC 4.3 Hgb 7.0 Hct 26 Plat. 206 Anemic	Pap: Neg. Ca., ? Trichomonas	Anemia Rx Iron
Few macule rt. neck				WBC 7.3 Hct 41 Hgb 12.4 Plat. 238	Pap: endocervical atypia. Trichomonas REPEAT	
sl. hyperker. between 1st + 2nd. toes both feet				WBC 6.6 Hct. 46 Plat. 105		
Mole lt. neck; few macule dorsum lt. ft.; inf. lt. big toe				WBC 13.9 Hct. 45 Plat. 289	Pap: Neg. Ca atypia clusters of benigh endometrial cells	Constipated

AGE,	NO.	SEX	PAST HISTORY	INJURIES	WEIGHT POUNDS	BLOOD PRESSURE HEART AND LUNGS	EENT	ABDOMEN GU OR GYN
75	18	F	Lost baby 4 mo. congenital heart?			110/70 sys M over aortic area		
77	33	M			113	126/76		
78	44	F			152 mod. obesity	120/78		small laceration, cervix
79	46	M		Bilateral incisions, groins		134/82	Enlarged parotid, rt.	
80	53	M		Left toe deformed		130/80 NSR fibrillation, pulse irregular 58		
82	57	M			119	112/70		
825	19	F			111	108/66		Liver down 1 cm, lactation sl. erosion cervix
826	21	F	dizziness and weakness, 1 mo. stillbirth '59		95	not taken	tonsilar hypertrophy	Lactation, cervical erosion mild
827	24	F						
828	20	M		Scars around knees	113	110/64		
829	22	?			116	110/72		Lactating, cervical laceration
830	22	M			139	116/66		

SKIN	TUMORS	CONGENITAL ABNORMALITIES	MISCELLANEOUS	BLOOD COUNT	OTHER LAB. DATA. URINE PAP. ETC.	COMMENTS
Few sm. macules back neck; multiple inf. lefs ? yaws		Short 5th. fingers bilateral		WBC 10.2 Hct. 41 Plat. 207	Pap: Neg. Ca., good estrogen level	
Absent fingers and toes with ulcers, of extremeties and soles of feet				WBC 8.6 Hct. 44 Plat. 402		Leprosy arrested
pig. var. and roughening back neck; raised bl. moles both sides neck part. left				WBC 5.2 Hct. 39 Hgb. 11.5 Plat. 360	Pap: Neg. Ca., cervical cell atypia, marked inflammation	
Scar rt. ear with pigmented white nodules; spotty alopecia back head; CH scar back lt. Head			Left inguinal node enlarged	WBC 7.9 Hct. 47 Hgb. 14.4 Plat. 133		
Irregular pig. front neck (fungus?) 1 macule lt. ant. cub. fossa; pig face with macules.				WBC 7.7 Hct. 43 Hgb. 13.2 Plat. 210		X-ray-sl. enlarged heart. emphesema
			Prostate 2λ normal, left testis 3λ right, left facial nerve impaired.	WBC 6.2 Hct. 41 Hgb. 11.8 Plat. 343		
				WBC 7.3 Hct. 39 Hgb 12.1 Plat. 355		
		Patellar move laterally, flexion deformity of fingers		WBC 8.7 Hct. 40 Plat. 253	Pap: Neg., good estrogen level	
Pigment. back of neck			Tiny axillary nodes	WBC 8.9 Hct. 47 Plat. 206		
				WBC 5.3 Hct. 33 Hgb. 9.1 Plat. 443 Anemic		R_x Iron
			Small axillary node	WBC 5.3 Hct. 45 Plat. 277		

NO. AGE, SEX	PAST HISTORY	INJURIES	WEIGHT POUNDS	BLOOD PRESSURE HEART AND LUNGS	EENT	ABDOMEN GU OR GYN
831 21 M	—		121	110/56	Tonsilar hypertrophy	Guarding RLQ ?irritated intestine
832 23 F	Left ovarian cyst, history 1958		98	116/74		L.L.Q. mass, slightly tender ? cyst
833 28 M	Uniform pigmentation hands and feet		139	118/76 sys M over aortic area	Tonsilar hypertrophy	
834 27 M			121	120/70		
835 27 F			90	112/60		Left breast > ovary palp., ? thickened cervix
836 28 M			126 1/2	116/70		
838 29 M			145 slightly obese	130/80		
839 33 F	Fibroma uterus, by history				Congenital nystigmus 1959	
840 31 M			151	112/68		
841 28 F	Child died two days after birth 3/28/60		141	118/80		13 days post partum
842 37 M		Rt. hand little finger missing; left hand index finger missing	141	120/80		
843 32 F	History of yaws. Old retinal hemorrhage, 1958		126	100/68 Split S2		9 mo. pregnan

SKIN	TUMORS	CONGENITAL ABNORMALITIES	MISCELLANEOUS	BLOOD COUNT	OTHER LAB. DATA. URINE PAP. ETC.	COMMENTS
		—		WBC 7.5 Hct. 47 Hgb. 13.2 Plat. 255		
		Short fifth fingers		WBC 11.5 Hct. 38 Plat. 241	Pap: Neg., good estrogen level	
				WBC 4.2 Hct. 45 Hgb. 13.6 Plat. 204		
				WBC 8.9 Hct. 48 Hgb. 14.0 Plat. 303		
				WBC 6.6 Hct. 40 Hgb.--- Plat. 287	Pap: Neg., very marked inflammation	
				WBC 6.9 Hct. 52 Plat. 298		
				WBC 6.8 Hct. 50 Hgb. 14.8 Plat. 292		
Few macule back neck; some pig.				WBC 6.9 Hct. 35 Hgb. 9.7 Plat. 317 Anemic		
				WBC 10.4 Hct. 47 Plat. 279		
				WBC 5.2 Hct. 36 Hgb. 10.6 Plat. 242		$\mathbb{R}_x$ Iron

NO. AGE, SEX	PAST HISTORY	INJURIES	WEIGHT POUNDS	BLOOD PRESSURE HEART AND LUNGS	EENT	ABDOMEN GU OR GYN
844 42 F			115 1/2	130/84		Liver down 3 cm.
845 31 M			140	114/70		
846 38 F	Syphilis?					
849 42 M	Depigmented Areas		191 obese abd.	120/70 ? pulm. sys M		Hemorrhoids
851 52 F	Hypertension(?) 150/96 1959 (?) cerebral polyp		166 obese	120/70		Cervical laceration and erosion, bleeds easily
852 57 F	Hypertension (?)		116 108(1959)	118/78 spl. 2nd. sound.sys. M.		
853 56 M			160	112/84	Cataracts 2 cm. nodule	Liver 2 cm.
856 54 M			124 124(1959)	140/80		
858 66 F	Hypertension Osteoarthritis Cervix discharge Goitre, fixed chest		109 114 (1959)	160/90		Whitish Discharge, cervix small
859 68 F	Mild hypertension		128 117 (1957)	150/96	Rt. corneal ulcer	Lacerated cervix
860 71 M	Acromegaly? 1957 ext. hemorrhoids	Burn-severe scar, rt. arm and elbow; old fracture rt. lower arm	121 130 (1959) 123 (1957)	110/66	Cataracts, blindness left eye	

SKIN	TUMORS	CONGENITAL ABNORMALITIES	MISCELLANEOUS	BLOOD COUNT	OTHER LAB. DATA. URINE PAP. ETC.	COMMENTS
				WBC. 8.7 Hct. 40 Hgb. 11.2 Plat. 381	Pap: neg, marked inflammation and blood	
Few macule around neck and ant.cub.fossa. vesicle right buttock				WBC 5.9 Hct. 46 Hgb. 14 Plat. 276		
				WBC 7.0 Hgb. 14.0 Plat. 314		
				WBC 7.5 Hct. 36 Hgb. 11.2 Plat. 245		
				WBC 10.5 Hct. 37 Hgb. 10.3 Plat. 319		
Vitiligo, trunk	Lipoma, rt. thigh 6 X 6 cm.		2 cm. nodule lt. lobe thyroid	WBC 5.0 Hct. 44 Hgb. 12.8 Plat. 258 Diabetes	R_x	Diabetes, hypertension, Sugar 4+ Fasting blood sugar. Cholesterol
			bil. 1/2 cm. node, axillae	WBC 7.2 Hct. 40 Hgb. 11.2 Plat. 260		
		Bilateral enlargement of ulna	Osteoarthritis, 6 X 5 cm. goitre, rt.	WBC 7.0 Hct. 37 Hgb. 10.0 Plat. 227	Pap: Neg. for malignant cells. Fairly good estrogen level	
		Short left 4th. toe	Kyphosis, osteoarthritis rt. shoulder, fingers	WBC. 9.8 Hct. 40 Plat. 356	Pap: Neg for malignant cells	
			Osteoarthritis Kyphoscoliosis enlarged knees difficulty walking ? tabes, degeneration spinal cord	WBC. 6.1 Hct. 37 Hgb. 10.9 Plat. 277		R_x Iron

NO.	AGE, SEX	PAST HISTORY	INJURIES	WEIGHT POUNDS	BLOOD PRESSURE HEART AND LUNGS	EENT	ABDOMEN GU OR GYN
865	28 F	3 mo. pregnant 7 children, 1 died 1951, unilateral exophthalmos		93 98 (1957)	80/50		Liver down 1 cm.
868	38 M	Face pocked, ? diag. pain in legs.		166 170) steady weight 182) loss	110/70		
875	44 M	Occassional convulsions		? 125 131 (1959)	106/70		
877	23 M	Keloid right shoulder		133	124/80		
878	61 M	Hypertension, emphysema, ? prolapsed disc, 1959		193	156/94 sounds distinct		
880	40 M			195 heavy muscle 143 ? 158 weight gain	120/80		
882	28 M	Tumor, left parotid, removed 2 yrs. ago. Benign		128	104/50		
884	66 M	? Scar, left groin. old TBC ? myocarditis ECG, 1959		152	118/60	arcus sinilis, tonsilar hypertropny	small testicles
885	66 M	Cut left 4th. finger 1955		138	112/66		Tender area RLQ ? appendicitis
889	36			106 102) in 92) past	100/62 A27P2		Hard cervix, ? induration rt. adnexia, sl. prolapse
893	43 F	Diabetes losing weight		112	104/76 wheezing both lungs		friable cervix, bleeds easily, severe erosion, prolapse
894	64 F	Coughing, rales present earlier, abd. pain		112	140/80 rales, left base heart, upper limit of normal	corneal opacity	Guarding RLQ ? mass neg. on Repeat Exam

SKIN	TUMORS	CONGENITAL ABNORMALITIES	MISCELLANEOUS	BLOOD COUNT	OTHER LAB. DATA. URINE PAP. ETC.	COMMENTS
Bl. mole chest				WBC 5.1 Hct. 35 Hgb. 10.9 Plat. 277		
				WBC 6.6 Hct. 48 Plat. 251		Weight loss
	2x3 cm lipoma left thorax			WBC 5.6 Hct. 49 Plat. 360		
				WBC 7.1 Hct. 47 Plat. 318		
Hyperker. areas neck			? Osteoarthritis	WBC 6.3 Hct. 40 Plat. 182		X-ray, rt. hilum prominent (RECHECK)
				WBC 11.4 Hct. 46 Plat. 24[illegible]		Weight gain
		? enlarged distal ulnae		WBC 8.1 Hct. 45 Plat. 357		
			Arthritis, spine	WBC 11.0 Hct. 39 Plat. 348		
	1/2 cm. scar nodule below xyphoid		Small axillary nodes	WBC 8.8 Hct. 43 Plat. 282		
				WBC 7.0 Hct. 36 Hgb. 11.8 Plat. 314	Pap: Neg., good estrogen level	Gaining weight
few macules side neck			small axillary nodes, thrombosed hemorrhoids	WBC 6.0 Hct. 36 Plat. 237	Pap: Neg. Trichomonas urine sugar 4+	R_x Cholesterol ? diabetes fasting blood sugar
			Left axillary nodes	WBC 4.7 Hct. 40 Plat. 304	Pap: Neg., very poor smear	

NO.	AGE,	SEX	PAST HISTORY	INJURIES	WEIGHT POUNDS	BLOOD PRESSURE HEART AND LUNGS	EENT	ABDOMEN GU OR GYN
896	21	F	Backache; worms in stool		95 100 (1959)	108/60		
895	31	F	2 spontaneous abortions in past 2 years		127	120/80		Cervix not enlarged ? pregnant 2 mo.
897	63	M	Pain, left shoulder weak		162 1/2	128/66	Arcus senilis left eye	
898	63	F	Vaginitis, arthritis		165	120/85	Arcus senilis left eye. Ulcer left tonsil	Cervix bleeds easily
899	65	M	Dupuytrens contracture both hands		124 (1959)	136/70		
914	26	F	Worms		102 (1959)	92/158		Menstrating
915	64	M	Thickened rt. cord, 1959		125	120/72		Prostate 2X normal
916	37	F	Deafness? not confirmed. 4 mo. preg.		143 153 (1959)	92/85		
917	42	M			183 175 (1959)	124/80 split P2.? BBB ECG, 1959		Prost. norma
918	63	M	No other history. Diabetes	Scars around groin and rectum		130/76 wheezes, lt. chest	Arcus senilis	
920	29	M	Healed burn, scar buttocks		132 (1959)	108/72		
922	37	F	Lactating 2 1/2 post part.		97 weight ↓ 2 yrs. 104	110/70		Cervix biops.

SKIN	TUMORS	CONGENITAL ABNORMALITIES	MISCELLANEOUS	BLOOD COUNT	OTHER LAB. DATA. URINE PAP. ETC.	COMMENTS
		Rt. leg shorter short right femur due to injury as child or congenitally		WBC 6.2 Hct. 35 Plat. 259	Pap: Neg., sl. endocervical cell atypia	
				WBC 7.4 Hct. 42 Plat. 311		
				WBC 11.6 Hct. 40 Plat. 189		
			Left knee swollen, stiff atrophy thigh ? arthritis ? traumatic	WBC 11.1 Hct. 38 Plat. 153	Pap: Neg., high estrogen level	
			Osteoarthritis	WBC 6.5 Hct. 36 Hgb. 13.8 Plat. 470		
				WBC 9.2 Hct. 34 Plat. 276		
				WBC 5.8 Hct. 40 Plat. 167		
Few macule back neck				WBC 5.5 Hct. 30 Hgb. 9.4 Plat. 244 Anemia	Urine sugar 2+	R_x Iron Fasting blood sugar
				WBC 5.1 Hct. 50 Hgb. 14.8 Plat. 200		
			Urine sugar 4+		Urine sugar 4+	R_x Fasting blood sugar, cholesterol
			Small cervical nodes	WBC 9.2 Hct. 51 Plat. 265	Urine sugar 3+	R_x F. blood sugar, cholesterol
			Osteoarthritis	WBC 5.6 Hct. 36 Plat. 330	Pap: neg., endocervical atypia	

NO.	AGE,	SEX	PAST HISTORY	INJURIES	WEIGHT POUNDS	BLOOD PRESSURE HEART AND LUNGS	EENT	ABDOMEN GU OR GYN
928	48	F		Scar, left chest, burns	124 132 (1959)	122/76		Cervix O.K.
929	63	F	Never pregnant		138	128/84	decreased vision left eye	small cervix
932	26	F	One child dead meningocoele 8/60;3 mo. preg		103	90/52 sys M		
934	26	F			123	110/70	Slight tonsilar hypertropny	Tender RLQ
938	22	F			87 94 (1959)	100/70		
941	60	F	Hypertension, ? lump in back		114	140/90		
942	46	F			132 (1959)	108/72		
943	33	M				120/80		
944	36	M	Backache		170	130/70		
945	36	F	History of productive cough		86	116/70 sys M pulmonic area		Cervix lacerated, resistance RLQ
947	53	M	Hypertension, arthritis		117	185/105 wheezes both lungs		
951	28	F	Lactating, night-blindness.		125	O.K.		Slight cervical erosion
956	52	F	Baby died at birth. One stillbirth		123	130/80		

SKIN	TUMORS	CONGENITAL ABNORMALITIES	MISCELLANEOUS	BLOOD COUNT	OTHER LAB. DATA. URINE PAP. ETC.	COMMENTS
			Artnritis symptoms, Osteoarthritis	WBC 4.4 Hct. 33 Plat. 246 Anemic	Pap: Neg., Inflammation, endocervical atypia.	R_x Iron
				WBC 11.0 Hct. 37 Plat. 351	Pap: Neg. some endocervical atypia	
Superficial ulcers on extremities				WBC 6.9 Hct. 33 Plat. 378 Anemia		R_x Iron
				WBC 6.9 Hct. 37 Plat. 236	Pap: Neg.	
		6 toes, left foot webbed		WBC 10.1 Hct. 41 Plat. 169	Pap: Neg., Good estrogen level	
				WBC 9.4 Hct. 38 Plat. 371	Pap: Neg. Good estrogen level. Marked inflammation. Trichomonas	
				No count		
				WBC 8.4 Hct. 49 Plat. 476		
				WBC 10.3 Hct. 46 Plat. 338		
				Not done	Pap: Neg.for malignant cells	
Depig. areas				WBC 7.9 Hct. 39 Plat. 342		
				WBC 9.0 Hct. 37 Plat. 312		
			Urine sugar 2+	WBC 6.7 Hct. 35 Plat. 326	Pap: Neg. with atypia due to dryness--inflammation	R_x Fasting blood sugar

AGE,	NO.	SEX	PAST HISTORY	INJURIES	WEIGHT POUNDS	BLOOD PRESSURE HEART AND LUNGS	EENT	ABDOMEN GU OR GYN
957	51	F	Deafness, ? hypertension		150	118/76		
958	51	F	G.I. symptoms ? ameoba ? perineal cyst		124 1/2 wheezes both lungs	110/68		Liver down 3 cm.
961	68	M	20 lb. weight loss, Parasthesia legs, Diabetes		144	136/80		Bilateral Hydrocoele
963	43	M			139 1/2	120/80		Scar left breast from tumor, removed 2 yrs. ago. Liver down 1 cm.
964	85	M	Lipoma left knee		128	130/76	Opacity both lenses	
969	43	M			123 124	110/58		
970	47	F			105 110 (1959)	10[illegible]/64		Liver down 1 cm.
973	52	M	Hypertension, mild	Burn, left lower leg. Inguinal incisions.	133	140/90 Frothy sputum, wheezing.		Sl. enlarged prostate
982	40	F	Hypertension		137 144 (1959)	145/100		Thickened area around ext. os.
984	29	F				114/74		Liver down 1cm. Mass RLQ probably feces, prolapsed rectum
991	53	F	Hypertension ? diabetes '59 blood sugar 278		173 184 (1959)	124/84		Lacerated cervix
1001	27	F	Receiving iron		107 109	114/60 sys M P27A2		Rt. breast large than left,

SKIN	TUMORS	CONGENITAL ABNORMALITIES	MISCELLANEOUS	BLOOD COUNT	OTHER LAB. DATA. URINE PAP. ETC.	COMMENTS
				WBC 7.4 Hct. 41 Plat. 532		
			Pilonidal cyst not draining	WBC 11.9 Hct. 40 Hgb. 11.2 Plat. 292		
			Awkward walk Romberg neg. ? past column disease	WBC 5.6 Hct. 42 Plat. 176		R_x ? diabetes Fasting blood sugar. Cholesterol
				WBC 5.0 Hct. 44 Plat. 186		
	Lipoma left knee 5 cm.		Severe kyphosis, Osteoarthritis	WBC 4.0 Hct. 37 Plat. 229		Consider cataract removal
				WBC 6.8 Hct. 40 Plat. 418 Hgb. 11.5		
Impetigo, legs	Ganglion left wrist			WBC 7.3 Hct. 37 Hgb. 10.6 Plat. 283	Pap: Neg. Good estrogen level	
	Lipoma left shoulder 6x5 cm.			WBC 6.8 Hct. 45 Plat. 427		R_x Dedral X-ray, fibrotic markings, Lt. base
			Several small nodes left neck	WBC 9.6 Hct. 39 Plat. 212 Hgb. 11.5	Pap: Neg. Good estrogen level	
				NONE		
				WBC 7.1 Hct. 43 Hgb. 14 Plat. 182	Pap: neg. Endocervical atypia. Urine sugar 4+	R_x Fasting blood sugar. Cholesterol
				WBC 10.8 Hct. 35 Plat. 236	Pap: Neg. Good estrogen level; inflammation--	

NO. AGE, SEX	PAST HISTORY	INJURIES	WEIGHT POUNDS	BLOOD PRESSURE HEART AND LUNGS	EENT	ABDOMEN GU OR GYN
1005 28 M	Small inguinal hernia. Rt. indirect, easily reduced		157 (1959)	136/88		
1007 50 M		Rt. inguinal scar	170 (1959)	124/80 130/86(1959)		Small testes