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MEDICAL SURVEY OF RONGELAP PEOPLE EIGHT YEARS AFTER EXPOSURE TO FALLOUT

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Introduction

The results of a medical survey of the people of Rongelap in the Marshall Islands, carried out in March 1962 at 8 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. This number has since increased to about 200. 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 Kwajalein and Majuro Atolls for examination of a number of Rongelap people, now residing at these atolls, and also groups of children who represent

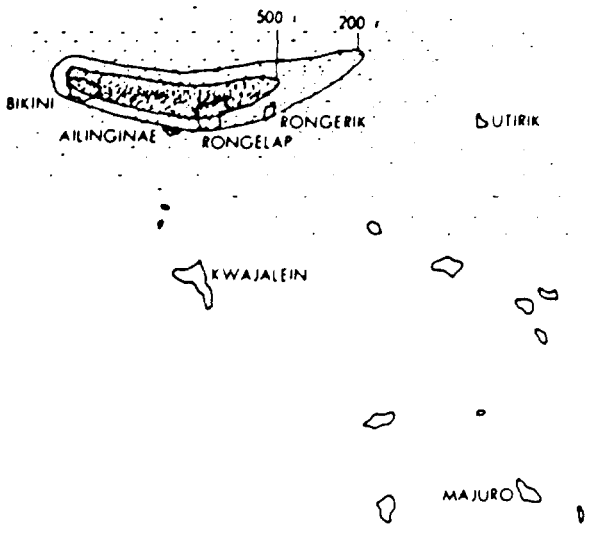

Figure 1. Map of fallout area (March 1, 1954), Marshall Islands.

part of the control group used for the growth and development studies of the exposed children.

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] 5 and 6 years,[7] and 7 years.[8] 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 lacrymation 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 RADIATION

One of the earliest findings indicative of significant exposure in these people was lowering of levels of *leukocytes* and *platelets* of the peripheral blood. This was most marked in the 64 people on Rongelap who had received 175 r, and was less marked in the other groups receiving less exposure. The hemopoietic depression was roughly proportional to the dose of radiation received. Even in the 157 Utirik people who received only an estimated 14 r, it was possible to distinguish slight platelet depression in the group as a whole. The smaller group on Ailingnae and Rongerik showed peripheral blood levels between those of the high and low exposure groups. The chronological records of blood findings in the Rongelap and Ailingnae groups are presented in Table 10 and in Appendices 1 and 2.

Lymphopenia of about half the level of the comparison Marshallese population was evident when the Rongelap people were first examined on their arrival at Kwajalein 3 days after exposure. This depressed level was maintained steadily with only slight increase noted by one year. In the following year, mean counts approached the levels of the comparison population but have since generally remained slightly below.

Neutrophil levels fluctuated considerably during the first month; possibly this was related to the prevalence of beta burns of the skin during that period. Neutrophil depression became evident by 5 and 6 weeks post exposure with levels reaching about half that of the comparison population. This degree of neutropenia was insufficient to result in any apparent increased infectious processes, and indeed it was noted that neutrophilic leukocytosis was possible in people showing casual infections at this time. Neutrophil levels recovered more rapidly than lymphocyte levels and reached near control levels by one year. Subsequent annual surveys have revealed that recovery does not appear to be complete, particularly in younger and older age groups.

Platelet counts showed less fluctuation than other blood counts and fairly consistently showed increasing depression, reaching levels of about 30% that of the comparison population by the 4th week. A spurt of recovery to about 75% of comparison levels occurred during the following few weeks, which was followed by slower recovery but with mean levels never reaching higher than 85 to 90% that of the comparison population during the 7 years post exposure.

Erythropoietic depression has not been a consistent finding as with the leukocytes and thrombocytes. Slight depression of red blood counts, hematocrits, and hemoglobin have been noted at times.

No gross abnormalities of *bone marrow* smears were reported at 6 months post exposure.

Depressi ~ of peripheral blood elements in the Ailingnae and Rongerik groups was not so pronounced as in the Rongelap group. However, a slight lag in complete recovery in the Ailingnae peripheral blood count has also been noted.

The persistent depression of peripheral blood elements in the exposed people makes it appear likely that there is slight residual bone marrow damage.

A general *anemic* tendency has been evident in both exposed and unexposed Marshallese. Price-Jones curves, on the average, showed a slight microcytic tendency. Serum iron levels have generally been normal, and the cause of this anemic tendency has been undetermined.

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

Except for radiation-induced lesions of the skin, patchy epilation, and early gastrointestinal symptoms, *clinical examinations* have revealed no disease processes or symptoms which could be related directly to radiation effects. No prophylactic or specific therapy of radiation effects was ever considered necessary or given. Epidemics of chicken pox and measles that occurred showed no greater incidence or severity in the exposed than in the unexposed Marshallese people.

During the first months post exposure about half of the exposed group exhibited *loss of weight* of several pounds. This may possibly have been related to their radiation exposure, although it is difficult to rule out effects possibly due to change of environment.

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 35-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 7.1 per 1000 population, compared with 8.3 for the Marshall Islands as a whole.

Growth and development studies on the children (height, weight, anthropometric measurements, radiographic studies for bone age) have revealed that slight retardation in growth and development has occurred in the exposed boys who were under 12 years of age at the time of exposure, particularly those 12 to 18 months of age at exposure. Only slight immaturity was noted in the exposed female children. It was also noted that children born of exposed parents were slightly retarded and that they had slightly lower levels of neutrophils, lymphocytes, and platelets, compared with male children of unexposed parents.

It was difficult to evaluate the effects on fertility. However, a review of the *birth rate* of the exposed group over the past 7 years seems to indicate no noticeable effect of their exposure on fertility. The 31 births represent a rate of 54 per 1000 population, compared with 37.3 for the Marshall Islands (1957). The 21 births over a 4-year period for the comparison population represent a rate of 72 per 1000 population. A somewhat greater incidence of *miscarriages and stillbirths* was noted in the exposed women during the first several years after exposure, but because of the paucity of vital statistics on 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)[7] 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)[7] 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 pterygia, pingueculae, and corneal scars 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[7] showed no significant differences in caries rate between exposed and unexposed groups. However, the incidence and severity of peridontal disease was slightly greater in the exposed group. It is not known whether or not this finding is related to radiation effects. The poor oral hygiene generally observed in the Marshallese had its usual results; namely, high caries rate in teenage 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; cardiovascular 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 has had slightly elevated 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, with the possible exception of suggestive evidence of increased miscarriages and stillbirths in the exposed women and the slight retardation of growth noted in the male children of exposed parents.

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. During the past several years an increased number of pigmented maculae and moles have been noted in previously irradiated areas of the skin, but these have appeared to be quite benign.

Numerous histopathological studies have been made,[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 noted during the first few months after exposure was the development of bluish-brown pigmentation of the semilunar areas of the fingernails and toenails in about 90% of the people. By 6 months this pigmentation had disappeared, having grown out with the nail. The cause of this phenomenon has not been explained.

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 1958 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. 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.

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. In 1961 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 in 1959; 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 in 1959. 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. 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. The body burdens are 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 excreters 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. Studies of Gm *phenotypes* showed the Marshallese to have 100% $Gm^{(a+)}$ and nearly 100% $Gm^{(b+)}$. There was a complete absence of Gm^x and a high frequency of Gm-like (Gm^e). Considerable caution 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 abnormally 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 over-all 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 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.

1962 Survey — Organization and Procedures

The medical team consisted of 5 medical specialists from the U.S., 2 Marshallese practitioners from the Trust Territory, and 8 technicians, 4 from the U.S. and 4 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 with officials of the Navy Pacific Missile Range Group and the Trust Territory of the Pacific Islands.

Figure 2. Medical team. Left to right, front row: E. Adamik, L. Meyer, W. Moloney, R. Conard, A. Lowrey, A. Hicking, D. Clareus; standing: K. Mizutoni, W. Scott, I. Jones, S. Shonfber, E. Riklon, K. Kittien, A. Obten. (W. W. Sutow is not in the picture.)

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.

A Trust Territory ship, the M/V *Ran Anim*, transported the team and equipment to Rongelap on March 4, 1962 (Figure 3). A tent had been set up on the Island and one of the village houses rented to serve as living quarters for the team. On arrival, after greeting the Magistrate and other village dignitaries, it was learned that the death of an aged woman had occurred the previous day, and that an autopsy was to be permitted on the body. Establishing living quarters and setting up laboratory equipment and examination facilities in addition to carrying out the autopsy occupied the better part of the first day. Late in the afternoon the funeral for the deceased took place. The medical examinations on the people began the following day.

The 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. 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, urine collections and analyses, and x-ray examinations.

Figure 3. Trust Territory ship *Ran Anim* at anchor, Rongelap Lagoon.

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); special examinations of the skin with color photography of selected lesions; a special cancer detection survey; and an ophthalmological 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 information, since the incidence of familial diseases including cancer was generally unknown by the people. The history yielded some information on changes in weight, history of illness, and, in the case of women, menstrual, obstetric, and nursing history. In the physical examination particular emphasis was placed on examination of the skin, node-bearing areas, head and neck, chest, breast, abdomen, and external genitalia. Pelvic examinations were carried out on all mature females, and vaginal and cervical smears for Papanicolaou examination were obtained.* Rectal examinations were carried out on all persons >40 years of age. This included, in the case of men, palpation of the prostate gland. Chest plates were not taken routinely but were obtained on about 70 adults >40 years of age (and on certain other cases where indicated).** Hematological data were obtained and were available for evaluation.

In detection of possible leukemia (or preclinical evidence of incipient leukemia) the lymph nodes and spleen were carefully examined, and hematological data were taken including routine hemograms, percent basophils in a 4000 white cell count, and alkaline phosphatase examinations of the white blood cells on differential smears.

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*We wish to thank Dr. Genevieve Bader of Memorial Sloan-Kettering Cancer Center, N.Y.C., for interpretation of the Papanicolaou smears.

**We are grateful to Dr. Paul Lichtblau of Rockville Centre, L.I., N.Y., for interpretation of the chest roentgenograms.

GROWTH AND DEVELOPMENT STUDIES IN CHILDREN

In addition to the routine pediatric examinations, certain special anthropometric measurements on the children were recorded. Such data included age, weight, stature, sitting height, head circumference, biacromial width, bi-iliac width, and calf circumference. Roentgenographs of the left wrists were studied for skeletal maturation.

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 has 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. Hematocrits were obtained by the microhematocrit method. 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.

Blood smears were obtained for differential study, alkaline phosphatase examinations*, and basophil counts on 4000 white cells, as part of the leukemia survey.

Bone marrow aspirations were done on 9 exposed people, and smears were prepared for differential counts and morphological studies. In addition, chromosome preparations were made on the aspirated material.

Blood volume determinations were carried out on 8 unexposed Rongelap people and 7 Americans during the 1962 survey. During the 1961 survey such studies had been made on 5 Marshallese and 5 Americans. The analyses were done by the radioactive sodium chromate (Cr^{51}) technique.*

Blood samples were returned to the U.S. for *electrophoretic studies* for glucose - 6 - phosphate dehydrogenase and hemoglobin types.* Electrophoretic studies of 171 sera for immune globulins were kindly carried out by Dr. R. Bütler and Dr. A. Hassig at the Swiss Red Cross Laboratory, Berne, Switzerland.

Protein-bound iodine determinations were made on sera from 10 individuals who had previously shown slightly elevated levels. The method of Foss, Hankes, and Van Slyke[10] was used.**

For *chromosome studies*, short-term blood cultures were done on 10-ml samples from about 50 people. This blood was taken at the time of sampling for routine hematological studies. A modification of the technique of Moorehead[11] was used. The leukocytes were separated by centrifugation and placed in culture bottles with growth medium (Difco-199) and phytohemagglutinin (red kidney bean extract prepared by Dr. L. Schiffer of BNL). Dextran (3%) was used in the initial separation in some cases but was discontinued later since it was found to be unnecessary in view of the rapid settling of the red cells in the Marshallese. The cells were cultured for 3 days at 37°C, after which chromosome preparations were made as follows: Cells were separated by centrifugation, washed with Hanks' solution, treated with hypotonic solution (distilled water plus Hanks' solution) for 8 to 10 min, and fixed in methyl alcohol—glacial acetic acid (3 to 1); smears were prepared by blowing a drop of the cell-rich sediment on a slide and air drying. Only occasional slides were stained with Giemsa's stain for evaluation in the field. Final staining and chromosome studies were carried out at the Holy Ghost Hospital in Boston .†

Fasting *blood sugar* levels[12] were obtained on 8 individuals who had shown positive urine sugars.†† Blood collected routinely for hematologi-

*We are grateful to Miss Lila Fliegelman of the Boston City Hospital for carrying out the alkaline phosphatase analysis of blood smears.

*We are grateful to Dr. Samuel H. Boyer of the Johns Hopkins Hospital for carrying out these studies.

**We are grateful to Dr. D. D. Van Slyke and Miss Dorothy Ripperger of BNL for performing these analyses.

† Under the direction of one of us (W. C. M.). We are grateful to Miss Geraldine Dowd and Miss Catherine Dunn at the Holy Ghost Hospital, Boston, for their assistance with these analyses.

†† We wish to thank Dr. D. D. Van Slyke, Dr. L. V. Hankes, and Miss D. Ripperger at BNL for carrying out these analyses.

Table 1

Location of Rongelap People

	Exposed			Unexposed		
	Adult	Children	Children of exposed parents	Adult	Children	Total
Majuro	3	1	4	8	7	23
Kwajalein	6	3	9	25	24	67
Rongelap	33	26	23	59	75	216
Eniaetok	8	0	1	5	1	15
Other atolls	2	0	0	12	13	27
Total	52	30	37	109	120	348

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 (1961)	U.S. (1960)
<15	45.4%	50.0%	43.1%	28.7%
15-24	12.3	13.8	14.4	13.8
25-44	23.6	17.2	20.0	27.1
45-64	14.1	10.3	14.6	20.8
>65	4.5	8.6	7.4	9.6
Median age, yr	19.5	14.5	18.0	29.5

cal analyses was also examined for sugar in the case of 199 Rongelapese.[12]*

Urine Analyses

Urine total iodides and creatinine levels were obtained on 10 casual urine samples of Rongelapese who had previously shown slight elevation of their protein bound iodine (PBI) levels. Total iodine and creatinine were determined.

Routine urine analyses were carried out on the majority of people. This included determinations of protein and hyperglucosuria by reagent paper strips.**

Radionuclide Body Burden Evaluation

Since results of the last survey indicated that Cs^{137} levels in the Rongelap people had about reached equilibrium and that Zn^{65} levels were dropping, it was considered that gamma spectroscopy with the 21-ton whole-body counter would not be necessary again for several years. Therefore, no gamma spectrographic analyses were done during this survey. However, thirty-five 24-hr urine samples and one pool sample (12 liters) were collected for radiochemical analysis for Sr^{90}. In addition, samples of rib and vertebrae taken at autopsy from the 78-yr-old woman who had died were brought back for Sr^{90} analysis; also 4 coconut crabs collected at Rongelap.**

Results and Discussion

The census of Rongelap people in March 1962 was 348, of which 82 were in the exposed group (including 4 children exposed *in utero* at the time of the accident), 37 were children of exposed parents, and 229 were unexposed people; Table 1 shows their locations. In Table 2 the population is broken down according to percent distribution in various age groups and compared with that of the Marshall Islands as a whole for 1961 and of the U.S. for 1960. The table also shows the median age. The lower median age of the Marshallese

*The Clinical Laboratory of the South Nassau Communities Hospital carried out these analyses.

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

**We are grateful to Dr. E. P. Hardy, Jr. and Dr. J. Harley of the Health and Safety Laboratory of the New York Operations Office of the AEC for performing these analyses.

would tend to support the impression that their life span is shorter than that of people in the continental U.S., but there has been a "population explosion" in these islands which might account for this discrepancy.

Of the 348 Rongelap people, 308 were examined during the survey at Rongelap, Kwajalein, and Majuro Atolls. Examined were 80 in the exposed group, 37 children of exposed people, and 191 control adults and children.

INTERVAL MEDICAL HISTORY

Mortality

One death occurred in a 78-yr-old exposed woman on March 5, 1962, the day before the arrival of the survey team. This death will be described below.

The 5 deaths that have occurred in the 8-yr period since exposure represent a rate of 7.65 per 1000 population, which is not very different from the rate for the Marshall Islands as a whole (8.3 per 1000 for 1960). Comparison with the unexposed population of Rongelap is complicated by variations in the size of this population, which has generally increased from year to year since the people were first included in the study in 1957. However, the death rate in this group, which has fewer older people, appears to be only slightly less than that in the exposed Rongelap group.

Births

The birth rate for the past year was again calculated, as in the preceding survey, from the number of births per woman of childbearing age (16 to 45 yr). There were 22 such women in the exposed group and 32 in the unexposed group. (Not included in either group were 4 unexposed women whose spouses were exposed males.) In the exposed group 4 babies were born, giving an average of 0.182 births per woman; in the unexposed group 4 babies were born, giving a slightly lower birth rate per woman (0.125). The births were all full-term normal deliveries.

Congenital Anomalies

No congenital anomalies were reported in children born of either exposed or unexposed parents during the past year. Specific genetic studies have not been included on this relatively small population. Routine examination of babies born to exposed women since the accident has not revealed any increased incidence of anomalies as compared with children born of unexposed parents. In 1960, one of the babies born of an unexposed mother (No. 75) had a congenital heart defect. The baby died at 4 months of age. Unfortunately, an autopsy was not possible and the diagnosis was not confirmed. Most of the anomalies reported had occurred in children of unexposed parents; they include a low incidence of patent ductus arteriosus, congenital deformity of the hip, and congenital hypoplasia of the middle phalanx of the 5th finger.

Miscarriages and Stillbirths

During the past year no miscarriages or stillbirths were reported in exposed or unexposed women. During the past several years the incidence of miscarriages 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 in most instances it has not been possible to have a physician examine the products of miscarriage.

Death of an Exposed Woman

On March 5, 1962, a 78-yr-old exposed woman died. During the past few years she had become very feeble and was bedridden and incontinent with senile deterioration. She was kept in a penned-in portion of her room. The major findings on previous examinations had been acute kyphoscoliosis (she was so badly stooped that she could not stand upright), bilateral cataracts, hypertension, arteriosclerotic heart disease, osteoarthritis, and senility. Several weeks before her death she developed an upper respiratory infection with nausea and vomiting. She became delirious and excited at times and attempted to pull herself upright. Four days before her death she became comatose and went rapidly downhill. Fortunately an autopsy was

possible since the team arrived the day after her death. Principal gross findings included fractures of the 5th, 6th, and 7th ribs and the first lumbar vertebra with ecchymoses and internal hemorrhage, atherosclerosis of the heart and aorta, bilateral pulmonary edema, and benign nephrosclerosis. Atrophy of the kidney, liver, and spleen were noted. Principal findings on microscopic examination included myocardial fibrosis, aortic atherosclerosis, and pulmonary edema. It was believed that death resulted from trauma and hemorrhage, possibly from a fall. Details of the autopsy findings are reported in Appendix 6.*

Illnesses

Only 4 people, 2 exposed and 2 controls, required hospitalization during the past year. Two cases of hemorrhoids and an anal fistula required surgical correction; one case of an acute exfoliative type of dermatitis required hospital treatment. The health aide reported that during the past year about 20 people on Rongelap developed a sickness associated with eating improperly prepared arrowroot flour. The sickness was characterized by soreness of the mouth and throat, anorexia, nausea, vomiting, and diarrhea, and lasted from a few days to several weeks. Occasional cases of fish poisoning (numbering about 20) occurred during the past year. They were characterized by the typical gastrointestinal and neurological symptoms noted in the past. Fish poisoning is a ubiquitous illness in the South Sea Islands. The only other sicknesses reported were the usual number of cases of upper respiratory infections, gastroenteritis, and numerous skin conditions.

ADULT EXAMINATIONS

In Table 3 are listed the various physical abnormalities in the adults, except for the ophthalmological findings, which are listed and discussed in the following section. As noted in previous examinations, the incidence of various abnormalities in the exposed group did not appear to be significantly different from that in the unexposed comparison group. Appendix 5 lists major findings on each adult examined.

Certain abnormalities such as moderate to severe arteriosclerosis showed a higher incidence in the exposed group, but these increases were probably 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. In the exposed group several of the older people (No. 57-F, age 107; No. 46-M, age 86; No. 55-M, age 82; and No. 28-F, age 75) showed marked infirmities of old age and presented such findings as arteriosclerotic heart disease, kyphoscoliosis, osteoarthritis, cataracts, and blindness. These people had to be assisted to the examination room. Only two cases in the unexposed group were in this category (No. 862-M, age 88, and No. 946-M, age 85).

OPHTHALMOLOGICAL EXAMINATIONS

In Table 4 the incidence of various ophthalmological findings in the Rongelap people is presented. Pterygia and pingueculae were the most common, occurring in 43% of the exposed adults and 36.9% of the control group, but these were not found in individuals <20 years of age and were most common in those >40. All lens opacities could be classified as senile, presenile, or congenital. There were 14.5% of the exposed adults and 13.6% of the control group that had senile cataracts. Lenticular opacities (including presenile and congenital) occurred in 22.9% of the exposed group and 21.7% of the control group; they were most common in those >45 years of age. No opacities were noted characteristic of those induced by radiation exposure.

Corneal pigmentation could be classified into two groups, congenital and acquired. There were 20.8% of the exposed and 23.9% of the control group that had congenital corneal pigmentation not unlike that seen in all dark-skinned races in any part of the world. Three cases (6.2%) in the exposed group had a noncongenital pigmentation, consisting of a dark linear streak of pigment ex-

*The gross autopsy findings are reported by one of us (L. M.). We are grateful to Drs. Hans Cottier, W. Calvo, and V. Alcober of BNL for reporting the histological findings.

Table 3

Physical Findings in Rongelap Adults, 1962

	Exposed (48 examined)		Control (86 examined)	
	Subject Nos.	%	Subject Nos.	%
Anemia, anemic tendency	30, 45, 46, 55, 70	10.4	860, 865	2.3
Arteriosclerosis, peripheral, mild	11, 52	4.1	851, 852, 855, 859, 878, 884, 894, 898, 917, 956, 915, 957, 969, 970, 982	17.4
Arteriosclerosis, peripheral, moderate to severe	13, 28, 29, 30, 41, 46, 55, 43, 57, 60	18.7	853, 860, 862, 908, 935, 947, 964	8.1
Asthma	29, 45	4.2		
Auricular fibrillation with myccardial damage	80	2.8		
Biadycardia	27	2.1		
Bronchitia			894	1.2
Cardiac enlargement	1, 30, 60, 80	8.3	858, 859, 862, 942	4.6
Cervical erosion	12, 18, 34, 63, 64, 74	14.2	825, 826, 846, 851, 895, 898, 936, 938, 951, 970, 982, 1001, 1042	15.1
Cervical lacerations	45, 58, 71, 78	8.3	829, 851, 945, 991	4.6
Cervical prolapse	12	2.1		
Colloid goiter			853	1.2
Congenital defects				
Dislocation of hip	41	2.1		
Prominent head ulna	14, 28	4.2	858, 882, 915	3.5
Bilateral shortening of 5th finger	75, 78	4.2	832, 836, 910	3.5
Polydactylism			938	1.2
Shortened left thumb	57	2.1		
Flexion deformity, fingers			826	1.2
Constipation	74	2.1	835	1.2
Cyst, ovarian			832	1.2
Cyst, pilonidal			958	1.2
Cystocele	1, 14	4.2		
Deafness	1	2.1	853, 884, 916, 964	4.6
Diabetes mellitus			853, 893, 932, 936, 991	5.8
Dupuytren's contracture			884	1.2
Emphysema			853	1.2
Fungus infection, skin	30	2.1	895	1.2
Furunculosis			942	1.2
Gynecomastia	55	2.1		
Hallux valgus	50	2.1		
Heberden's nodes	57	2.1	928	1.2
Hemiplegia, partial	46	2.1		
Hemorrhoids	27	2.1	849, 884, 893	3.5
Hydrocele			961	1.2
Hypertension (>140/90)	1, 11, 28, 30	8.3	858, 908, 947, 982	4.6
Hypotension			914, 932, 969	3.5
Kyphosis, scoliosis	13, 43, 52, 57, 77	8.3	859, 860, 864	3.5
Leprosy, arrested	77	2.1		
Liver palpable	14	2.1	825, 844, 853, 865, 958, 963, 970	8.1
Myocardial damage or insufficiency (EKG)	46, 60	4.2	851, 858, 878, 884, 893, 917, 947, 956, 957, 969, 970	12.8
Obesity	1, 49, 50, 60, 71, 74, 78	14.6	849, 851, 941, 991, 1005, 1041	7.0
Osteoarthritis	13, 29, 46, 52, 55, 57, 60	14.6	858, 859, 860, 884, 894, 896, 898, 915, 922, 928, 935, 936, 961, 964	16.3

Table 3 (continued)

Physical Findings in Rongelap Adults, 1962

	Exposed (48 examined)		Control (86 examined)	
	Subject Nos.	%	Subject Nos.	%
Paralysis, facial	82	2.1		
Pleural thickening or adhesions	58	2.1	823, 826, 875, 969	4.6
Prolapse of vaginal wall	63, 64	4.2		
Prostatic hypertrophy	11, 29, 82	6.2	855, 864, 910, 915, 947, 964	7.0
Rheumatic heart disease	76	2.1		
Rheumatoid arthritis			878	2.2
Senility	29, 55, 57, 46, 28	10.4	862, 946	2.3
Sinusitis	63	2.1		
Syphilis (?) arrested	11, 55	4.2	846, 860	2.3
Tachycardia	28	2.1		
Tonsilar hypertrophy	27, 76	4.2	826, 831, 833, 864, 867, 898, 934, 958	9.3
Trichomonas	9, 12, 58	6.2	942	1.2
Tumor, benign	4, 7, 9, 10, 13, 57, 64	14.6	853, 864, 875, 885, 964, 969, 970, 1007	9.3
Ulceration, lower colon	30	2.1		
Varicose veins	13	2.1		
Vitiligo			853, 971	2.3

Table 4

Eye Findings in Adult Marshallese People, 1962

	Exposed (48 examined)		Controls (92 examined)	
	Number	%	Number	%
Pterygium and pinguecula	21	43.0	34	36.9
Cataracts	7	14.5	12	13.0
Lenticular opacities (presenile and congenital)	11	22.9	20	21.7
Corneal scars	9	19.0	9	9.8
Corneal pigmentation				
Congenital	10	20.8	22	23.9
Acquired	3	6.2		
Healed choroiditis	3	6.2	3	3.2
Retinal scars	2	4.16		
Strabismus	3	6.2		
Nystagmus	1	2.0	1	1.0
Phthisis bulbi	1	2.0		
Anterior staphylomata	1	2.0		
Argyll Robertson pupil	1	2.0		
Seventh nerve weakness	2	4.6		
Healed uveitis	1	2.0		
Corneal foreign body (coral)			1	1.0
Retinal arteriosclerosis	6	12.4	11	13.0
Diabetic retinopathy	1	2.0		
Arcus senilis	12	25.0	23	25.0
Vitreous opacities	6	12.4	10	10.9
Macular degeneration	2	4.16	3	3.2
Drüsen	1	2.0		
Congenital abnormalities				
Retinal vessels	3	6.24	5	5.4
Large corneas	2	4.6	4	4.3
Remnant of hyaloid	1	2.0		

tending from the limbus 2 to 3 mm toward the pupil and lying in the horizontal axis of the palpebral aperture. The pigment was in the epithelial layers of the cornea. Since this is not the usual congenital type of corneal pigmentation, it seems possible that it may have resulted from the irradiation contamination of the eyes of the exposed group by fallout (probably the beta component).

In both the exposed and control groups, there was a low incidence of myopia and strabismus. Visual acuity was higher in both groups than that seen in America. No cases of retinitis pigmentosa, glaucoma, or eye findings characteristic of hyperthyroidism were noted.

Two unusual congenital defects were found among the 140 people examined. Six people exhibited large corneas, and 8 had abnormally large punctate retinal vessels. The latter were not typical of hemangiomata. It is planned to document these findings by pictures in a future survey.

As has been noted before, arcus senilis occurs in the Marshallese with a higher incidence and at an earlier age than generally seen in Americans. The incidence in both exposed and control groups was about the same.

In a recent survey 12 children had a night vision defect due to vitamin A deficiency. In this survey no similar cases were encountered, possibly because of the increased growth and consumption of papaya and squash. Leprosy and yaws are endemic in the Marshall Islands. Two cases of 7th nerve weakness were noted, one in a leprous patient. In

another case, loss of the outer one-third of the eyebrow hairs was suggestive of the disease, but a skin biopsy revealed no acid-fast bacilli.

No cases of retinal scars were observed that might have resulted from retinal burns possibly caused by observation of the fireball of the nuclear explosion that resulted in the fallout accident, although many people on Rongelap reported seeing the fireball and described it as resembling the "rising sun" in the western sky.

PEDIATRIC EXAMINATIONS

During the 1962 survey, 30 exposed chidren, 71 control children, 37 children born to exposed parents and 25 children born to unexposed parents were examined. The size of the pediatric study population (Rongelap series) during each of the last 5 surveys (1958 through 1962) is shown in Tables 5 and 6. The fluctuations from year to year in the numbers of children seen have resulted from (a) movement, generally temporary, to other atolls, (b) graduation from the pediatric to the adult study, and (c) addition of newborn babies. Attrition due to unavailability has been minimal in the exposed group. One child in 1959 and one in 1961 could not be examined, but each of these children returned to the study the subsequent year. In the control group, the attrition rate has been higher, 11.3% for the last 5 years.

The frequencies of various abnormal findings on physical examination have been summarized in Table 7. As in previous years, no consistent relationship was noted between the occurrence of these abnormalities and exposure to radiation.

Growth and Development Studies

In the analysis of the growth data, previously described statistical methodology was used.* The children were divided into 4 groups: (a) those exposed to radiation, including the 4 children *in utero* at the time of exposure, (b) those born before the fallout but not exposed to radiation, (c) those born to exposed parents subsequent to the fallout, and (d) those born to unexposed parents subsequent to the fallout.

Height and weight data on children born before the fallout showed the expected pattern of pubertal

Table 5

Variations in Composition of Pediatric Population, 1958 through 1962:

Rongelap Group

	Numbers of children				
	1958	1959	1960*	1961	1962
Exposed group					
Total seen	39	34	36	30	30
Not available	—	1	0	1	0
Transferred to adult study	—	4	0	5	1
Control group					
Total seen	88	75	51	75	71
Not available	—	13	25	0	10
Transferred to adult study	—	3	2	3	0
Not seen previous year	—	3	3	27	2
New subjects added	—	—	—	—	4

*Limited survey.

Table 6

Variations in Composition of Pediatric Population, 1958 through 1962:

Children Born After Fallout (Rongelap)

	Numbers of children				
	1958	1959	1960**	1961	1962
Exposed parents*					
Number seen	13	20	10	24	37
New babies added	—	7	0	5	12
Not available from previous year	—	0	10	1	0
Old subject not seen previous year	—	0	0	10	1
Unexposed parents:					
Number seen		7	0	14	25
New babies added		7	0	8	10
Not available from previous year		0	0	1	1
Old subject not seen previous year		0	0	0	1

*This category includes only those who were babies at the time they were added to the study. Those who were older at the time they were first seen have been grouped with the regular control children.

**Limited survey.

Table 7

Summary of Physical Findin , in Children, 1962

	Exposed (30)*	Control**		
		Born before 1954 (39)	Born after 1954 (57)	Nonexposed of exposed parents (37)
Active skin lesions	8	5	4	8
Adenopathy	3	5	9	2
Palpable liver	0	0	4	0
Rhinitis	0	2	8	3
Blood pressure taken	25	33	3	2
Hypertension	0	0	0	0
Acute otitis media	1	0	4	2
Chronic otitis media	0	0	2	1
Molluscum	0	0	3	2
Tinea versicolor	2	2	0	0
Tinea cruris	0	1	0	0
Vitiligo	1	1	0	0
Miliaria	0	0	2	2
Seborrhea—scalp	0	0	0	1
Warts	4	2	4	3
Papilloma	1	0	0	0
Cheilosis	0	0	4	1
Sore on lip	0	0	1	0
Black spots—tongue	2	1	0	0
Denuded areas—tongue	0	0	1	0
Pigmentation—lips			1	
Rheumatic heart disease	1	0	0	0
Systolic cardiac murmur (grade II)	5	2	4	2
Swelling, preauricular soft tissues		1		
Umbilical hernia			1	
Hydrocele			1	
Trombocytopenia		1		
Tracheotomy scar	1			
Thoracotomy scar			1	
Obesity	1			1
Pigeon breast				1
Contracture—fingers		1		

*Number examined.

**A part of the group born after 1954 was used as the group "children of unexposed parents" to serve as a control for "children of exposed parents." Cf. footnote to Table 6.

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, for those exposed at ages 1 through 5 to be shorter than the unexposed boys of the same age (Figure 4). Although the differences were statistically significant only in the measurements at ages 9, 11, and 12, the retardation in stature of these boys exposed at an early age was apparent at all ages at which measurements have been made. No tendency toward diminution in the magnitudes of the differences was noted as the boys grew older. The boys exposed at ages 6 through 8 showed no differences in stature from the control boys of the same age. Since there were only two boys exposed in the 12 through 13 age range, the data available did not justify any conclusion regarding the effect of exposure about the time of puberty.

The weights of the exposed and control boys

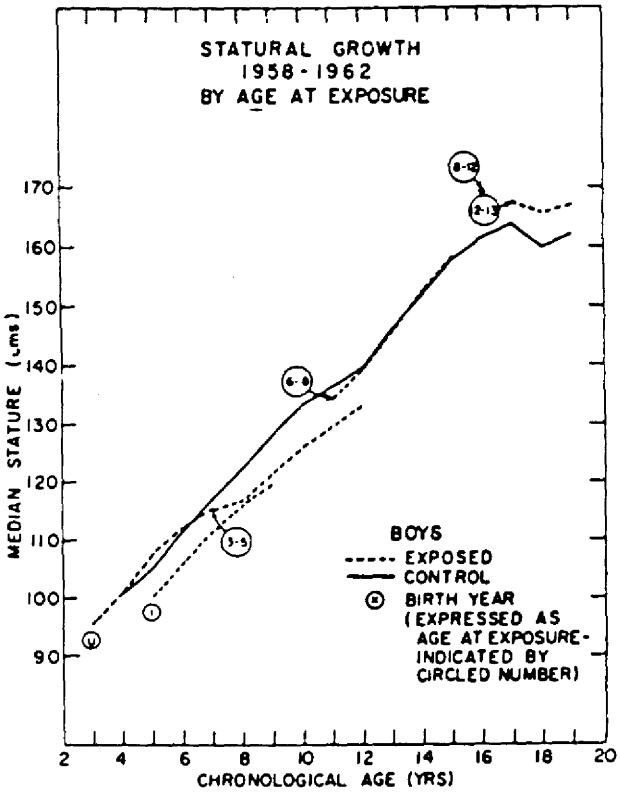

Figure 4.

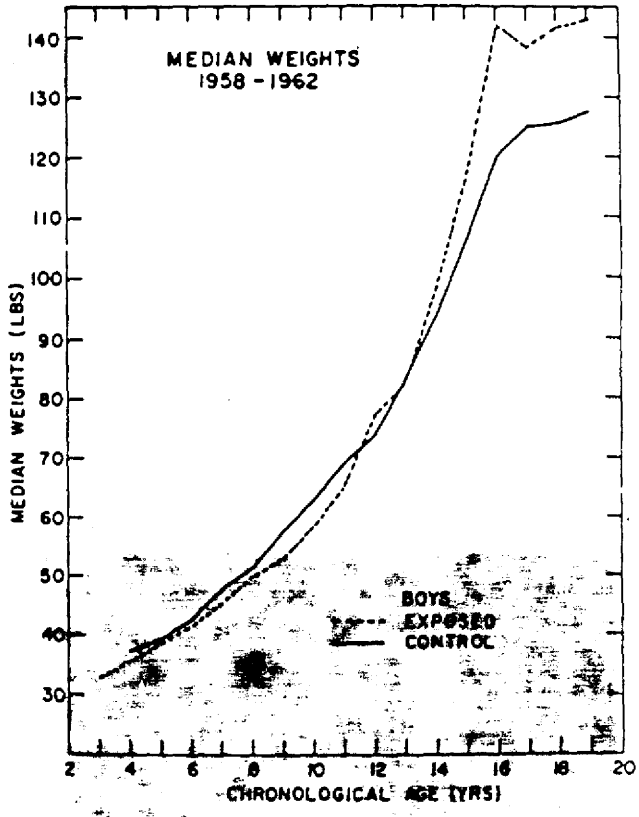

Figure 5.

showed trends parallel to those of their statures (Figure 5). However, the weights were more variable, and the differences were not statistically significant.

The exposed girls did not differ significantly from their controls in either stature or weight at any age level (Figures 6 and 7).

Among the children born after the fallout, the males with exposed parents were smaller in stature at all ages than those with nonexposed parents (Figure 8). The difference was statistically significant at ages 1 through 4. This trend had been noted in the previous study. However, the boys in the group with exposed parents were, on the average, 4 months younger than their controls (the boys with unexposed parents). The median difference in stature between the two groups was 4.4 cm, and from their growth curves the boys with exposed parents would be expected to be 2.3 cm shorter on the basis of the age difference. Since much of the difference in stature was readily accounted for by the age difference, the data did not justify a conclusion that there was a difference in stature associated with the exposure of the parents. The girls of exposed parents did not differ in age from their controls; nor were there age differences between any of the comparison groups of children born before fallout. The boys of exposed parents did not differ significantly from the boys of unexposed parents in weight or head circumference (Figures 9 and 10). The girls of exposed parents did not differ from the girls of unexposed parents in stature, weight, or head circumference (Figures 11, 12, and 13).

Skeletal ages, based on the standards of Greulich and Pyle, paralleled the statural development of the children. 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 (Figure 14). However, the boys were significantly less mature skeletally than the girls, being on the average 7 months retarded, as compared with 2 months for the girls. Also, the exposed children were significantly less mature skeletally than the controls. The median skeletal retardation of the exposed children was 8 months, as compared with 3 months for the controls. The difference in skeletal maturation associated with exposure was more prominent in the boys than the girls. The average exposed boy was

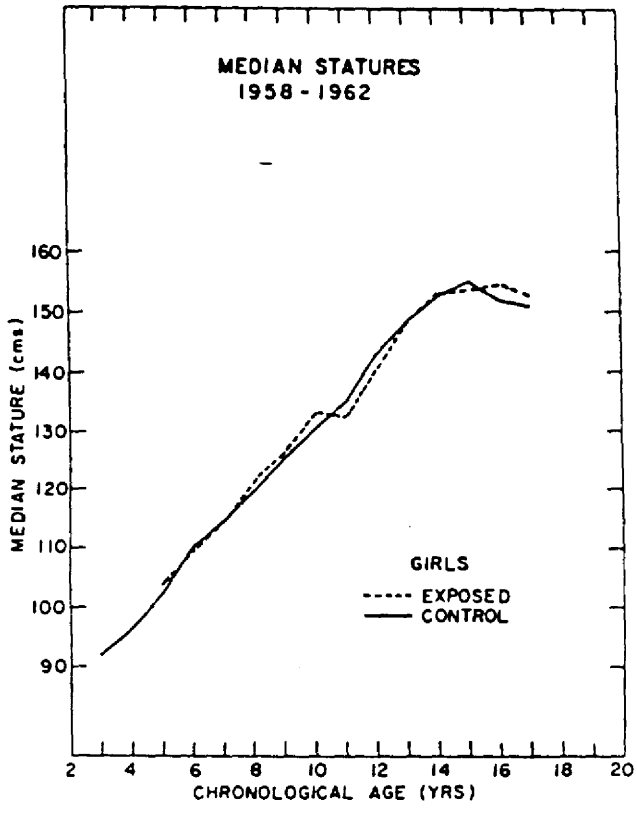

Figure 6.

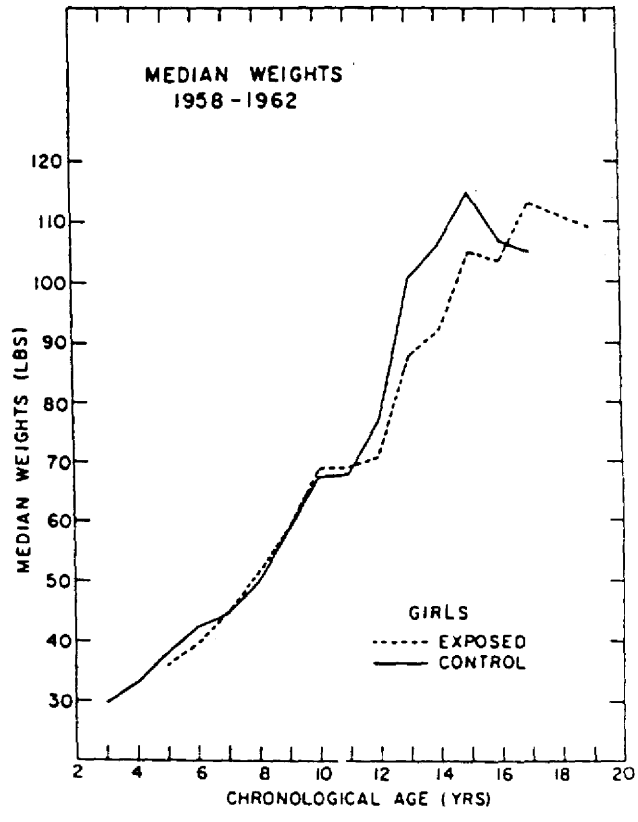

Figure 7.

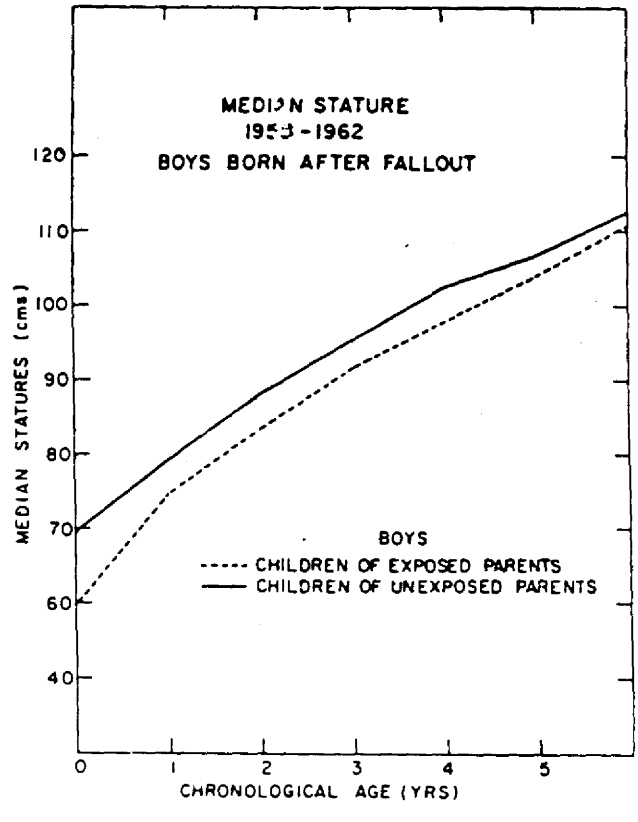

Figure 8.

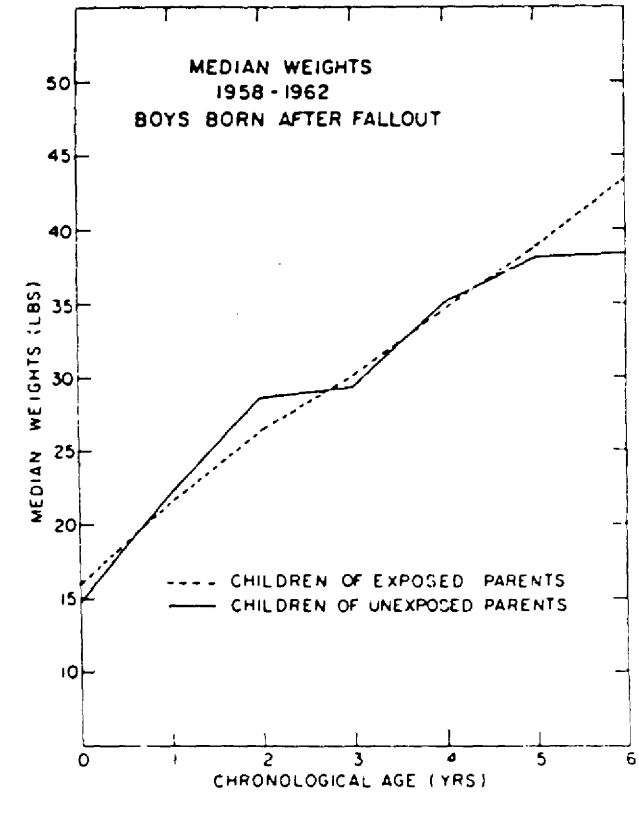

Figure 9.

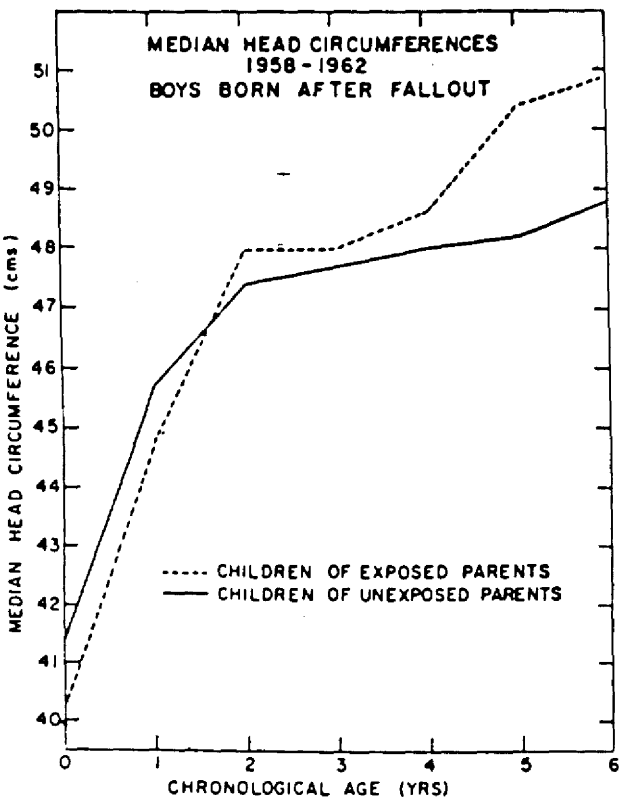

Figure 10.

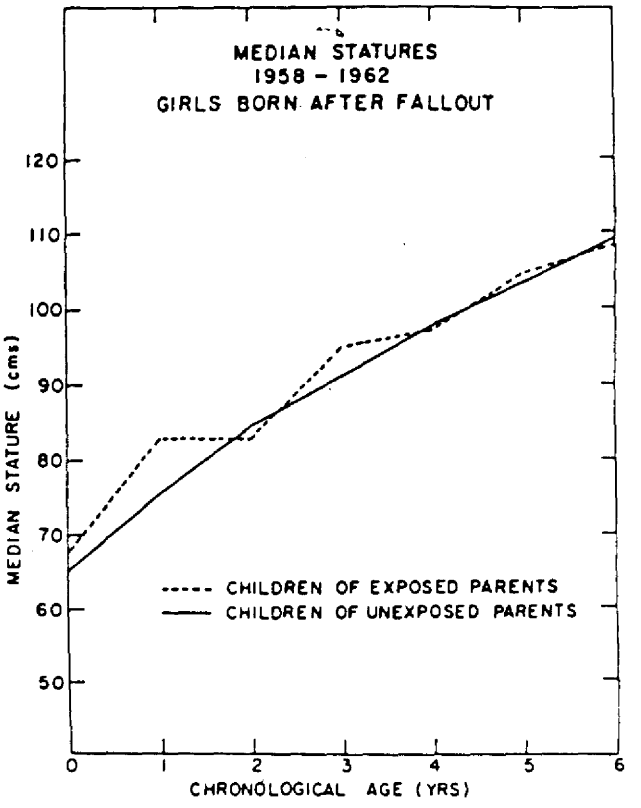

Figure 11.

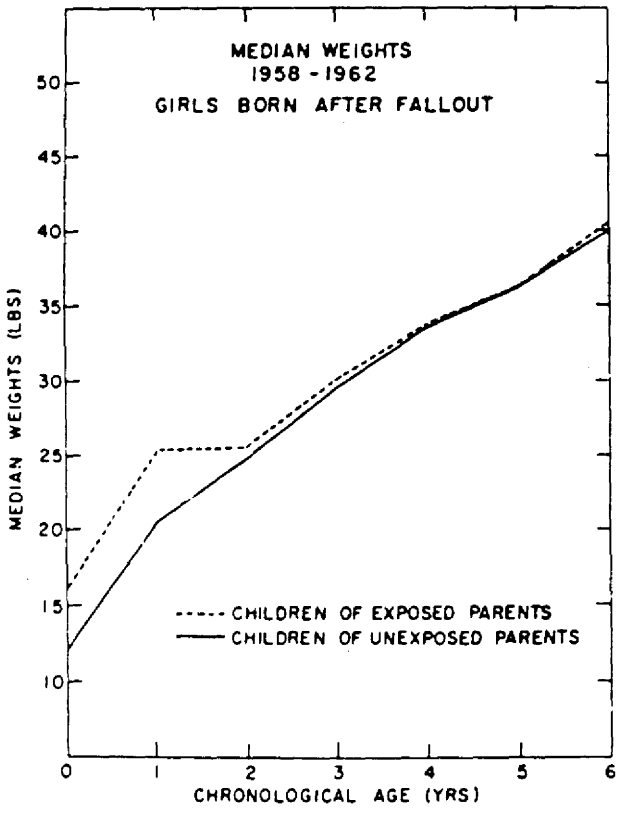

Figure 12.

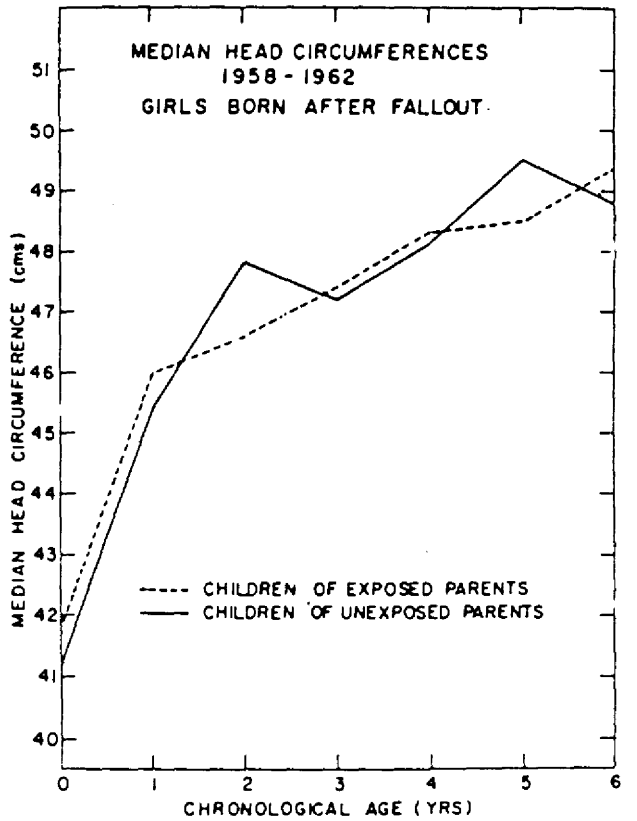

Figure 13.

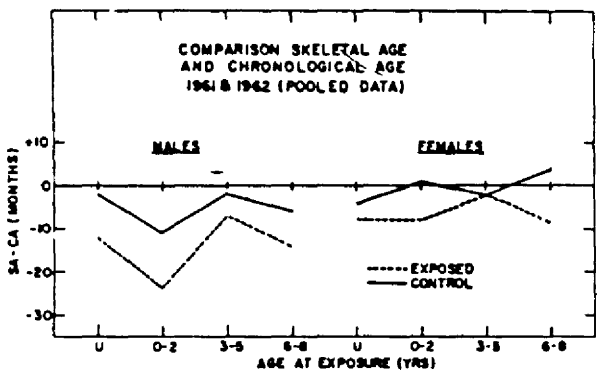

Figure 14.

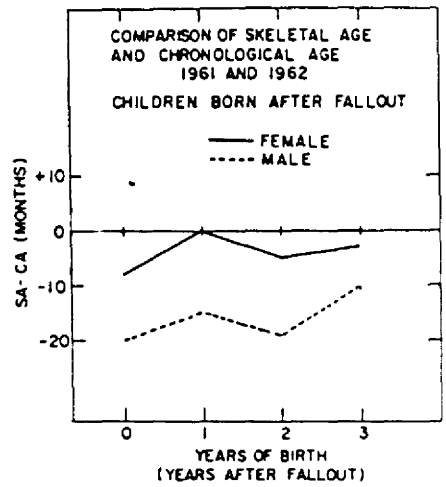

Figure 15.

13 months retarded skeletally as compared with 6 months for the average unexposed boy. The average skeletal retardation for exposed girls was 7 months as compared with 2 months for the control girls. The most marked retardation of skeletal maturation, as well as the most marked statural retardation, occurred in the 4 boys who were exposed at 16 to 17 months of age (Table 8). In two of these, Subjects No. 2 and No. 6, the degree of skeletal and statural retardation remained relatively constant from one year to the next: they were behind in their development but were progressing at approximately the same rate as their peers. However, the other two boys, Subjects No. 5 and No. 3, not only were more severely retarded in their development, but were retarded in the rate of their development, so that they fell further behind their peers each year. From age 6 to age 9, No. 5 gained only 3 months in skeletal age, and at age 9 is $5\frac{8}{12}$ yr behind the standards of Greulich and Pyle. During the same 3-yr period he gained only 8.7 cm in stature, while the controls gained 16.5 cm, and he now is 20.8 cm shorter than the controls. Similarly, No. 3 gained only 2 months in skeletal age from age 6 to age 9, which puts him $6\frac{8}{12}$ yr behind the standards of Greulich and Pyle, and he gained only 8.2 cm in stature so that he is 17.9 cm shorter than his peers. With respect to weight, No. 2 and No. 6 have maintained weights <1 yr behind their peers. No. 5 and No. 3 have had decreased rates of weight gain, but the decrease has not been as marked as in statural development. No. 3 is 6¼ lb lighter than his controls, corresponding to children ≈ 1 yr younger than himself, whereas his stature is comparable to that of children >3 yr younger. No. 5 is 11¼ lb lighter than his controls, corresponding to children ≈ 2 yr younger, and his stature is comparable to that of children almost 4 yr younger.

The skeletal ages of the children born after the fallout were also somewhat retarded according to the standards of Greulich and Pyle (Figure 15). This was attributable primarily to the boys, who have significantly more retarded skeletal ages than the girls, the average retardation being 14 months for the boys as compared with 2 months for the girls. There were no differences between children of exposed parents and children of unexposed

Table 8

Skeletal Age and Stature in Males Exposed at Age 16 to 17 Months

Subject No.	Age at exposure, months	Skeletal age minus chronological age, months			Height minus median control height, cm				
		Age 6	Age 8	Age 9	Age 5	Age 6	Age 7	Age 8	Age 9
2	16	−22	−28	−19	−2.3	−3.5	−1.9	−2.8	−3.1
6	16	−11	−20	−11	−4.9	−5.5	−5.7	−6.3	−8.4
5	16	−35	−59	−68	−9.6	−13.0	−15.3	−17.9	−20.8
3	17	−44	−66	−78	−6.8	−9.6	−10.8	−14.5	−17.9

parents. It was interesting that the boys born soon after the fallout had more retarded skeletal ages than those born more recently, and that the skeletal ages of all boys were slightly more retarded in 1962 than in 1961 (15 months as compared with 13 months). However, the differences were not statistically significant, and the data available at this time would not support any conclusion concerning factors related to the retardation of skeletal maturity in these boys.

CANCER DETECTION, LEUKEMIA SURVEY

Examinations as thorough as possible under field conditions were carried out for the detection of malignancy. All tumors were recorded. Most of the tumors seen were of the benign type of the skin and subcutaneous tissues. One exposed woman (No. 64) had a tumor of the fourth left phalanx; this was surgically removed, and histological examinations showed it to be a benign giant cell type. 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 carried out on vaginal and cervical smears in those women that had not had the procedure done during the past survey. None was positive for malignant cells. As noted last year, 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. A number of cases of endocervical atypia and trichomonas infection were noted. The results of the individual examinations are reported in Appendix 5.

Rectal examinations were carried out on all adults, and proctoscopic examinations were done when indicated. Several cases of prostatic enlargement were noted but no evidence of malignancy was apparent.

In view of the increased incidence of leukemia reported in the Japanese and others exposed to radiation, careful examinations for this blood dyscrasia are carried out annually on the Marshallese. On physical examination, no evidence of lymphadenopathy, splenomegaly, or other signs of leukemia were detected. Hematological examinations showed no excessive leukocytosis or increased numbers of immature leukocytes. Basophil counts on 4000 leukocytes on each individual showed no elevation except in one 9-yr-old exposed boy who has shown about a 3% basophil count over the past few years. However, in this case no other evidence of incipient leukemia has been apparent. Alkaline phosphatase studies on the blood smears show that although some people had low levels, this finding was not associated with any other findings suggestive of leukemia (see Appendix 4).

RESIDUAL BETA BURNS

Table 9 lists those cases that showed residual skin changes in areas previously exhibiting beta burns. Variation in pigmentation, hyperkeratosis, atrophy, and scarring in varying degrees were noted in these cases. The development of pigmented macular and papular (nevus-like) lesions in areas of the skin that had sustained beta burns was first observed several years ago. These lesions appear to be increasing slightly in number. Similar lesions are also occasionally seen in the unexposed group. Figure 16 shows the appearance of acute beta burns in an exposed woman several weeks after exposure and Figure 17 shows development of pigmented nevus-like lesions at 8 yrs post exposure in the skin area previously involved with beta burns. A nevus was removed in one case because of a complaint of irritation by clothing. The histological report on this lesion was as follows: "Fragment of skin with abundant pigment in the epithelium. In a small area, there is a superficial infiltration of dermis composed of nevus type cells and pigmentary cells. No evidence of malignancy. Diagnosis: nevus pigmentosus."

None of the residual changes noted in the skin showed any tendency to chronic radiation dermatitis or ulceration and none showed any gross evidence of malignant change. During the next survey it is planned to carry out an extensive biopsy program with histological study of skin lesions.

STUDIES OF AGING CRITERIA, DENTAL SURVEY

Studies of aging criteria and a dental survey were not carried out during this annual examination.

Table 9

Residual Beta Burns

Subject No.	Sex	Age	Description
2	M	9	Pigment variation with roughening of skin of front of neck. Several pigmented macules right antecubital fossa.
11	M	58	Slight hyperpigmentation and hyperkeratosis of left antecubital fossa. A few pigmented papular lesions on chest inferior axillae.
14	F	33	Back of neck — areas of slight increased pigmentation and hyperkeratosis.
17	F	11	Left antecubital fossa — scarring and hyperpigmentation; 2 small pigmented macules.
24	F	21	Dorsum of feet—mottled areas of pigmentation and depigmentation.
26	M	20	Dorsum right foot—considerable scarring and thickening with binding to subcutaneous tissue in area between 1st and 2nd toes. Slight scarring and hyperkeratosis with a few pigmented spots in right antecubital fossa.
34	F	53	Back of neck — slight hyperkeratosis with several nevus-like lesions on back and sides of neck.
39	F	22	Back of neck—pigment variation with slight hyperkeratosis; also to lesser extent on dorsum of feet.
49	F	23	Sides of neck — pigmented macules, particularly on right side; few also in left antecubital fossa.
54	M	9	Anterior neck — mottled pigmentation and depigmentation. Few pigmented macules left anterior axillary fold and lower abdomen; pigmented patch 2 × 3 cm interscapular area on left.
59	F	42	Back of neck — hyperkeratosis with some itching.
63	F	44	Considerable pigment variation on back of neck and to a slight degree on right forearm.
65	F	9	Front of neck—slight pigment variation and hyperkeratosis.
67	F	22	Dorsum of feet — atrophy and scarring.
71	F	36	Macular lesions on right side of neck and one also on back of neck.
78	F	44	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 17).
79	M	47	Back of left ear—scarring with 2 nodules of hyperkeratosis; pigment variation. Small spots of alopecia occipital region of scalp.

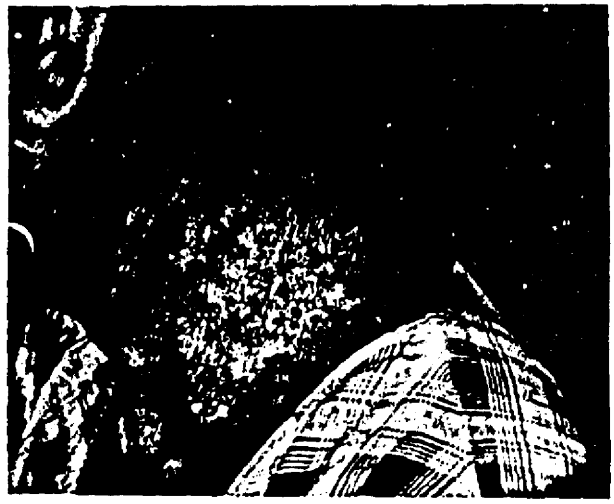

Figure 16. Wet dermatitis from beta exposure in Rongelap woman 4 weeks after exposure (Subject No. 78, March 29, 1954).

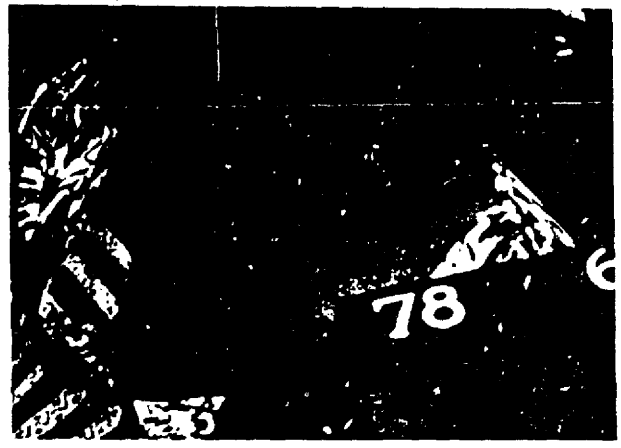

Figure 17. Same case as in Figure 16 at 8 yr post exposure. Note development of nevus-like lesions in area of neck previously involved with beta burn.

Laboratory Examinations

HEMATOLOGICAL

Summary tables of hematological data are presented in the tables and graphs in the text, and raw data on the 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, their data were not treated as fully as those for the larger groups, and are briefly summarized in a separate paragraph. 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 of age and sex groups. Ages 8 to 15, 16 to 40, and >40 yr for each sex are compared.

The hematological data are summarized in Tables 10 and 11 and in Figures 18 through 37. In Appendices 1 and 2 are presented summaries of the mean blood counts of the exposed population and of the various comparison populations since exposure in March 1954. In Appendix 3 are listed the individual blood counts for 1962. In Appendix 4, the individual alkaline phosphatase and basophil counts are presented.

Leukocytes

The leukocyte levels in both exposed and unexposed groups were slightly lower than last year (see Table 10 and Figure 18). The mean leukocyte level in the exposed group was slightly (12%) below that in the unexposed group. This was largely a reflection of deficits in the neutrophil and lymphocyte levels.

Neutrophils

The mean neutrophil level in the Rongelap exposed group was 13.6% below that in the unexposed group (see Table 10 and Figure 18). The levels were lower in the younger (age 8 to 15) and older (age >40) groups than in the middle (age 15 to 40) group. This is demonstrated in the histogram (Figure 30) and in the scattergrams (Figures 19 and 20), the latter showing more than half the individual counts below the mean line of the unexposed. An accumulative percentage distribution curve (Figure 21) also shows the lower counts (displacement of the curve to the left) in the exposed group. These findings conform generally with previous data except that the younger females tend to show more depression.

Lymphocytes

The mean absolute lymphocyte level in the exposed is 24% below that of the unexposed, which indicates a deficit greater than the mean neutrophil deficit (see Table 10 and Figure 22). The lymphocyte levels appear to be more depressed this year. The histogram (Figure 30) shows that the deficit in lymphocytes seems to be distributed fairly evenly in the age and sex groups, except that (as noted with neutrophils) a somewhat larger deficit appears among the younger females. The scattergrams (Figures 23 and 24) and the accumulative distribution curve (Figure 25) also confirm these observations.

Eosinophils, Monocytes, and Basophils

These all showed nearly the same mean levels in both groups. The general persistence of eosinophilia in both groups was apparent, as in past surveys.

Platelets

Mean platelet counts in both exposed and unexposed groups were higher this year than previously (Figure 26). The male counts this year averaged about the same as the female; previously the female counts had been higher. As noted consistently in the past, mean counts in both males and females in the exposed group were slightly below the corresponding unexposed levels (males by 5.7%, females by 10%). Also as previously noted, the younger and older groups showed the greater deficit (see histogram, Figure 30). Scattergrams (Figures 27 and 28) show that the majority of

Table 10

Mean Levels of Peripheral Blood Elements of Exposed Groups Compared With Those of Unexposed Groups by Age and Sex

	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, 8-15 yr							
Rongelap exposed**	393±120(10)*	7.90±1.9 (9)	3.98±1.4 (9)	2.88±0.6 (9)	0.16 (9)	0.84 (9)	0.46 (9)
Alingnae exposedt	430 (2)	6.98 (3)	3.64 (3)	2.50 (3)	0.08 (3)	0.77 (3)	0 (3)
Rongelap unexposed	415± 86(16)	8.12±1.9 (16)	4.31±2.1 (16)	3.15±0.9 (16)	0.17(16)	0.49(16)	0.10(16)
Females, 8-15 yr							
Rongelap exposedt	324± 77 (9)	6.86±1.03 (9)	3.55±1.37 (9)	2.69±0.8 (9)	0.18 (9)	0.42 (9)	0.20 (9)
Ailingnae exposed	315 (2)	7.25 (2)	4.06 (2)	2.59 (2)	0.21 (2)	0.32 (2)	0.30 (2)
Rongelap unexposed	390± 80(20)	9.08±2.41(20)	4.71±1.97(20)	3.45±0.88(20)	0.26(20)	0.62(20)	0.30(20)
Males, >15-40 yr							
Rongelap exposed	320±120(12)	6.65±1.26(12)	3.48±1.35(11)	2.60±0.78(11)	0.15(11)	0.35(11)	0.20(11)
Ailingnae exposed							
Rongelap unexposed	327± 87(29)	6.99±1.43(29)	3.49±1.08(29)	2.92±0.70(29)	0.18(29)	0.34(29)	0.20(19)
Females, >15-40 yr							
Rongelap exposed	350± 78(13)	6.86±2.15(12)	3.81±1.31(12)	2.26±0.48(12)	0.14(12)	0.63(12)	0.10(12)
Ailingnae exposed	350±121 (4)	5.70±1.39 (4)	3.01±1.27 (4)	2.32±0.90 (4)	0.09 (4)	0.25 (4)	0.40 (4)
Rongelap unexposed	340±140(27)	7.80±2.11(27)	4.41±1.90(26)	2.64±0.95(26)	0.22(26)	0.44(26)	0.10(26)
Males, >40 yr							
Rongelap exposed	266± 80 (9)	6.10±1.80 (9)	3.35±1.57 (9)	2.19±0.78 (9)	0.14 (9)	0.33 (9)	0.40 (9)
Ailingnae exposed	286± 43 (4)	6.01±1.27 (4)	2.89±0.62 (4)	2.73±0.87 (4)	0.13 (4)	0.24 (4)	0.20 (4)
Rongelap unexposed	326± 95(20)	7.14±2.28(20)	4.13±2.13(20)	2.49±0.79(20)	0.13(20)	0.38(20)	0.20(20)
Females, >40 yr							
Rongelap exposed	276± 99 (9)	6.37±1.89 (9)	3.50±1.28 (9)	2.45±0.60 (9)	0.18 (9)	0.31 (9)	0.50 (9)
Ailingnae exposed	309±122 (4)	7.26±0.99 (4)	4.02±1.24 (4)	2.69±0.85 (4)	0.19 (4)	0.34 (4)	0.20 (4)
Rongelap unexposed	306± 85(19)	7.70±2.04(19)	4.40±1.44(19)	2.82±0.90(19)	0.14(19)	0.34(19)	0.10(19)

	Hct., %	RBC ($\times 10^{-4}$)	Hgb., g	Serum protein, g
Males, 8-15 yr				
Rongelap exposed**	38.5±3.3(10)	468±56(10)	12.8±1.4(10)	8.0±0.4 (8)
Ailingnae exposedt	37.0 (3)	451 (3)	12.3 (3)	7.7 (2)
Rongelap unexposed	38.3±2.8(16)	460±35(14)	12.9±0.7(16)	7.8±0.3(11)
Females, 8-15 yr				
Rongelap exposedt	39.3±2.0 (9)	478±32 (5)	13.6±1.0 (9)	7.9±0.3 (9)
Ailingnae exposed	40.0 (2)	455 (2)	13.6 (2)	7.7 (2)
Rongelap unexposed	39.0±1.6(20)	455±24(19)	12.9±0.8(20)	7.8±0.3(12)
Males, >15-40 yr				
Rongelap exposed	44.8±3.3(12)	488±75 (9)	15.1±1.2(12)	7.6±0.5(12)
Ailingnae exposed				
Rongelap unexposed	45.2±2.8(29)	508±39(27)	15.0±0.9(29)	7.6±0.5(28,
Females, >15-40 yr				
Rongelap exposed	40.3±4.0(13)	449±32(11)	13.5±1.2(13)	8.0±0.6(13)
Ailingnae exposed	35.0±3.7 (4)	434 (3)	11.4±2.4 (4)	8.1±0.3 (4)
Rongelap unexposed	38.8±2.8(27)	451±33(26)	12.9±1.0(27)	7.8±0.4(27)
Males, >40 yr				
Rongelap exposed	40.6±6.2 (9)	444±81 (8)	13.7±1.9 (9)	7.3±0.7 (9)
Ailingnae exposed	42.5±2.7 (4)	512±49 (4)	14.6±0.8 (4)	7.4±0.4 (4)
Rongelap unexposed	42.4±3.0(20)	467±38(20)	14.1±1.1(20)	7.8±0.5(20)
Females, >40 yr				
Rongelap exposed	37.9±3.7 (9)	413±22 (7)	12.4±1.0 (9)	7.7±0.9 (9)
Ailingnae exposed	39.5±1.8 (4)	426±32 (4)	13.5±0.8 (4)	8.0±0.3 (4)
Rongelap unexposed	39.3±2.7(19)	432±30(17)	13.1±0.9(19)	7.7±0.5(19)

*Standard deviation and number of people in group.

**Includes 2 children exposed *in utero*.

†Includes 1 child exposed *in utero*.

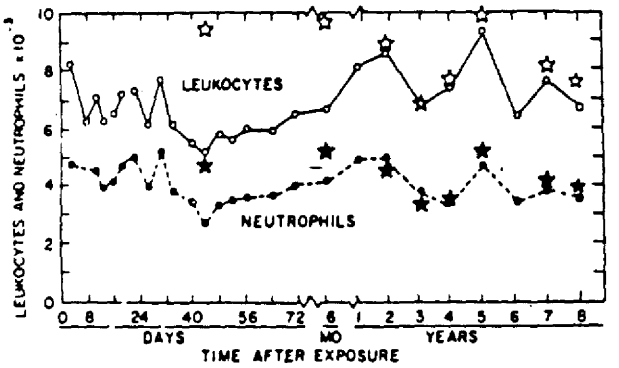

Figure 18. Mean neutrophil and white blood counts of exposed Rongelap people from time of exposure through 8 yr post exposure. Stars represent mean values of comparison population.

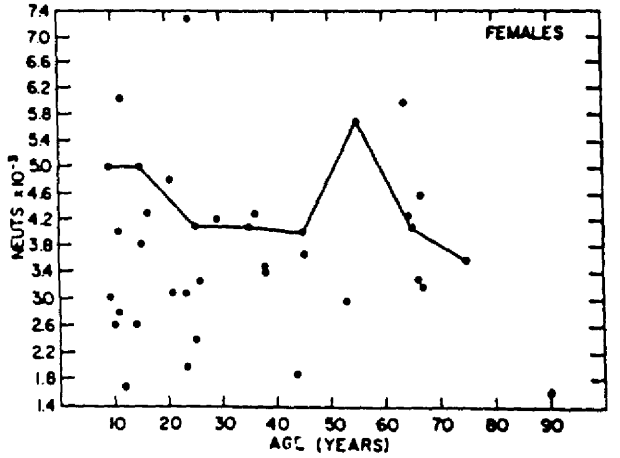

Figure 20. Neutrophil counts of exposed Rongelap females plotted against age. Solid line represents mean level of unexposed female population.

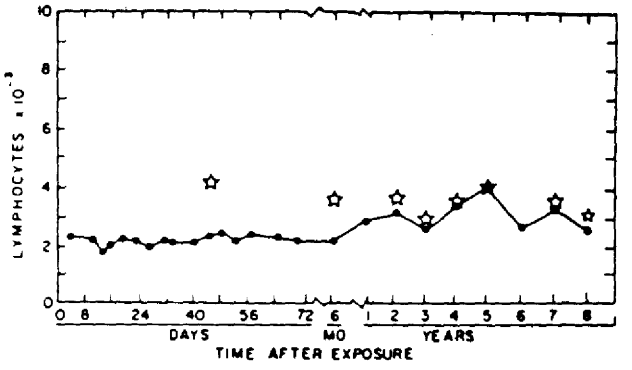

Figure 22. Mean lymphocyte counts of exposed Rongelap people from time of exposure through 8 yr post exposure. Stars represent mean values of comparison population.

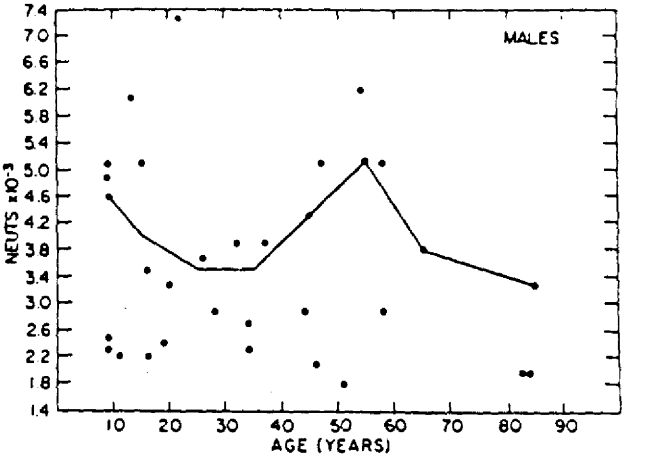

Figure 19. Neutrophil counts of exposed Rongelap males plotted against age. Solid line represents mean level of unexposed male population.

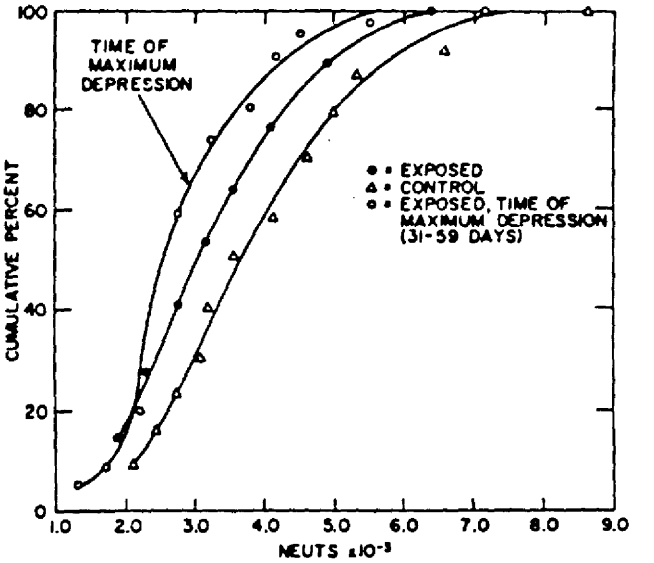

Figure 21. Neutrophil cumulative percent distribution curves.

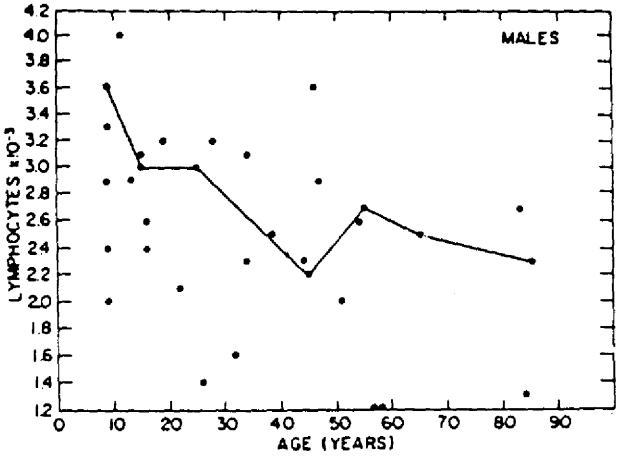

Figure 23. Lymphocyte counts of exposed Rongelap males plotted against age. Solid line represents mean level of unexposed male population.

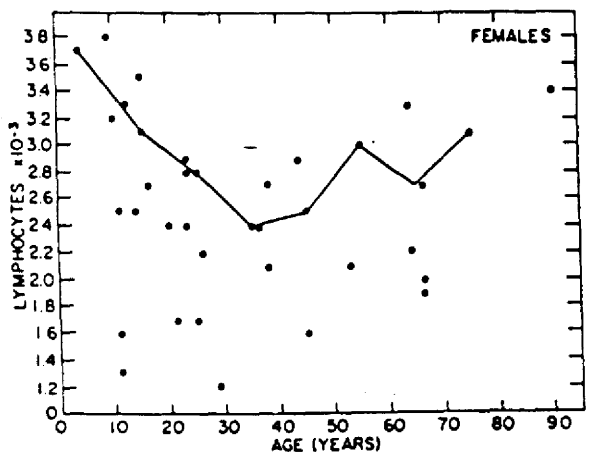

Figure 24. Lymphocyte counts of exposed Rongelap females plotted against age. Solid line represents mean level of unexposed female population.

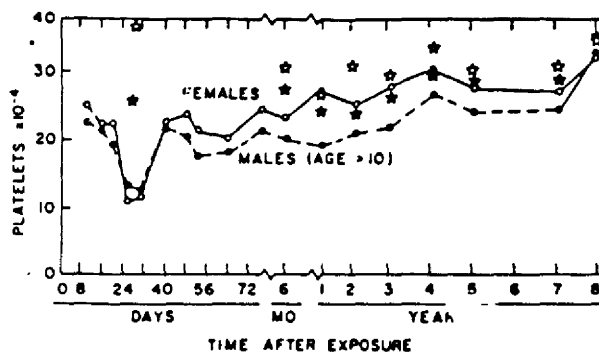

Figure 26. Mean platelet values of exposed Rongelap people from time of exposure through 8 yr post exposure. Stars represent mean values of unexposed comparison population.

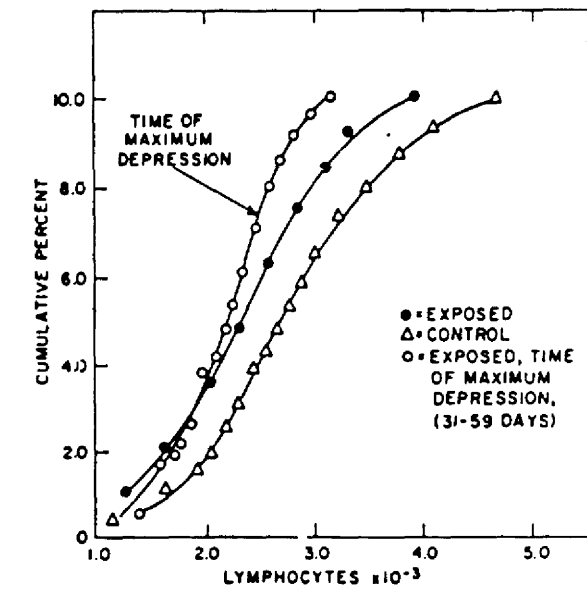

Figure 25. Lymphocyte cumulative percent distribution curves for exposed compared with unexposed population.

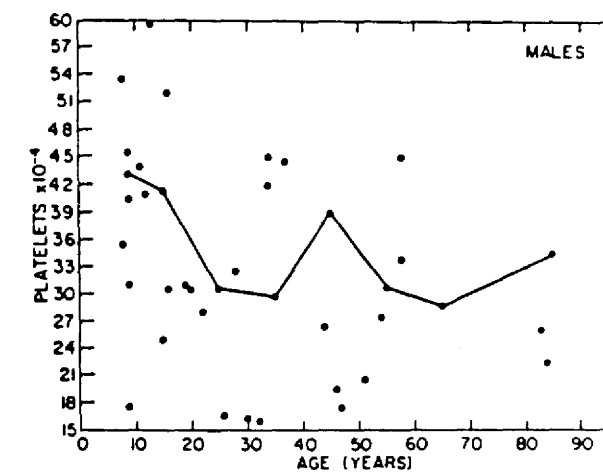

Figure 27. Platelet counts of exposed Rongelap males plotted against age. Solid line represents mean level of unexposed male population.

counts in males and females were below the mean line of the unexposed group. An accumulative distribution curve shows displacement of the exposed curve to the left of the unexposed (Figure 29). The increase in platelet counts this year in both exposed and unexposed groups is demonstrated by the fact that only 11.3% of the exposed group and 5.3% of the unexposed show counts below 200,000, as compared with 18% and 9.7% last year.

Erythropoietic Elements

Erythrocyte, hemoglobin, and hematocrit levels showed no marked differences between the exposed and unexposed groups (see Table 10 and Figures 31 through 36). In the older groups the values were slightly lower in the exposed than in the unexposed groups.

Ailingnae Blood Counts

Hematological examinations were carried out on all 17 Ailingnae people. The levels of peripheral blood elements in this group were generally the same as in the exposed Rongelap group. These data are shown in Table 10 and Appendices 2 and 3.

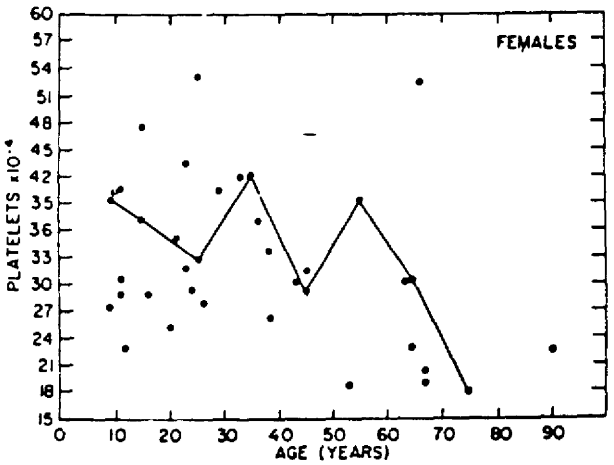

Figure 28. Platelet counts of exposed Rongelap females plotted against age. Solid line represents mean level of unexposed female population.

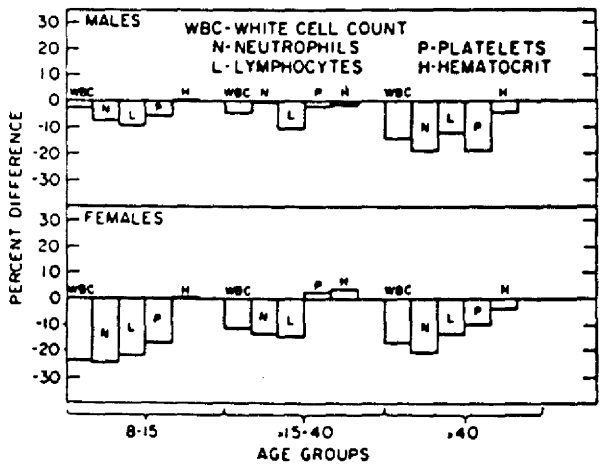

Figure 30. Sex and age distribution of percent difference of peripheral blood elements in exposed compared with unexposed groups.

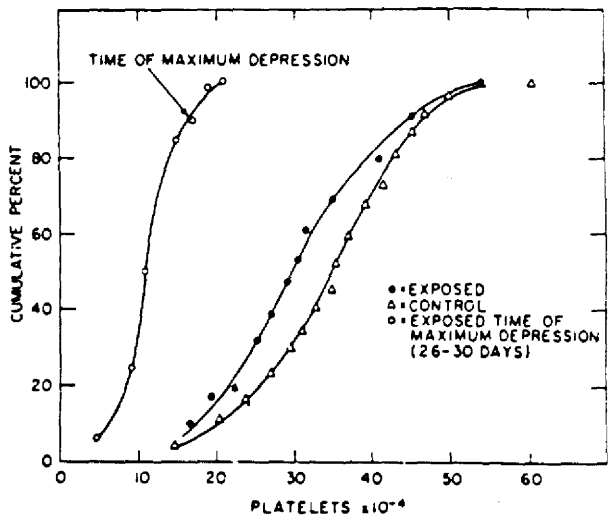

Figure 29. Platelet cumulative percent distribution curves for exposed compared with unexposed population.

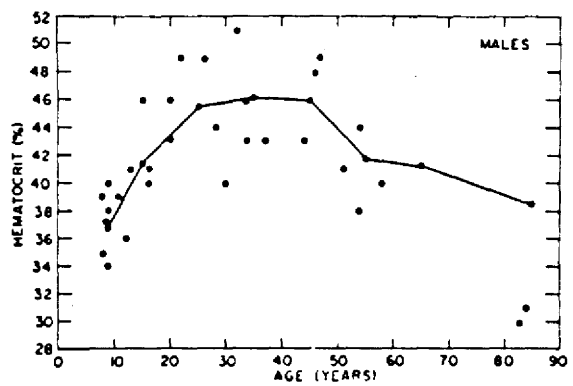

Figure 31. Hematocrit values of exposed males plotted against age. Solid line represents mean level of unexposed male population.

Morphology of Blood Cells

Examination of peripheral blood smears showed no unusual cellular morphology in either the exposed or controls. A few bilobed lymphocytes were seen in the 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 in the red cells. Bone marrow smears were obtained on 9 exposed Rongelap people. These examinations showed a reduced M/E ratio in 8 of 9 cases with abnormalities of the erythroid and myeloid precursors in 5 marrows (see Table 11).

Children of Exposed Parents

Peripheral blood elements of 24 children with parents one or both of whom had been exposed were compared with those of 50 children with unexposed parents. These children were <8 yr of age. The results are presented in Table 12 and Figure 37. There appeared to be a slight depression of leukocytic elements in both male and female children of exposed parents but a slight increase in platelets. Thus the findings reported last year of male children (but no female) of exposed parents showing depression of all blood elements is not substantiated in this year's findings.

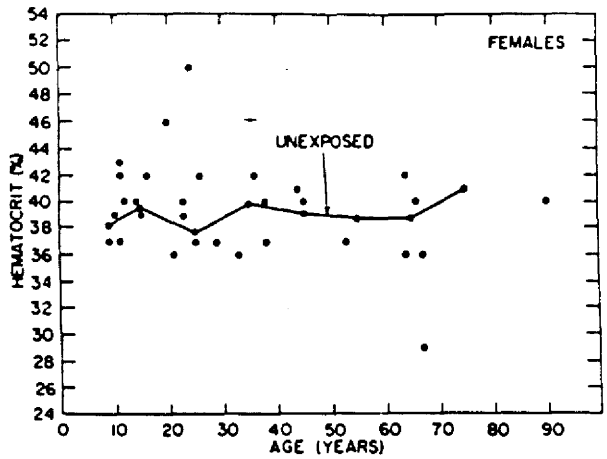

Figure 32. Hematocrit values of exposed females plotted against age. Solid line represents mean level of unexposed female population.

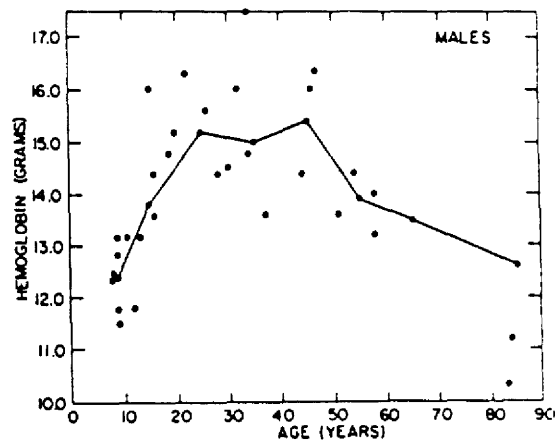

Figure 33. Hemoglobin values of exposed males plotted against age. Solid line represents mean level of unexposed male population.

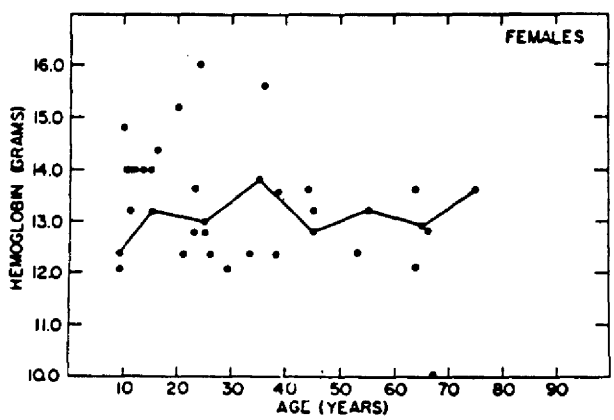

Figure 34. Hemoglobin values of exposed females plotted against age. Solid line represents mean level of unexposed female population.

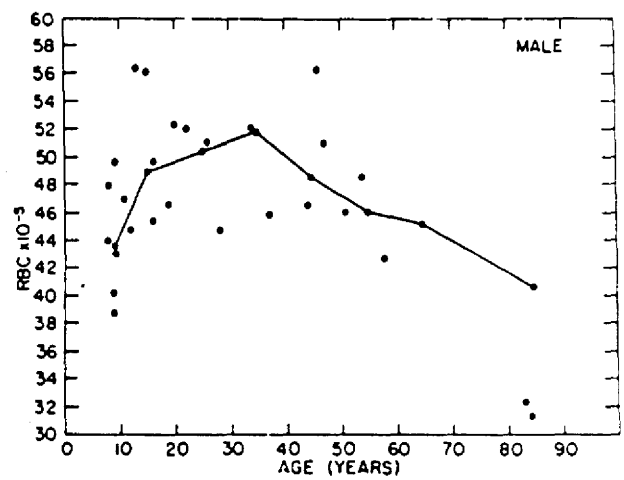

Figure 35. RBC values of exposed males plotted against age. Solid line represents mean level of unexposed male population.

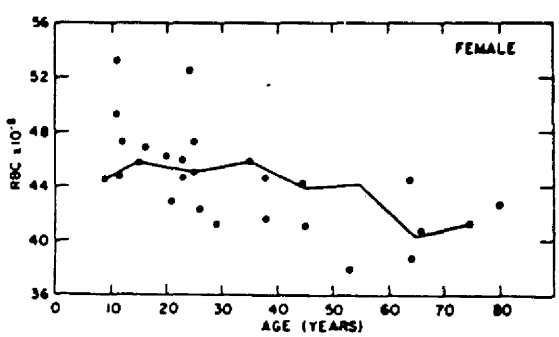

Figure 36. RBC values of exposed females plotted against age. Solid line represents mean level of unexposed female population.

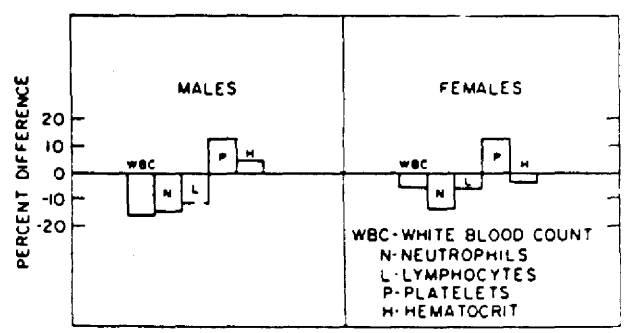

Figure 37. Sex and age distribution of percent differences of peripheral blood elements in children of exposed parents (one or both exposed) compared with children of unexposed parents.

Table 11

Peripheral Blood and Bone Marrow Findings

Subject No.	Hgb., g	Hct., %	WBC	Differential	Bone marrow smear
27	18	41	6,100	normal	Erythroid hyperplasia with increased immature red and myeloid elements.
7	14.4	43	6,500	15% eosin.	As in #27.
40	13.6	46	6,600	normal	No abnormalities.
50	15.7	46	7,500	normal	Changes as in #27 but in less degree.
52	13.8	42	9,700	normal	Slight increase in immature red and myeloid elements.
30	10.0	29	7,100	normal	As in #52.
53	13.2	38	7,900	normal	Normal.
66	13.6	40	6,400	normal	Normal.
80	14.4	44	9,100	normal	Erythroid and myeloid immaturity.

Table 12

Mean Levels of Peripheral Blood Elements of Children of Exposed Parents Compared With 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, <8 yr							
Of exposed parents	511±123(15)*	8.74±1.34(15)	4.17±0.87(15)	3.92±1.00(15)	0.15(15)	0.49(15)	0.07(15)
Of unexposed parents	451± 80(29)	10.37±2.55(29)	4.89±1.68(29)	4.42±1.50(29)	0.30(29)	0.73(29)	0.40(29)
Females, <8 yr							
Of exposed parents	473±149(19)	10.98±2.41(19)	4.72±1.47(19)	5.01±1.36(19)	0.30(19)	0.84(19)	0.30(19)
Of unexposed parents	419± 97(21)	11.63±2.50(21)	5.35±2.16(21)	5.28±1.92(21)	0.26(21)	0.70(21)	0.40(21)

	Hct., %	RBC ($\times 10^{-6}$)	Hgb., g	Serum protein, g
Males, <8 yr				
Of exposed parents	38.4±3.0(15)	456±35(13)	12.0±0.9(15)	
Of unexposed parents	36.7±2.5(29)	457±39(28)	11.8±1.0(29)	
Females, <8 yr				
Of exposed parents	36.7±2.8(19)	454±37(17)	11.7±1.2(19)	7.6(1)
Of unexposed parents	37.8±2.7(21)	458±31(16)	12.2±0.9(21)	

*Standard deviation and number of people in group.

Comments on Hematological Data

The 1962 leukocyte levels in both exposed and unexposed groups were generally slightly lower than last year. The level in the exposed group was about the same as in 1960; no hematological examinations were carried out on the unexposed population at that time. Fluctuations in mean leukocyte levels from year to year, which have been commented on previously, have been unexplained thus far. In contrast to the lowering of leukocyte levels this year, it was noted that the platelets and erythropoietic elements in all groups were elevated as compared with last year, the platelets having

Table 13

Peripheral Blood Cultures

Subject No.	Total counts	Diploid numbers 10	22	32	36	37	39	40	41	42	43	44	45	46	47	48	67	pp
10	59						2	2		2	7	3	4	33 (56%)	5	1		
11	24		1									2		20 (80%)	1			
14	26											1		22 (84%)	1	1		1
27	30										1	1	4	18 (60%)	5	1		
41	27									2				25 (92%)				
50	69			1	1	1		1		4	1			51 (74%)	6	2	1	
58	32										1		1	29 (91%)				
69	19	1												18 (95%)				
79	19								2		1		1	15 (79%)				
80	16				1	1			1		1		1	8 (50%)	1			

reached the highest levels yet recorded. The general anemic tendency in the people is no longer as pronounced. Perhaps a better diet (increased amounts of fruits and vegetables) may partly account for this change.

Comparison of hematological data in the exposed and unexposed groups demonstrates a deficit in total leukocytes and absolute neutrophils and lymphocytes which is even slightly more pronounced this year than last year. Platelet levels in the exposed group show slightly less deficit this year than in the past. As noted previously, this deficit in leukocytes and platelets is more apparent in the younger and older groups. Erythrocyte counts and hematocrit and hemoglobin levels were again found to be about the same in the exposed as in the unexposed group.

In view of these findings in the peripheral blood and the evidence of slight persisting bone marrow abnormalities, it does not seem unreasonable to assume that there is some residual effect of the radiation exposure on hematopoiesis.

The finding of slightly lower leukocyte levels in the male and female children of exposed parents is difficult to evaluate.

OTHER LABORATORY STUDIES

Chromosome Studies

Although moderate to good growth of peripheral blood cultures was obtained in samples from 13 of 20 unexposed and 30 of 51 exposed people, the final chromosome smears unfortunately yielded countable metaphases in only 10 preparations in the exposed group. The difficulty in obtaining good smears is believed to have been related to the high temperature and humidity of the Islands, which interfered with rapid drying of the smears. This was particularly disappointing since excellent smears had been obtained in preliminary perfection of the technique under laboratory conditions at BNL. Similar difficulties with chromosome spreads and poor staining were encountered in 9 direct bone marrow metaphase studies. Nevertheless, small numbers of dicentrics were noted in some of these preparations along with abnormal diploid numbers (see Tables 13 and 14). These preliminary findings suggest that persistent damage to blood cells occurred in these people. In view of the importance of these studies, a concerted effort will be made on the next survey to obtain a large number of satisfactory preparations from peripheral blood and also from cultures of the skin in areas previously involved with beta burns. It is believed that the difficulties encountered in the field in making the chromosome smears can be corrected by using a hot air dryer on the next survey.

Routine Urine Analyses

Routine urine analyses were carried out on the majority of the people. Ten of the samples were positive for sugar. Several of these showed only

Table 14

Abnormal Chromosomes in Peripheral Blood Cultures

Subject No.	Total counts	Dicentrics	Other
64	2	—	—
10	59	—	—
11	24	—	—
14	26	1	—
27*	30	1	1 minute
41	27	—	—
50	69	—	—
58	32	—	—
69	19	—	—
79	19	—	—
80	16	—	—

*Bone marrow smears showed 3 dicentrics on scanning.

small amounts, however, and glycosuria was demonstrated only in those that showed urine elevations of 2+ to 4+. Four urines showed a slight amount of protein, but other examinations did not reveal abnormalities which might be associated with proteinuria.

Blood Sugar Determinations

Fasting blood sugar analyses were carried out on 8 people (all in the unexposed group) who had shown urine positive for sugar on the previous survey. Of these, 4 showed elevated levels (Nos. 853, 884, 893, and 991). Non-fasting blood sugar determinations were carried out routinely on 72 people in the exposed and 125 in the unexposed groups. Elevations >160 mg % were found in 4 of these, 1 exposed (No. 29) and 3 unexposed (Nos. 932, 936, and 1042). The somewhat higher incidence of diabetes in the Marshallese people has been commented on in previous reports.

Protein-Bound Iodine

Since previous survey results had shown protein-bound iodine levels on the high side of normal, 14 sera were obtained on individuals for repeat analyses this year. The levels varied between 4.6 and 12.0 μg % with a mean of 8.6; these are again generally on the high side of normal.

Table 15

Blood Volume Studies (1961, 1962)

Subject	Race*	Wt., lb	Increase, cc		Decrease, cc	
			RBC	Plasma	RBC	Plasma
	C	158	—	—	100	750
	C	105	—	250	100	—
	C	153	—	—	400	600
	C	161	—	—	550	1000
	C	165	—	—	350	600
	C	156	—	—	250	100
	C	110	—	—	250	500
	M	172	—	—	250	500
	M	150	—	—	—	100
	M	138	100	—	—	—
	M	155	—	—	250	100
	M	122	—	—	550	600
	M	102	—	—	400	—
	M	109	—	—	300	200
	M	126	—	—	300	—
	M	135	—	—	200	150
	M	140	—	—	600	500
	M	123	123	100	—	—
	M	140	—	—	200	700
	M	132	—	—	800	770
	C	156	—	—	450	400
	C	183	—	—	400	700
	C	—	—	—	200	200

*C = Caucasian, M = Micronesian.

Total Urine Iodine and Creatinine

The purpose of these analyses was to determine whether the rather high protein-bound iodine levels reported in the Marshallese might be related to high iodine levels in the diet. Total iodine and creatinine analyses were carried out on 10 urine samples obtained from subjects who had previously shown relatively high protein-bound iodine levels. The levels for total iodine varied between 5.2 and 66.0 μg % (av, 18.6), and the creatinine levels varied between 0.025 and 0.80 g/l (av, 0.52). These levels were considered to be in the normal range, although the creatinine levels were somewhat high because of alkalinity of the urine samples. Therefore it did not appear that the iodine in the diet could account for the generally higher protein-bound iodine levels observed. The cause of this slight elevation remains to be clarified.

Table 16

Immunoelectrophoretic Analyses

Findings	Subject Nos.
Normal	15, 30, 20, 78, 8, 76, 92, 77, 9, 14, 33, 16, 2, 21, 57, 47, 4, 37, 10, 73, 12, 914, 835, 838, 875, 844, 865, 896, 982, 836, 832, 936, 955, 956, 957, 830, 960, 939, 893, 979, 840, 833, 915, 967, 975, 882, 898, 1005, 868, 822, 1042, 841, 825, 885
Slightly increased γ globulin precipitation-line	53, 71, 86, 52, 63, 22, 32, 87, 59, 54, 79, 6, 66, 1, 45, 928, 856, 883, 940, 1036, 944, 992, 858, 813, 864, 853, 887, 942, 829, 820, 828, 924, 961, 969, 895, 855, 884
Markedly increased γ globulin precipitation-line	862, 891
Slightly increased precipitation-lines for β_{2A}, β_{2M}, and γ	34, 75, 27, 817, 998, 888, 814
Markedly increased precipitation-lines for β_{2A}, β_{2M}, and γ	64, 958, 1035
Markedly increased precipitation-lines for β_{2M} and γ	829, 892, 1001
Slightly increased precipitation-lines for β_{2A} and γ	3, 41
Markedly increased precipitation-lines for β_{2A} and γ	897
Slightly decreased precipitation-line for β_{2M}	5, 900
Slightly increased precipitation-line for β_{2M}	29, 852
Decrease of β_{2A} and slight increase of γ globulin precipitation-lines	866
Decrease of β_{2A} and γ globulin precipitation-lines	60, 68, 824

Glucose-6-phosphate Dehydrogenase Activity and Hemoglobin Types

Dr. Boyer at Johns Hopkins Hospital reported that all subjects examined had normal glucose-6-phosphate dehydrogenase activity. On the starch gel electrophoresis all were of a uniform glucose-6-phosphate dehydrogenase electrophoretic type Class B. This is the type observed in all Americans of European ancestry and in 70% of Americans of mixed African and European ancestry.

Electrophoretic studies of hemoglobin showed that all Marshallese subjects examined had type AA_2.

Blood Volume Studies

During the 1961 and 1962 surveys, blood volume determinations, with use of Cr^{51}-labeled sodium chromate, were performed on 25 normal Micronesian and Caucasian persons living in the Marshall Islands for 1 yr or longer. Table 15 shows the data for 23 of these on red cell mass and plasma volume based on body weight. From these data it appears that there was a significant reduction in red cell mass and/or plasma volume in 15 of 23 subjects, both Marshallese and Americans. During the anticipated 1963 survey, it is planned to repeat these studies in conjunction with estimations of lean body mass by use of tritiated water. Evaluation of the above results will be withheld until completion of the 1963 survey.

Immunoelectrophoretic Studies

Immunoelectrophoretic analyses were carried out on a number of Marshallese sera. These results are shown in Table 16. Dr. R. Bütler, who carried out these analyses, reported that "in summary we have found neither a paraproteinemia nor a typical picture of antibody-deficiency-syndrome. The high frequency of increases of some of the immunoglobulins is perhaps a typical sign of the investigated population."

Estimation of Body Burden of Sr^{90}

Gamma spectrographic analyses on the Rongelap people in 1961 by whole-body counting in a 21-ton steel room indicated that the mean body burden of Cs^{137} had not shown any significant further increase over the 1959 level and that the mean Zn^{65} level had been reduced by a factor of about 6. Since these results implied that the body burdens had reached equilibrium with these gamma emitters in the environment, further gamma spectrographic analysis was deferred, the next being planned for about 1965. Therefore the steel room was not taken to Rongelap on this survey and no whole-body gamma counting was done. However, urine and other samples were collected and brought back for radiochemical analyses.

Sr^{90} and calcium analyses were carried out on 24-hr urine samples from 27 Rongelap people, 18 in the exposed group and 9 in the unexposed, and on a 12-liter pooled sample from the population at large; on vertebra and rib samples (autopsy specimens from the 78-yr-old woman who died in March 1962); and on 4 coconut crabs. The results of these analyses are presented in Tables 17, 18, and 19. The urinary levels of Sr^{90} are somewhat higher than last year.

The fluctuations in calcium level in the samples cause variations in the Sr^{90} level expressed per g Ca. The pooled urine samples contained 7.2 pC/l Sr^{90} in 1961 and 7.9 in 1962 (1 pC = 1 $\mu\mu$C), but the calcium levels differed so markedly (141 mg/l in 1961 and 18 in 1962) that they were not used in calculating the body burdens of Sr^{90}. In 1962 the mean Sr^{90} values from the individual adult 24-hr samples were 12.45 ± 1.30 pC/l or 114 ± 14 pC/g Ca. From these values, on the basis of previous calculations,[14] the body burden was estimated as 12.0 mμC. This is about 6.0% of the maximum permissible concentration (MPC) of Sr^{90} (200 mμC) for nonindustrial populations, and represents about 49% of the previously estimated[14] equilibrium value of 23 mμC.

Thus the return of the Rongelap people to their home island has been reflected in annual increases in estimated body burdens of Sr^{90} based on urinary excretion values. The estimates, in mμC, have increased as follows: 2.0 in 1958, 6.0 in 1959, 6.9 in 1961, and 12.0 in 1962. It is not clear why the greater increase occurred during the past year.

In view of the uncertainties associated with extrapolation from urinary excretion data to body burden of Sr^{90}, other corroborating evidence has been derived from analyses of bone samples. Because of the difficulty of obtaining autopsies on these people, this means of analysis has been very limited. However, bone samples were obtained from an autopsy on the 78-yr-old woman who died this year, and the Sr^{90} analysis of ribs and vertebral specimens showed an average of 15 pC/g Ca, representing a body burden of 11.4 mμC or about 5.7% of the MPC (see Table 19). This compares favorably with the estimate of 12.0 mμC based on the 24-hr urine samples. Bone samples from the autopsy of a 35-yr-old man in 1958 showed a Sr^{90} body burden of 2 mμC. The estimated body burden derived from the 1962 bone samples therefore shows about a sixfold increase over that from the 1958 samples.

The Sr^{90} levels in the coconut crabs in 1962 (Table 19) are close to those previously reported by Held.[15] It is not clear why the crabs continue to show such high levels, but the ban on their consumption cannot yet be lifted.

Summary

Medical examinations were carried out in March 1962 on the people of Rongelap Island, 8 years after accidental exposure to fallout irradiation. The medical team consisted of 15 physicians and medical technicians, 9 from the U.S. and 6 from the Department of Public Health of the Trust Territory of the Pacific Islands. Examinations were carried out principally at Rongelap Island, but some Rongelapese who had moved away were examined at Kwajalein and Majuro Islands. A total of 308 people were examined, 80 in the exposed group, 37 children of exposed parents, and 191 unexposed Rongelap people who serve as a comparison population. The Trust Territory ship, *Ran Anim*, was used to transport the medical team and equipment from Kwajalein to Rongelap and return. The team lived in a tent at Rongelap Village during the survey.

The examinations were carried out with complete cooperation of the Rongelap people, and it was believed that the presence of the team living

Table 17

Sr^{90} Analyses of Rongelap Urine Samples

Subject No.	Age	Sex	Other Identification	Sample vol., ml	Sr^{90}, dis/min per total sample	Sr^{90}, pC/l	Ca, g/l	Sr^{90}, pC/g Ca
			pool	12,360	216.2±4.9*	7.9±0.2	0.018	439±11
7	44	M	24-hr	1,280	24.7±1.6	8.7±0.6	0.178	49± 3
14	33	F	24-hr	1,400	51.0±1.9	16.4±0.6	0.060	273±10
15	15	F	24-hr	1,760	28.6±1.6	7.3±0.9	0.014	521±64
16	47	M	24-hr	1,000	23.7±0.8	10.7±0.4	0.209	51± 2
18	29	F	24-hr	1,060	30.8±2.0	13.1±0.8	0.131	100± 6
19	13	M	24-hr	1,540	20.7±1.6	6.0±0.5	0.028	214±18
20	15	M	24-hr	2,060	47.4±2.1	10.4±0.4	0.052	200± 8
22	25	F	24-hr	680	15.8±1.7	10.5±1.1	0.082	128±13
			2nd 24-hr	980	21.9±1.7	10.1±0.8	0.068	148±12
23	12	M	24-hr	1,180	30.3±1.9	11.6±0.7	0.076	153± 9
24	21	F	24-hr	890	46.7±1.2	23.6±0.6	0.122	193± 5
27	34	M	24-hr	1,360	17.4±1.5	5.8±0.5	0.120	48± 4
			2nd 24-hr	2,020	21.4±1.8	4.8±0.4	0.125	38± 3
36	16	M	24-hr	1,250	46.0±1.8	16.6±0.6	0.077	216± 8
39	23	F	24-hr	600	34.8±1.0	26.1±0.8	0.131	199± 6
			2nd 24-hr	710	32.9±1.7	20.9±1.1	0.090	232±12
40	37	M	24-hr	1,960	43.3±1.8	10.0±0.4	0.123	81± 3
			2nd 24-hr	1,180	26.3±1.2	10.0±0.4	0.155	64± 3
41	52	M	24-hr	800	23.9±2.0	13.4±1.1	0.312	43± 4
50	42	M	24-hr	880	91.1±2.9	46.6±1.5	0.268	174± 6
59	42	F	24-hr	1,100	15.6±1.3	6.4±0.5	0.149	43± 3
69	12	F	24-hr	2,040	54.0±1.9	11.9±0.4	0.036	330±11
818	11	M	24-hr	1,660	105.4±2.5	28.6±0.7	0.140	204± 5
822	16	M	24-hr	2,000	18.0±1.5	4.0±0.3	0.104	38± 3
838	29	M	24-hr	1,500	52.4±2.0	15.7±0.6	0.152	103± 4
			2nd 24-hr	1,860	31.2±2.5	7.6±0.6	0.142	54± 4
843	33	F	24-hr	740	46.0±2.5	28.0±1.5	0.390	72± 4
865	29	F	24-hr	640	14.3±0.8	10.1±0.6	0.051	198±12
			2nd 24-hr	450	8.4±1.2	8.4±1.2	0.031	271±39
885	22	M	24-hr	1,730	31.7±1.6	8.2±0.4	0.119	69± 3
893	44	F	24-hr	1,580	20.9±2.0	6.0±0.6	0.067	90± 9
895	32	F	24-hr	260	4.8±0.9	8.3±1.6	0.077	108±21
			2nd 24-hr	1,240	9.3±1.2	3.4±0.4	0.037	92±11
896	22	F	24-hr	1,600	16.6±1.5	7.5±0.7	0.045	167±16

*The error term accompanying each result is the Poisson error of counting.

ashore in Rongelap Village enhanced the rapport with the people, possibly by allaying any fears that they might have had concerning lingering radio activity on the Island.

A review of the medical status of the people during the past year revealed that they had generally been in good health with the usual number of minor ailments, and that no disease epidemics had occurred. Some cases of sickness from eating improperly prepared arrowroot flour and poisonous fish were reported. One death occurred in a 78 yr. old woman in the exposed group who was quite senile; death resulted from a fall. Autopsy and histological studies showed advanced senile changes, but there was no reason to relate radiation exposure to such findings in a woman of this age. Four normal births had occured in the exposed group and four in the comparison group. No miscarriages or stillbirths were reported during the past year.

Table 18

Sr^{90} in Marshallese 24-hr Urine Samples, April 1962

Group	Age, yr	No. of subjects	$-Sr^{90}$, pC/l	Ca, mg/l	Sr^{90}, pC/g Ca
Exposed					
Male	1-15	3	9.33±1.70*	52±14	189±18
	>15	7	9.06±1.11	175±26	53± 5
Female	1-15	2	9.60±2.30	25±11	426±96
	>15	8	15.89±2.50	104±12	165±26
Total	1-15	5	9.44±1.18	41±11	284±66
	>15	15	12.70±1.66	137±16	113±20
Nonexposed					
Male	1-15	1	28.60±0	140± 0	204± 0
	>15	4	8.88±2.46	129±11	66±14
Female	1-15	—	——	——	——
	>15	7	10.24±3.07	100±49	143±28
Total	1-15	1	28.60±0	140± 0	204± 0
	>15	11	9.75±2.07	110±31	115±21
Total					
All	1-15	6	12.63±3.34	42± 9	270±55
All	>15	26	11.45±1.30	126±16	114±14

*Standard error of estimate.

Routine physical examinations of adults revealed that the slight differences in incidence of various diseases in the exposed group compared with the control group could likely be explained by the somewhat greater number of older people in the exposed group.

An ophthalmological survey revealed a slight increase in the incidence of pterygia and pinguecula in the exposed group. As reported previously, this may be related to fallout exposure of the eyes. There were also several cases of corneal pigmentation which may have been related to fallout exposure. There was no evidence in the exposed people of retinal burns which might have resulted from observation of the fireball at the time of the accident in 1954.

A cancer detection survey was done which included, in addition to thorough physical examinations, pelvic examinations in mature women with Papanicolaou staining of smears, rectal examinations in adults, and hematological studies for leukemia detection, which included the determination of alkaline phosphatase levels of leukocytes and basophil counts of 4000 white cells. No cases of malignancy or leukemia were detected.

Pediatric examinations revealed no marked differences in abnormalities between exposed and unexposed children and no diseases that could be related to radiation effects. Growth and development studies, as previously reported, showed that the boys exposed at an early age (<6 yr and particularly 12 to 18 months) were slightly shorter in stature and weighed slightly less than the unexposed children of comparable age. No such differences were noted in the exposed compared with the unexposed girls. Skeletal age determined by wrist roentgenography showed that both the boys and the girls in the exposed group were slightly retarded, the boys showing the greater deficit. The greatest retardation was noted in 4 boys who had been 16 to 17 months old at exposure. Male children (but not female) born of exposed parents (after the fallout) tended to be smaller in stature at all ages than children of unexposed parents.

Table 19

Sr^{90} Analyses of Bone and Crab Samples

	Original net wt., g	Ash		Sr^{90}, dis/min		Ca, % in ash	Sr^{90}, pC/g Ca
		g	%	Per total sample	Per g ash		
Bone							
Rib		1.48		5.4±0.6*	11.1±0.4	36.6	13.7±0.5
Vertebra		2.60		33.8±0.0	13.0±0.3	36.0	16.3±0.4
Crab	878	111	12.7		1462 ±5	50.0	1317 ±4
	902	136	15.1		964 ±4	40.0	1086 ±4
	1165	209	17.9		850 ±4	34.4	1113 ±5
	705	99	14.0		1518 ±6	49.6	1378 ±5

*The error term accompanying each result is the Poisson error of counting.

However, since the latter children were on the average 4 months older, the data did not justify a conclusion that the difference in stature was associated with the exposure of the parents.

Residual beta burns of the skin consisting of varied degrees of atrophy, scarring, pigment aberrations, and hyperkeratosis were noted as in past surveys in about 10 people. During the past several years an increase in the number of pigmented nevus-like lesions has been noted in areas previously involved with beta burns. These appear to be increasing slightly in number. Histological diagnosis of one such lesion that was biopsied indicated that it was a typical pigmented nevus. No case of chronic radiation dermatitis or malignant change in the residual burns was noted.

Genetic studies have not been specifically conducted in view of the small population involved and the generally negative results of the studies on first-generation Japanese children of exposed parents. However, routine examination has revealed little in the way of congenital anomalies in the children of exposed parents. One baby born of an exposed mother in 1960 was diagnosed as having congenital heart disease and died several months after birth. The suggested evidence of increased miscarriages and stillbirths in the exposed woman during the first several years after exposure might be related to irradiation of the germ plasm, although this tendency was not apparent in recent years.

Hematological studies showed mean leukocyte levels lower this year than last year in both exposed and unexposed groups, but the exposed group continued to show lower neutrophil and lymphocyte levels than the unexposed group. Platelet levels were higher this year in both groups than previously, but the exposed group continued to show slightly lower levels. There was an increase in erythrocytes in both exposed and unexposed groups, with little difference noted between the groups. The deficit in leukocytes and platelets in the exposed people was more apparent in the younger and older groups than in the middle group. Bone marrow examinations showed a reduced myeloid-erythroid ratio in 8 of 9 cases with abnormalities of the erythroid and myeloid precursors in some. In view of these findings, it is believed that there is some residual effect of radiation exposure on hematopoiesis in these people.

Peripheral blood cultures or chromosome studies were carried out on samples from a number of people. Successful cultures were obtained in some 40 of 70 bloods; however, difficulties were encountered under field conditions of high temperature and humidity in obtaining satisfactory chromosome smears. Nevertheless small numbers of dicentrics along with abnormal diploid numbers were noted in a few smears. These data suggest that resistant damage to blood cells occurred, and during the next survey further chromosome studies of blood and skin of beta burned areas and possibly of bone marrows are being planned.

Gamma spectrographic analyses were not carried out this year since previous levels of gamma emitting isotopes (Cs^{137} and Zn^{65}) had not shown significant further increase at the time of the 1961 survey. However, urinary excretion of Sr^{90} was determined by radiochemical analysis on 24-hr samples from 18 exposed and 9 unexposed people. As previously noted, there were no differences between the exposed and the unexposed urinary levels of Sr^{90}. The mean level of Sr^{90} was 114 ± 14 pC/g Ca. On the basis of previous calculations, the estimated body burden of Sr^{90} was 12.0 mμC, which is about 6% of the MPC (200 mμC for non-industrial populations). Several bone samples obtained from autopsy on the 78-yr-old exposed woman showed an average of 15 pC/g Ca, representing a body burden of 11.4 mμC or about 5.7% of the MPC, which compares favorably with the estimate based on urinary excretion. These levels represent about a sixfold increase in Sr^{90} over the 1958 levels. Analysis of 4 coconut crabs from the Island showed high levels with little reduction during the past few years, necessitating a continuation of the ban on eating these crabs.

Although 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 persisting in the exposed people which 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.

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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 ($\times 10^{-4}$)				Hematocrits (%)			RBC ($\times 10^{-6}$)		
Postexposure day	<5	>5	<5	>5	<5	>5	Male <10	Male >10	Female all ages	Total group	Male <15	Male >15	Female all ages	Male <15	Male >15	Female all ages
3	9.0	8.2	6.4	4.7	1.8	2.2	—	—	—	—	—	—	—	—	—	—
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	—	—	—	—	—	—
12	5.9	6.3	3.5	3.9	2.1	1.7	—	—	—	—	—	—	—	—	—	—
15	5.9	6.5	3.2	4.1	2.4	1.9	27.1	21.3	21.7	22.5	—	—	—	—	—	—
18	6.7	7.2	3.4	4.7	2.4	2.1	21.8	19.1	21.8	21.0	—	—	—	—	—	—
22	7.0	7.4	4.3	5.0	2.6	2.1	16.8	14.6	15.2	15.3	37.5	43.9	39.0	—	—	—
26	5.7	6.1	3.0	3.9	2.3	1.8	13.2	12.9	10.9	11.9	36.3	41.6	37.5	—	—	—
30	7.6	7.8	4.0	5.3	3.2	2.1	14.1	12.3	11.8	12.3	37.9	42.2	37.1	—	—	—
33	6.5	6.2	3.1	3.8	3.2	2.0	17.9	16.6	15.1	16.0	37.4	42.2	36.8	—	—	—
39	5.7	5.5	3.0	3.3	2.6	2.0	25.5	22.0	22.4	22.8	37.8	42.4	37.4	—	—	—
43	5.2	5.2	2.0	2.6	2.9	2.3	26.8	20.9	23.2	23.2	37.3	41.8	37.6	—	—	—
47	5.9	5.8	2.6	3.3	3.1	2.4	24.6	20.6	23.9	23.1	39.0	43.4	38.3	—	—	—
51	6.7	5.6	2.6	3.5	3.4	2.1	22.1	17.5	21.2	20.3	—	—	—	—	—	—
56	7.0	6.0	3.5	3.5	3.7	2.4	—	—	—	—	—	—	—	—	—	—
63	7.7	6.0	3.9	3.6	3.7	2.3	23.1	18.2	20.2	20.1	—	—	—	—	—	—
70	7.6	6.5	3.8	4.0	3.3	2.2	—	—	—	—	—	—	—	—	—	—
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	38.0	41.7	38.2	—	—	—
1-yr survey	10.1	8.1	4.7	4.8	4.6	2.8	26.6	19.5	27.6	24.9	37.5	41.1	36.9	—	—	—
2-yr survey	11.8	8.6	5.9	4.8	4.7	3.1	30.0	21.4	25.5	24.7	38.7	41.2	38.1	—	—	—
3-yr survey	8.6	6.9	4.1	3.7	3.7	2.7	32.0	22.1	28.1	—	35.6	38.7	35.4	—	—	—
4-yr survey	8.9	7.5	3.3	3.4	4.6	3.6	32.5	27.1	30.8	—	35.6	41.0	35.8	—	—	—
5-yr survey	13.5	9.5	6.9	4.8	6.0	4.0	32.3	24.4	27.6	—	—	—	—	4.45	4.71	4.21
6-yr survey	—	6.5	—	3.5	—	2.7	—	—	—	—	—	—	—	—	—	—
7-yr survey	—	7.4	—	3.9	—	2.9	—	24.6*	27.3	—	37.6	41.7	37.0	4.54	4.45	4.11
8-yr survey	—	6.9	—	3.6	—	2.6	—	32.8**	32.1	—	38.5	43.0	39.3	4.68	4.67	4.44
Majuro controls	13.2	9.7	4.8	4.8	7.4	4.1	41.2	25.8	36.5	33.4	39.6	46.0	39.9	—	—	—
Rita cont. 6 mo	10.7	7.6	5.4	5.2	4.7	3.7	35.0	27.3	30.9	30.4	—	—	—	—	—	—
Rita cont. 1 yr	—	—	—	—	—	—	37.5	24.5	29.4	27.6	—	—	—	—	—	—
Rita cont. 2 yr	14.0	8.9	7.0	4.4	5.6	3.6	35.5	24.2	31.2	29.5	38.9	42.1	39.8	—	—	—
Rong. cont. 3 yr	9.8	6.9	4.0	3.4	4.7	2.9	32.6	26.9	30.0	—	35.6	41.0	35.9	—	—	—
Rong. cont. 4 yr	11.2	8.0	4.0	3.6	6.2	3.7	38.8	30.7	34.0	—	35.5	42.8	35.1	—	—	—
Rong. cont. 5 yr	13.7	10.1	6.2	5.2	6.2	4.1	35.8	28.0	33.6	—	—	—	—	4.60	4.80	4.40
Rong. cont. 7 yr	—	7.8	—	4.2	—	3.1	—	28.5*	31.4	—	37.2	44.4	37.0	4.52	4.68	4.12
Rong. cont. 8 yr	—	7.7	—	4.2	—	2.9	—	34.8**	34.5	—	38.3	44.1	39.0	4.60	4.90	4.47

* Includes all males >7.
** Includes all males >8.

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 ($\times 10^{-4}$)				Hematocrits (%)			RBC ($\times 10^{-6}$)		
Postexposure day	<5	>5	<5	>5	<5	>5	Male <10	Male >10	Female all ages	Total group	Male <15	Male >15	Female all ages	Male <15	Male >15	Female all ages
3	6.0	7.0	3.0	5.0	2.8	2.2	—	—	—	—	—	—	—	—	—	—
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	—	—	—	—	—	—
12	6.3	7.6	1.8	4.7	3.1	2.2	—	—	—	—	—	—	—	—	—	—
15	7.1	7.0	2.3	4.5	4.2	2.2	29.0	20.2	24.6	23.9	—	—	—	—	—	—
18	6.8	7.8	2.9	5.0	3.5	2.4	27.5	21.7	24.9	24.3	—	—	—	—	—	—
22	8.9	8.7	5.3	5.4	2.7	2.9	23.5	17.0	22.9	21.3	37.5	43.7	39.2	—	—	—
26	8.4	7.0	4.8	4.4	3.2	2.2	20.0	13.8	17.4	16.7	36.5	43.2	36.8	—	—	—
30	9.6	8.6	5.3	6.2	3.7	2.0	19.5	12.8	18.2	16.8	36.0	44.6	36.7	—	—	—
33	7.7	7.8	3.3	5.2	3.5	2.2	24.0	15.8	22.7	17.6	35.5	43.8	37.3	—	—	—
39	7.5	6.2	2.9	4.2	4.7	1.9	26.5	20.8	27.0	25.2	35.0	45.6	37.4	—	—	—
43	6.9	6.5	2.7	3.6	3.9	2.7	28.0	19.6	25.3	24.0	36.0	45.2	36.8	—	—	—
47	7.3	6.7	3.5	3.8	3.4	2.7	27.0	20.0	26.1	24.5	—	46.5	40.2	—	—	—
51	8.4	6.3	3.8	3.6	4.0	2.2	32.0	18.2	25.0	23.9	—	—	—	—	—	—
54	4.6	6.3	2.8	3.5	3.2	2.5	37.0	19.8	23.8	24.2	—	—	—	—	—	—
6-mo survey	7.7	6.5	4.8	3.9	2.7	2.2	25.2	19.2	23.9	22.7	37.5	40.1	37.3	—	—	—
1-yr survey	11.1	7.8	4.2	4.7	6.5	5.6	38.7	21.4	28.3	27.5	33.0	44.6	36.2	—	—	—
2-yr survey	11.0	9.1	4.9	5.1	4.8	3.2	51.2	17.4	26.4	26.7	35.7	44.4	37.5	—	—	—
3-yr survey	12.1	7.0	5.5	3.9	5.6	2.6	40.8	22.4	31.2	—	37.5	40.6	35.6	—	—	—
4-yr survey	11.5	7.5	2.8	3.7	7.0	3.3	33.2	24.7	33.6	—	36.1	43.1	35.7	—	—	—
5-yr survey	—	9.7	—	5.1	—	3.7	40.9	26.3	26.8	—	—	—	—	4.46	5.15	4.31
6-yr survey	—	7.3	—	3.6	—	3.0	—	—	—	—	—	—	—	—	—	—
7-yr survey	—	7.7	—	4.1	—	3.1	—	25.6*	28.1	—	36.0	44.2	37.0	4.56	5.11	4.19
8-yr survey	—	6.5	—	3.4	—	2.6	—	33.4**	32.7	—	37.0	42.5	37.8	4.51	5.12	4.35
Majuro controls	13.2	9.7	4.8	4.8	7.4	4.1	41.2	25.8	36.5	33.4	39.6	46.0	39.9	—	—	—
Rita cont. 2 yr	14.1	8.9	7.0	4.4	5.6	3.6	35.5	24.2	31.2	29.5	38.9	42.1	39.8	—	—	—
Rong. cont. 3 yr	9.8	6.9	4.0	3.4	4.7	2.9	32.6	26.9	30.0	—	35.6	41.0	35.9	—	—	—
Rong. cont. 4 yr	11.2	8.0	4.0	3.6	6.2	3.7	38.8	30.7	34.0	—	35.5	42.8	35.1	—	—	—
Rong. cont. 5 yr	13.7	10.1	6.2	5.2	6.2	4.1	35.8	28.0	33.6	—	—	—	—	4.60	4.80	4.40
Rong. cont. 7 yr	—	7.8	—	4.2	—	3.1	—	28.5*	31.4	—	37.2	44.4	37.0	4.52	4.68	4.12
Rong. cont. 8 yr	—	7.7	—	4.2	—	2.9	—	34.8**	34.5	—	38.3	44.1	39.0	4.60	4.90	4.47

* Includes all males >7.
** Includes all males >8.

APPENDIX 3

Individual Hematological Findings, 1962

Subject No.	Plate. ($\times 10^{-3}$)	WBC ($\times 10^{-3}$)	Neut. ($\times 10^{-3}$)	Lymph. ($\times 10^{-3}$)	Mono. ($\times 10^{-1}$)	Eosin. ($\times 10^{-3}$)	Baso. ($\times 10^{-2}$)	Hct., %	RBC ($\times 10^{-4}$)	Hgb., g	Serum protein, g
Exposed Males, Age 8–15											
2	310	5.39	2.53	2.37	0.11	0.32	0.50	38	432	13.2	8.0
3	405	9.01	4.87	2.88	0.18	0.99	0.90	34	388	11.5	8.4
5	455	8.83	5.12	3.27	0.18	0.26	0	37	403	11.8	7.6
19	595	9.08	6.08	2.90	0	0.90	0	41	563	13.2	8.6
20	250	9.23	5.08	3.13	0.18	0.83	0	46	561	16.0	8.2
23	410	10.10						36	448	11.8	7.8
32	440	7.96	2.22	3.98	0.16	1.43	0.16	39	470	13.2	7.2
54	175	5.24	2.31	1.99	0.21	0.73	0	40	497	12.8	8.2
83*	355	5.56	3.06	2.11	0.11	0.28	0	35	440	12.4	
85*	535	10.89	4.57	3.27	0.33	2.61	0.11	39	480	12.4	
Mean	393 (±120)**	8.12 (±1.93)	3.98 (±1.37)	2.88 (±0.60)	0.16	0.84	0.19	38.5 (±3.3)	468 (±56)	12.8 (±1.4)	8.0 (±0.4)
Ailingnae Exposed Males, Age 8–15											
6	430	7.36	4.71	1.91	0	0.74	0	36	450	12.4	7.6
44	430	5.75	2.76	2.30	0.23	0.46	0	39	498	12.4	7.8
84*		7.83	3.45	3.29	0	1.10	0	36	406	12.1	
Mean	430	6.98	3.64	2.50	0.08	0.77	0	37.0	451	12.3	7.7
Exposed Females, Age 8–15											
15	475	7.52	3.83	3.53	0.15	0	0	39		14.0	8.4
17	290	8.14	6.02	1.63	0.16	0.33	0	42	493	14.0	8.0
21	405	6.84	2.80	2.53	0.27	1.09	0.14	43	532	14.0	7.8
33	400	6.62	2.58	3.24	0.33	0.46	0	39		14.8	8.2
42	305	5.90	3.95	1.30	0.18	0.47	0	37	449	13.2	7.8
65	275	7.28	2.98	3.79	0.07	0.44	0	37		12.1	7.6
69	230	5.30	1.70	3.29	0	0.27	0.05	40	472	14.0	8.0
72	270	5.70	2.57	2.45	0.34	0.34	0	40		14.0	8.0
86*	270	8.43	5.56	2.44	0.08	0.34	0	37	444	12.1	7.2
Mean	324 (±77)	6.86 (±1.03)	3.55 (±1.37)	2.69 (±0.88)	0.18	0.42	0.02	39.3 (±2.0)	478 (±32)	13.6 (±1.0)	7.9 (±0.3)
Ailingnae Exposed Females, Age 8–15											
8	430	7.49	4.72	2.32	0.07	0.37	0	38	422	12.8	7.4
48	200	7.00	3.40	2.87	0.35	0.28	0.07	42	488	14.4	8.0
Mean	315	7.25	4.06	2.59	0.21	0.32	0.03	40	455	13.6	7.7
Exposed Males, Age >15–40											
9	160	7.25						44		14.5	7.2
10	160	5.95	3.87	1.55	0.36	0.18	0	51		16.0	8.0
26	305	7.62	3.28	4.27	0	0.08	0	46	523	15.2	8.0
27	420	6.15	2.34	3.14	0.18	0.43	0.06	41		18.0	7.4
35	280	9.87	7.30	2.07	0.39	0	0.10	49	520	16.4	8.4
36	305	5.00	2.20	2.60	0.10	0.10	0	41	496	13.6	8.0
37	325	7.74	2.94	3.17	0.15	1.47	0	44	448	14.4	7.2
40	445	6.65	3.92	2.53	0.13	0.07	0	43	459	13.6	6.8
47	520	6.33	3.54	2.41	0.13	0.25	0	40	454	14.4	7.4

* Exposed *in utero*.
** Standard deviation.

Individual Hematological Findings, 1962

Subject No.	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. ($\times 10^{-3}$)	Hct., %	RBC ($\times 10^{-6}$)	Hgb., g	Serum protein, g
Exposed Males, Age >15–40 (continued)											
73	165	5.46	3.71	1.37	0.16	0.22	0	49	509	15.6	7.0
76	310	5.94	2.44	3.15	6	0.30	0	43	466	14.8	7.6
77	450	5.80	2.72	2.32	0	0.70	0.06	46	519	14.8	7.8
Mean	320 (±120)	6.65 (±1.26)	3.48 (±1.35)	2.60 (±0.78)	0.15	0.35	0.02	44.8 (±3.3)	488 (±75)	15.1 (±1.2)	7.6 (±0.5)
Exposed Females, Age >15–40											
12	275	5.80	3.30	2.15	0.29	0.06	0	42	405	12.8	7.8
14	420							36		12.4	7.8
18	405	5.80	4.18	1.22	0.06	0.29	0.06	37	412	12.1	6.6
22	530	5.25	2.42	1.73	0.05	1.05	0	37	472	12.8	8.0
24	350	5.44	3.05	1.74	0.44	0.22	0	36	429	12.4	7.8
39	435	4.87	2.04	2.39	0.05	0.34	0.05	39	446	12.8	8.8
49	320	6.41	3.08	2.88	0.06	0.38	0	40	459	13.6	8.0
61	290	7.55	4.30	2.72	0.15	0.38	0	42	469	14.4	8.2
64	340	5.84	3.39	2.10	0.29	0.06	0	37	416	12.4	7.2
66	265	6.39	3.45	2.68	0.12	0.06	0.06	40	446	13.6	8.0
71	370	7.68	4.30	2.38	0.08	0.92	0	42		15.6	8.2
74	295	13.30	7.32	2.79	0.13	3.06	0	50	524	16.0	9.0
75	250	7.93	4.84	2.38	0	0.71	0	46	462	15.2	8.0
Mean	350 (±78)	6.86 (±2.15)	3.81 (±1.81)	2.26 (±0.48)	0.14	0.63	0.01	40.3 (±4.0)	449 (±32)	13.5 (±1.2)	8.0 (±0.6)
Ailingnae Exposed Females, Age >15–40											
45	225	4.16	1.25	2.33	0.17	0.33	0.08	35	377	11.8	7.6
53	550	7.97	4.14	3.59	0.08	0.08	0.08	38	450	13.2	7.6
70	300	5.50	3.69	1.49	0.11	0.22	0	29		7.6	8.4
81	325	5.16	2.94	1.86	0	0.36	0	38	476	12.8	8.6
Mean	350 (±121)	5.70 (±1.39)	3.01 (±1.27)	2.32 (±0.91)	0.09	0.25	0.04	35.0 (±3.7)	434	11.4 (±2.4)	8.1 (±0.3)
Exposed Males, Age >40											
4	195	6.42	2.12	3.59	0.32	0.38	0	48	563	16.0	7.6
7	265	6.49	2.86	2.34	0.26	0.97	0.06	43	466	14.4	8.2
11	340	4.47	2.86	1.20	0.04	0.26	0.09	40	428	13.2	7.4
46	225	3.96	2.03	1.29	0.11	0.08	0	32	314	11.2	6.4
55	260	4.85	2.04	2.67	0.05	0.10	0	30	323	10.3	6.2
68	205	4.16	1.83	1.99	0	0.25	0.08	41	461	13.6	6.6
79	175	8.65	5.10	2.85	0.17	0.43	0.09	49	510	16.4	8.2
80	275	9.10	6.19	2.55	0.18	0.18	0	44	486	14.4	8.2
82	450	6.85	5.14	1.23	0.14	0.28	0.07	38		14.0	7.0
Mean	266 (±80)	6.10 (±1.50)	3.35 (±1.57)	2.19 (±0.78)	0.14	0.33	0.04	40.6 (±6.2)	444 (±81)	13.7 (±1.9)	7.3 (±0.7)
Ailingnae Exposed Males, Age >40											
16	335	5.19	2.39	2.60	0.10	0.10	0	39	580	13.6	7.2
29	235	6.86	2.74	3.43	0.07	0.54	0.07	41	441	14.0	8.0
41	320	4.41	2.65	1.54	0.04	0.18	0	44	504	15.2	7.6
50	255	7.58	3.79	3.34	0.30	0.15	0	46	525	15.6	6.8
Mean	286 (±43)	6.01 (±1.27)	2.89 (±0.62)	2.73 (±0.87)	0.13	0.24	0.02	42.5 (±2.7)	512 (±49)	14.6 (±0.8)	7.4 (±0.4)

Individual Hematological Findings, 1962

Subject No.	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. ($\times 10^{-3}$)	Hct., %	RBC ($\times 10^{-4}$)	Hgb., g	Serum protein, g
Exposed Females, Age >40											
13	525	6.55	3.27	2.69	0.07	0.46	0.07	40	405	12.8	7.4
30	205	7.10	4.62	1.99	0.21	0.28	0	29		10.0	10.0
34	190	5.44	2.99	2.12	0	0.33	0	37	379	12.4	7.6
52	300	9.73	6.03	2.24	0.49	0.78	0.19	42	444	13.6	8.2
57	225	5.12	1.59	3.38	0	0.10	0.05	40	425	11.8	7.4
58	190	5.28	3.17	1.90	0.10	0.10	0	36		12.4	7.0
60	230	8.21	4.27	3.28	0.16	0.41	0.08	36	387	12.1	7.2
63	305	5.08	1.93	2.85	0.10	0.20	0	41	440	13.6	7.0
78	315	5.70	3.65	1.60	0.23	0.17	0.06	40	410	13.2	7.2
Mean	276 (±99)	6.37 (±1.89)	3.50 (±1.28)	2.45 (±0.60)	0.18	0.31	0.05	37.9 (±3.7)	413 (±22)	12.4 (±1.0)	7.7 (±0.9)
Ailingnae Exposed Females, Age >40											
1	265	6.75	3.78	2.90	0.07	0	0	42	472	14.4	8.4
43	200	7.39	5.17	1.48	0.30	0.37	0.08	40	436	13.2	8.0
59	515	8.78	4.74	2.90	0.26	0.88	0	37	388	12.4	7.6
28	255	6.10	2.38	3.48	0.12	0.12	0	39	409	14.0	8.0
Mean	309 (±122)	7.26 (±0.99)	4.02 (±1.24)	2.69 (±0.85)	0.19	0.34	0.02	39.5 (±1.8)	426 (±32)	13.5 (±0.8)	8.0 (±0.3)
Male Children of Exposed Parents, Age <8											
88	445	6.86	3.70	2.95	0.07	0.14	0	40	427	11.8	
89	365	8.74	4.54	2.97	0.09	1.14	0	43	455	14.4	
91	390	6.46	3.41	2.62	0	0.52	0	39	432	12.1	
93	465	9.80	4.61	4.51	0	0.69	0	38	388	11.8	
96	510	8.65	4.24	3.11	0.26	1.04	0	40	451	12.4	
97	685	7.24	3.04	3.76	0.14	0.29	0	36	433	12.1	
98	505	10.83	5.31	4.66	0.22	0.65	0	43	456	12.1	
102	430	8.70	4.70	3.57	0.17	0.26	0	37	484	11.8	
104	420	9.37	4.59	4.31	0.09	0.37	0	38	455	12.8	
110	320	8.47	3.81	3.98	0.08	0.60	0	42	532	12.1	
111	720	9.25	3.52	5.27	0.37	0.09	0	36		11.2	
113	615	6.69	2.94	3.14	0.20	0.40	0	39	512	12.4	
115	490	10.50	3.47	6.41	0.42	0.21	0	38	450	12.1	
118	585	9.78	4.30	4.50	0.10	0.88	0	36	453	11.2	
126	720	9.62	6.35	3.08	0	0.10	0.10	31		10.1	
Mean	511 (±123)	8.74 (±1.34)	4.17 (±0.87)	3.92 (±1.00)	0.15	0.49	0.007	38.4 (±3.0)	456 (±35)	12.0 (±0.9)	
Female Children of Exposed Parents, Age <8											
87	255	9.22	3.50	4.70	0.18	0.83	0	39	476	13.2	7.6
92	260	10.94	5.91	3.39	0.33	0.88	0	39	478	11.5	
94	380	8.94	2.68	5.19	0.27	0.80	0	40	470	13.6	
95	520	8.76	3.33	3.94	0.26	1.22	0	38	505	12.4	
100	525	9.10	4.37	3.73	0.55	0.46	0	36	400	14.8	
101	195	15.80	7.42	8.33	0.72	1.63	0	42	480	12.8	
103	540	11.40	5.70	5.02	0.45	0.11	0.11	38	450	11.5	
105	455	10.57	6.24	3.70	0.21	0.42	0	38	471	11.5	
106	595	10.30	5.46	3.91	0.41	0.51	0	39	472	12.1	
107	645	12.70	6.22	4.57	0.89	1.01	0	32	432	9.7	
108	460	12.70	5.59	5.59	0	1.54	0	34	461	11.2	

Individual Hematological Findings, 1962

Subject No.	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. ($\times 10^{-3}$)	Hct., %	RBC ($\times 10^{-6}$)	Hgb., g	Serum protein, g
Female Children of Exposed Parents, Age <8 (continued)											
112	765	8.03	3.21	4.18	0.08	0.40	0	36	464	11.5	
117	680	9.85	3.55	5.52	0.59	0.10	0.10	35		10.9	
119	310	16.70	5.68	6.85	0	4.01	0.17	37	496	10.6	
120	385	13.51	6.76	3.35	0.14	0.27	0	39	460	11.2	
122	450	10.30	3.09	6.80	0	0.41	0	36	422	12.1	
123	405	7.54	3.02	3.85	0	0.68	0	32	346	10.6	
124	575	9.87	2.86	6.22	0.20	0.39	0.20	32		10.0	
125	585	12.32	5.17	6.41	0.49	0.25	0	35	429	11.5	
Mean	473 (±149)	10.98 (±2.41)	4.72 (±1.47)	5.01 (±1.36)	0.30	0.84	0.03	36.7 (±2.8)	454 (±37)	11.7 (±1.2)	
Unexposed Males, Age 8–15											
813	335	7.18	3.02	3.73	0	0.43	0	37	429	12.8	8.0
814	315	8.57	5.14	3.08	0.17	0.17	0	36	450	12.8	7.6
815	295	7.26	3.19	3.85	0.15	0.07	0	42	474	14.0	8.2
817	395	13.40	8.44	3.62	0.40	1.07	0	36	460	13.2	8.0
818	460	9.07	4.81	2.99	0.27	1.00	0	39	460	13.2	
819	415	5.29	2.27	2.49	0.11	0.42	0	35	427	11.2	7.2
820	470	7.75	5.19	2.24	0.16	0.16	0	41	491	13.2	7.4
863	470	7.60	3.34	3.88	0.23	0.15	0	43	494	13.6	
912	470	9.63	4.72	3.47	0.48	0.96	0	33	450	12.1	7.6
913	335	5.55	3.89	1.17	0	0.44	0.06	38	508	12.8	
919	450	6.11	3.24	2.26	0.12	0.49	0	37	438	12.1	7.8
921	650	9.11	5.10	3.28	0	0.64	0.09	41	465	12.4	
940	480	7.16	3.08	3.72	0.14	0.21	0	40	508	14.0	7.8
981	388	9.92	5.06	4.27	0.20	0.40	0	36	388	12.1	
1033	390	8.70	3.48	4.26	0.09	0.87	0	40		14.0	8.0
1036	325	7.60	4.94	2.13	0.15	0.38	0	39		13.2	7.8
Mean	415 (±86)	8.12 (±1.90)	4.31 (±2.06)	3.15 (±0.85)	0.17	0.49	0.01	38.3 (±2.8)	460 (±35)	12.9 (±0.7)	7.8 (±0.3)
Unexposed Females, Age 8–15											
805	470	8.34	4.42	3.42	0.17	0.33	0	38	477	12.1	
812	369	9.99	4.80	4.40	0.20	0.50	0	37	453	12.1	
816	360	8.98	3.59	4.04	0.09	1.26	0	39	471	12.1	7.2
821	395	8.08	5.01	2.26	0.16	0.65	0	40	461	13.2	8.0
879	435	14.21	10.37	2.56	0.71	0.57	0	38	406	12.8	
891	485	6.42	2.38	3.21	0.13	0.64	0.06	40	450	14.0	7.4
911	505	10.74	5.48	3.47	0.48	0.96	0	41	453	12.1	
926	220	13.10	7.73	4.06	0.39	0.79	0.13	41	472	12.8	7.8
937	330	12.20	6.95	4.51	0.24	0.49	0	39	440	12.8	
946	235	8.25	3.63	4.04	0.08	0.41	0.08	42	472	14.8	7.8
955	370	6.90	3.04	3.38	0.14	0.35	0	40	459	13.2	8.0
959	440	8.32	5.24	2.58	0	0.50	0	38	440	12.8	7.6
960	310	13.20	6.86	5.54	0.53	0.26	0	38	454	12.1	8.0
962	325	6.14	3.07	2.51	0.31	0.25	0	37	476	12.1	
988	485	7.61	3.81	3.04	0.53	0.23	0	40	412	13.2	
990	310	8.42	4.38	2.53	0.34	1.01	0.17	37	445	12.4	
993	420	8.00	4.24	2.40	0.16	1.12	0.08	41	515	14.0	7.6

Individual Hematological Findings, 1962

Subject No.	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 8–15 (continued)											
996	435	6.56	1.71	4.40	0.07	0.39	0	35	424	11.5	7.4
998	430	7.01	3.58	2.59	0.21	0.63	0	39	474	13.6	8.2
1035	560	9.30	3.81	4.09	0.19	1.12	0.09	42		14.8	8.0
Mean	390 (±80)	9.08 (±2.41)	4.71 (±1.97)	3.45 (±0.88)	0.26	0.62	0.03	39 (±1.6)	455 (±24)	12.9 (±0.8)	7.8 (±0.3)
Unexposed Males, Age >15–40											
822	475	8.03	4.82	2.50	0.40	0.30	0	43	486	13.6	7.8
823	260	5.63	2.36	2.65	0.28	0.34	0	41	467	14.4	
824	505	6.33	2.34	3.54	0.06	0.38	0	43	454	14.8	8.4
828	220	7.25	4.06	2.68	0.22	0.29	0	47	538	15.6	8.0
830	350	6.24	3.24	2.68	0.12	0.19	0	47	452	15.6	7.0
833	280	4.77	1.62	2.77	0.10	0.24	0.05	45	537	14.8	7.2
834	255	8.76	3.33	4.73	0.17	0.53	0	46	508	15.2	7.2
836	320	6.60	2.38	3.83	0.26	0.13	0	46	524	14.8	7.6
838	265	5.72	2.12	2.75	0.23	0.57	0.06	49		16.0	7.2
840	305	7.15	4.43	2.15	0.29	0.29	0	46	577	14.8	7.4
842	325	8.79	4.75	2.63	0.26	0.26	0.09	49	510	16.4	7.6
864	380	7.81	2.97	3.83	0.39	0.54	0.08	48	521	15.6	7.4
868	350	4.90	2.45	2.01	0.05	0.39	0	44	482	14.0	6.8
869	440	10.60	6.36	3.71	0.11	0.41	0	45	496	14.8	8.0
881	275	6.34	2.41	3.30	0.25	0.38	0	43	476	14.4	7.8
882	155	7.41	4.08	2.96	0.15	0.22	0	40	488	14.0	7.8
885	365	7.75	3.02	4.11	0.16	0.39	0.08	45	527	16.0	7.8
887	325	6.10	2.75	2.99	0	0.37	0	41	465	14.0	7.6
892	245	7.03	3.94	2.81	0.14	0.07	0.07	45	515	14.5	7.8
939	440	8.53	4.78	2.99	0.34	0.43	0	42	507	13.6	7.4
943	355	7.78	3.73	3.50	0.16	0.31	0.08	46	519	16.0	8.0
944	230	6.72	4.03	2.08	0.27	0.34	0	48	567	16.4	8.6
958	450	10.18	7.33	2.24	0.20	0.41	0	41	448	12.8	7.2
963	280	6.10	2.56	2.99	0.06	0.49	0	45	505	14.4	6.8
966	360	6.17	3.27	2.28	0	0.56	0.06	46		16.0	7.2
967	395	5.64	2.88	2.37	0	0.34	0.06	49	559	16.0	7.6
971	365	7.23	3.76	3.18	0.07	0.22	0	46	592	14.8	8.6
975	155	4.78	3.30	1.29	0.05	0.10	0.05	43	460	14.8	6.6
1005	365	6.30	2.52	3.15	0.38	0.25	0	51	537	16.9	7.6
Mean	327 (±87)	6.99 (±1.43)	3.49 (±1.08)	2.92 (±0.70)	0.18	0.34	0.02	45.2 (±2.8)	508 (±39)	15.0 (±0.9)	7.6 (±0.5)
Unexposed Females, Age >15–40											
825	315	9.05	4.98	3.26	0.19	0.63	0	38	445	12.1	8.0
826	520	7.17	4.73	1.94	0.14	0.36	0	35	421	11.2	7.6
829	380	5.25	2.10	2.26	0.37	0.53	0	35	393	11.8	7.6
832	380	6.55	4.19	1.96	0.07	0.33	0	37	444	12.1	7.6
835	270	10.37	8.09	1.76	0.10	0.41	0	38	407	12.8	7.0
841	450	8.83	5.12	2.12	0.18	1.41	0	39	462	13.2	8.0
843	405	6.68	3.21	2.87	0.20	0.40	0	37	401	12.1	7.8
846	285	6.84	4.92	1.30	0.14	0.48	0	43	465	14.4	8.4
865	345	4.87	2.68	1.80	0	0.39	0	34	434	11.2	7.4
867	415	8.02						38	471	12.1	7.6

Individual Hematological Findings, 1962

Subject No.	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. ($\times 10^{-3}$)	Hct., %	RBC ($\times 10^{-6}$)	Hgb., g	Serum protein, g
Unexposed Females, Age >15–40 (continued)											
888	380	5.63	2.25	2.65	0.17	0.51	0.06	40	465	13.6	8.4
889	490	4.51	2.02	2.03	0.90	0.36	0	41	470	14.4	8.2
895	510	8.58	5.06	2.57	0	0.94	0	42	484	14.8	8.0
896	450	11.08	5.10	4.54	0.55	0.89	0	38		12.1	7.6
914	340	6.51	2.99	3.26	0.07	0.20	0	36	440	12.8	7.4
916	615	7.35	4.34	2.06	0.29	0.66	0	35	399	12.4	8.0
922	355	5.55	2.89	2.39	0.06	0.22	0	38	460	13.6	8.0
932	435	6.90	2.48	4.07	0.07	0.28	0	37	434	12.1	8.2
934	285	8.44	3.88	3.80	0.42	0.34	0	41	483	13.6	8.2
938	210	7.89	5.68	1.81	0.32	0.08	0	40	486	12.8	8.2
945	340	11.36	7.72	2.95	0.45	0.23	0	45	510	15.6	7.8
950	435	13.90	9.59	3.75	0.28	0.28	0	39	436	13.2	8.0
951	185	8.64	3.28	4.92	0.26	0.17	0	41	520	14.0	8.0
965	355	8.63	6.39	1.64	0.09	0.52	0	37	425	12.1	7.6
977	365	7.13	3.21	3.28	0.07	0.43	0.14	41	464	13.2	8.2
1001	140	6.99	5.04	1.40	0.28	0.28	0	39	444	12.4	7.4
1043	240	5.08	2.59	2.18	0.10	0.20	0	40	478	14.0	7.6
Mean	340 (±140)	7.80 (±2.11)	4.41 (±1.90)	2.64 (±0.95)	0.22	0.44	0.01	38.8 (±2.8)	451 (±33)	12.9 (±1.0)	7.8 (±0.4)
Unexposed Males, Age >40											
849	465	7.07	5.09	1.48	0.14	0.35	0	47	456	16.4	8.0
853	340	7.44	3.27	3.12	0.07	0.97	0	43	476	14.8	8.0
855	340	5.97	2.63	2.93	0.06	0.36	0	43	439	14.4	7.6
856	410	11.20	5.26	5.04	0.22	0.67	0	38	434	12.4	7.4
862	335	6.20	3.47	2.29	0.19	0.25	0	40	419	12.4	7.2
875	315	7.32	5.27	1.61	0.29	0.14	0	49	529	16.0	7.4
878	125	4.91	2.16	2.41	0.25	0.10	0	39	469	13.2	7.4
883	455	6.02	2.47	2.71	0.06	0.78	0	43	441	14.0	7.8
886	425	8.92	5.44	2.94	0.09	0.45	0	42	474	13.6	8.8
897	210	8.08	4.52	2.83	0.32	0.32	0.08	43	460	14.0	8.6
915	330	5.15	2.27	2.47	0	0.36	0.05	42	465	14.0	7.8
917	245	5.63	2.48	3.04	0	0.11	0	47	577	16.3	8.8
935	350	5.78	4.45	0.87	0.17	0.29	0	42	446	13.6	7.4
947	160	14.44	11.48	2.17	0.14	0.29	0	41	446	14.0	7.6
948	320	6.33	3.54	2.15	0.06	0.57	0	43	478	14.0	7.2
961	395	5.49	2.75	1.87	0.22	0.55	0.11	43	461	14.4	7.4
964	355	5.80	3.13	2.32	0.12	0.17	0.06	37	394	12.8	7.2
969	470	8.72	6.10	2.18	0.09	0.35	0	44	470	14.4	7.8
1007	265	6.76	3.99	2.63	0	0.14	0	39	455	12.8	7.6
1041	215	5.54	2.44	2.66	0.11	0.28	0.06	44	542	15.6	8.8
Mean	326 (±95)	7.14 (±2.28)	4.13 (±2.13)	2.49 (±0.79)	0.13	0.38	0.02	42.4 (±3.0)	467 (±38)	14.1 (±1.1)	7.8 (±0.5)
Unexposed Females, Age >40											
844	235	5.52	3.37	1.71	0.22	0.22	0	41	449	12.8	8.0
851	295	12.70	7.11	5.21	0.12	0.25	0	36	394	12.4	7.4
852	465	10.10	6.66	2.93	0.30	0.20	0	37		11.8	7.6
858	235	6.91	5.39	1.24	0.14	0.14	0	39	421	12.1	7.6
859	295	6.42	3.21	3.02	0.13	0.06	0	39	400	13.2	7.4

Individual Hematological Findings, 1962

Subject No.	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 ($\times 10^{-6}$)	Hgb., g	Serum protein, g
				Unexposed Females, Age >40 (continued)							
893	235	10.50	7.14	2.94	0.21	0.21	0	41	453	13.6	7.6
894	320	10.50	5.25	4.30	0.32	0.63	0	41		12.8	7.8
898	310	7.25	3.63	2.76	0.15	0.73	0	37	414	12.4	8.6
908	205	6.19	2.97	2.85	0	0.37	0	44	427	14.4	7.6
928	370	5.40	2.38	2.65	0.16	0.32	0	36	395	12.1	8.6
929	360	5.53	2.82	2.21	0.05	0.39	0.05	38	413	14.0	7.8
936	150	8.08	4.28	3.31	0.24	0.24	0	38	399	12.8	7.2
942	200	6.98	4.40	2.30	0	0.28	0	40	445	14.0	7.6
956	410	6.99	4.61	2.24	0.14	0	0	37	424	12.8	7.2
957	370	8.70	5.92	2.00	0.09	0.70	0	39	429	13.2	7.8
970	380	7.61	3.88	3.50	0.08	0.15	0	36	440	12.1	7.8
982	245	5.10	2.70	2.14	0	0.26	0	41	455	12.4	7.0
991	420	7.36	3.97	2.50	0.07	0.74	0.07	45	519	15.6	8.8
1042	310	8.40	3.95	3.70	0.25	0.50	0	42	467	14.0	7.8
Mean	306 (±85)	7.70 (±2.04)	4.40 (±1.44)	2.82 (±0.90)	0.14	0.34	0.01	39.3 (±2.7)	432 (±30)	13.1 (±0.9)	7.7 (±0.5)
				Male Children of Unexposed Parents, Age <8							
801	540	12.36	5.19	5.44	0.74	0.99	0	39	471	12.4	
802	490	7.73	4.72	2.62	0	0.39	0	37	441	13.2	
803	500	10.80	6.48	3.78	0.32	0.22	0	37	430	12.8	
806	390	9.13	3.10	4.66	0.28	1.00	0.09	35	433	12.1	
807	420	9.35	4.96	3.27	0	1.12	0	39	480	12.1	
809	430	6.81	2.52	3.41	0.27	0.61	0	35	394	10.9	
870	465	8.90	4.09	3.92	0	0.89	0	40	449	12.8	
904	415	9.77	4.10	3.61	0.10	1.76	0.19	38	409	12.4	
905	585	11.50	6.67	[illegible].03	0.58	0.23	0	39	480	12.1	
924	450	7.98	4.95	[illegible].47	0.24	0.32	0	37	498	12.1	7.2
952	460	9.38	4.78	3.94	0.28	0.28	0.09	36	444	11.8	
972	515	8.15	4.40	2.85	0.08	0.81	0	36	424	11.5	
1002	395	15.13	8.47	3.93	0.30	2.42	0	35	465	11.8	
1004	335	14.00	8.40	4.62	0.42	0.56	0	40	422	11.5	
1006	390	7.40	2.81	4.00	0.15	0.37	0.07	35	422	12.1	
1009	380	9.03	3.70	5.24	0	0.09	0	41	454	13.6	
1010	490	11.60	6.38	4.29	0.23	0.70	0	38	457	12.1	
1014	390	11.25	5.63	3.71	0.11	1.69	0.11	35	405	10.9	
1015	625	13.50	2.97	9.86	0.41	0.27	0	33	535	10.0	
1017	355	11.43	5.03	5.72	0.46	0.23	0	40	510	12.8	
1018	585	14.87	8.03	4.61	0.59	1.64	0	34	527	10.0	
1024	310	7.78	3.27	3.66	0.54	0.23	0.08	35	476	10.9	
1027	405	11.47	3.90	7.00	0.23	0.23	0.11	38	518	11.8	
1028	445	15.37	6.92	6.46	0.61	1.38	0	36		11.2	
1032	470	12.88	6.18	5.15	0.26	1.03	0.26	38	525	13.2	
1037	510	6.58	3.09	3.16	0.07	0.26	0	31	406	10.6	
1038	310	9.12	4.20	3.47	0.91	0.46	0.09	35	430	10.3	
1039	505	10.53	3.69	5.79	0.21	0.84	0	39	436	11.8	
1040	520	6.98	3.07	3.42	0.21	0.28	0	32	441	11.2	
Mean	451 (±80)	10.37 (±2.55)	4.89 (±1.68)	4.42 (±1.50)	0.30	0.73	0.04	36.7 (±2.5)	457 (±39)	11.8 (±1.0)	

Individual Hematological Findings, 1962

Subject No.	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
				Female Children of Unexposed Parents, Age <8							
808	620	10.80	4.97	4.10	0	1.73	0	39	442	11.8	
810	470	8.76	4.38	3.94	0.09	0.35	0	37	436	11.5	
866	415	9.99	6.29	3.30	0	0.30	0.10	40	461	12.8	
901	465	11.60	3.72	6.29	0	1.63	0	36	410	11.2	
902	485	12.10	3.87	7.38	0.61	0.24	0	35		11.5	
903	415	11.25	5.96	4.28	0	1.01	0	37		11.4	
906	285	13.10	5.50	6.81	0.52	0.13	0.13	40		12.8	
923	580	15.44	6.95	5.56	0.31	2.47	0.16	39	480	12.4	
930	525	10.94	6.56	3.50	0.22	0.55	0.11	39	460	12.1	
954	355	14.73	10.16	2.95	0.30	1.33	0	36	450	12.4	
979	325	5.49	2.36	2.42	0.05	0.60	0.05	38	465	12.8	7.6
995	380	11.10	5.44	4.88	0.11	0.56	0.11	40	476	13.2	
1012	355	15.60	9.67	4.84	0.78	0.31	0	37	449	11.8	
1019	420	12.54	5.27	6.02	0.13	1.00	0.13	36	493	12.8	
1021	575	13.30	6.25	6.25	0.13	0.67	0	36	480	12.4	
1022	400	9.43	5.47	3.39	0.38	0.19	0	37	402	11.5	
1025	430	15.95	3.83	11.32	0.48	0.32	0	42	541	12.4	
1026	395	12.23	4.89	6.60	0.49	0.24	0	37	430	11.2	
1029	300	9.03	2.98	5.42	0.36	0.18	0.09	38	454	10.9	
1031	350	9.70	2.91	6.01	0	0.68	0.10	35		12.1	
1034	250	11.18	4.92	5.59	0.45	0.22	0	40		15.2	
Mean	418 (±97)	11.63 (±2.50)	5.35 (±2.16)	5.28 (±1.92)	0.26	0.70	0.04	37.8 (±2.7)	458 (±31)	12.2 (±0.9)	

APPENDIX 4

Individual WBC, Basophil, and Alkaline Phosphatase Determinations, 1962

Subject No.	WBC $\times 10^{-3}$	A.P. % Neg.	% Baso./4000 cell count	Subject No.	WBC $\times 10^{-3}$	A.P. % Neg.	% Baso./4000 cell count
1	6.8	86	1.00	60	8.2	93	0.78
2	5.4	34	1.10	61	7.6	78	0.48
3	9.0	34	1.70	63	5.1	53	0.55
4	6.4	75	0.60	64	5.8	67	0.50
5	8.8	91	0.95	65	7.3	20	0.78
6	7.4	89	0.80	66	6.4	62	0.83
7	6.5	71	1.25	68	4.2	96	1.28
8	7.5	72	1.08	69	5.3	85	0.90
9	7.3	89	—	70	5.5	60	0.58
10	6.0	85	0.50	71	7.7	63	0.63
11	4.5	92	1.08	72	5.7	72	0.73
12	5.8	81	0.58	73	5.5	82	0.70
13	6.6	91	0.53	74	13.3	94	0.90
14	—	58	1.23	75	7.9	91	0.60
15	7.5	89	0.73	76	5.9	93	0.78
16	5.2	93	0.43	77	5.8	61	0.78
17	8.1	82	0.58	78	5.7	89	0.60
18	5.8	70	0.88	79	8.7	60	0.95
19	9.1	85	0.53	80	9.1	78	0.50
20	9.2	84	0.60	81	5.2	83	0.75
21	6.8	80	0.78	82	6.9	0	0.68
22	5.3	83	0.58	83	5.6	81	0.85
23	10.1	17	—	84	7.8	68	0.45
24	5.4	78	0.75	85	10.9	98	0.63
26	7.6	80	0.68	86	8.4	90	0.65
27	6.2	92	0.68	87	9.2	93	0.53
28	6.1	83	0.83	88	6.9	92	0.53
29	6.9	90	1.10	89	8.7	75	1.08
30	7.1	86	0.70	91	6.5	38	0.48
32	8.0	76	1.18	92	10.9	17	0.68
33	6.6	67	0.70	93	9.8	20	0.75
34	5.4	86	0.85	94	8.9	22	0.63
35	9.9	92	0.88	95	8.8	—	0.68
36	5.0	88	0.40	96	8.7	88	0.45
37	7.7	86	0.55	97	7.2	90	0.60
39	4.9	64	0.58	98	10.8	98	0.68
40	6.7	64	0.60	100	9.1	97	0.43
41	4.4	85	0.60	101	18.1	93	0.55
42	5.9	91	0.70	102	8.7	98	0.50
43	7.4	5	0.53	103	11.4	74	0.58
44	5.8	76	0.48	104	9.4	97	0.78
45	4.2	90	0.95	105	10.6	71	0.70
46	3.9	90	0.53	106	10.3	24	0.53
47	6.3	82	0.53	107	12.7	95	0.83
48	7.0	81	0.78	108	12.7	98	0.58
49	6.4	73	0.93	109	9.5	84	0.75
50	7.6	61	1.23	110	8.5	28	0.53
52	9.7	45	0.95	111	9.3	29	0.78
53	8.0	90	0.88	112	8.0	11	0.75
54	5.2	64	0.53	113	6.7	48	0.83
55	4.9	78	0.83	115	10.5	80	0.68
57	5.1	91	1.18	117	9.9	85	1.05
58	5.3	87	1.00	118	9.8	90	0.90
59	8.8	85	0.70	119	16.7	91	0.80

Individual WBC, Basophil, and Alkaline Phosphatase Determinations, 1962

Subject No.	WBC – $\times 10^{-3}$	A.P. % Neg.	% Baso./4000 cell count	Subject No.	WBC $\times 10^{-3}$	A.P. % Neg.	% Baso./4000 cell count
120	13.5	79	0.55	862	6.2	94	0.50
122	10.3	86	0.50	863	7.6	96	0.53
123	7.5	76	0.73	864	7.8	98	0.83
124	9.9	84	0.98	865	4.9	91	0.75
125	12.3	6	0.55	866	10.0	81	0.68
126	9.6	66	0.75	867	8.0	90	—
801	12.4	91	0.40	868	4.9	84	0.75
802	7.7	95	0.65	869	10.6	87	0.50
803	10.8	64	0.58	870	8.9	66	0.78
805	8.3	94	0.58	875	7.3	79	0.63
806	9.1	90	0.73	878	4.9	97	0.58
807	9.4	77	0.70	879	14.2	82	0.83
808	10.8	98	0.55	881	6.3	88	0.70
809	6.8	89	0.35	882	7.4	91	0.75
810	8.8	71	0.73	883	6.0	91	0.78
812	10.0	88	0.83	884	7.3	87	0.68
813	7.2	95	0.78	885	7.8	88	0.75
814	8.6	89	0.70	886	8.9	78	0.63
815	7.3	94	0.75	887	6.1	84	0.58
816	9.0	81	0.65	888	5.6	88	0.68
817	13.4	83	0.75	889	4.5	83	0.50
818	9.1	65	0.50	891	6.4	85	0.53
819	5.3	90	0.60	892	7.0	84	0.55
820	7.8	98	0.85	893	10.5	28	0.78
821	8.1	96	0.75	894	10.5	39	0.58
822	8.0	66	0.73	895	8.6	62	0.63
823	5.6	88	0.55	896	11.0	26	0.63
824	6.3	89	0.78	897	8.1	84	0.78
825	9.1	75	0.70	898	7.2	92	0.85
826	7.2	90	0.73	900	—	82	0.75
828	7.3	87	0.58	901	11.6	80	0.70
829	5.3	73	0.38	902	12.1	23	0.68
830	6.2	83	0.70	903	11.3	89	0.50
832	6.6	61	0.83	904	9.8	47	0.90
833	4.8	86	0.78	905	11.5	81	0.50
834	8.8	90	0.63	906	13.1	85	0.53
835	10.4	2	0.98	908	6.2	84	0.58
836	6.6	70	0.50	911	10.7	60	0.63
838	5.7	77	0.68	912	9.6	54	0.48
840	7.2	77	0.73	913	5.6	87	0.58
841	8.8	80	0.73	914	6.5	80	0.53
842	8.8	88	0.90	915	5.2	76	0.75
843	6.7	28	0.75	916	7.4	74	0.63
844	5.5	30	0.75	917	5.6	90	0.68
846	6.8	89	0.85	919	6.1	93	0.63
849	7.1	88	0.70	921	9.1	69	0.75
851	12.7	77	0.50	922	5.6	94	0.68
852	10.1	86	0.75	923	15.4	84	0.80
853	7.4	42	0.48	924	8.0	91	0.63
855	6.0	94	0.45	926	13.1	96	0.68
856	11.2	30	0.60	928	5.4	75	0.63
858	6.9	82	0.68	929	5.5	96	0.75
859	6.4	100	0.58	930	10.9	84	0.55
860	—	93	0.60	932	6.9	98	0.70

Individual WBC, Basophil, and Alkaline Phosphatase Determinations, 1962

Subject No.	WBC $\times 10^{-3}$	A.P. % Neg.	% Baso./4000 cell count	Subject No.	WBC $\times 10^{-3}$	A.P. % Neg.	% Baso./4000 cell count
934	8.4	65	0.63	991	7.4	92	0.75
935	5.8	81	0.58	993	8.0	63	0.68
936	8.1	97	0.70	995	11.1	92	0.83
937	12.2	95	0.78	996	6.6	97	0.58
938	7.9	96	0.63	998	7.0	93	0.53
939	8.5	84	0.55	1001	7.0	93	0.68
940	7.2	73	0.78	1002	15.1	12	0.73
942	7.0	90	0.75	1004	14.0	59	0.58
943	7.8	95	0.68	1005	6.3	91	0.63
944	6.7	96	0.50	1006	7.4	88	0.65
945	11.4	39	0.58	1007	6.8	91	0.53
946	8.3	94	0.63	1009	9.0	82	0.60
947	14.4	28	0.68	1010	11.6	—	0.60
948	6.3	76	0.60	1011	—	91	—
950	13.9	88	0.73	1012	15.6	55	0.58
951	8.6	92	0.60	1014	11.2	74	0.75
952	9.4	81	0.63	1015	13.5	87	0.65
954	14.7	91	0.78	1017	11.4	90	0.63
955	6.9	48	0.60	1018	14.9	0	0.50
956	7.0	73	0.68	1019	12.5	80	0.70
957	8.7	78	0.70	1020	—	0	0.58
958	10.1	85	0.85	1021	13.3	85	0.48
959	8.3	84	0.60	1022	9.4	11	0.60
960	13.2	81	0.53	1024	7.8	75	0.55
961	5.5	96	0.73	1025	15.9	16	0.55
962	6.1	98	0.53	1026	12.2	21	0.70
963	6.1	96	0.63	1027	11.5	58	0.73
964	5.8	87	0.70	1028	15.3	77	0.60
965	8.6	92	0.75	1029	9.0	70	0.85
966	6.2	90	0.88	1030	—	97	0.48
967	5.6	86	0.73	1031	9.7	88	0.68
969	8.7	96	0.63	1032	12.9	91	0.88
970	7.6	95	0.53	1033	8.7	87	0.58
971	7.2	93	0.45	1034	11.1	56	0.73
972	8.1	93	0.50	1035	9.3	40	0.70
975	4.8	92	0.65	1036	7.6	84	0.55
977	7.1	91	0.78	1037	6.6	85	0.68
979	5.5	75	0.73	1038	9.1	72	0.63
980	—	—	0.58	1039	10.5	81	0.73
981	9.9	93	0.68	1040	7.0	92	0.50
982	5.1	90	0.45	1041	5.5	95	0.78
988	7.6	91	0.78	1042	8.4	69	0.50
990	8.4	88	0.98	1043	5.1	94	0.58

APPENDIX 5

Individual Physical Examination Findings, 1962

No. Age, Sex	PAST HISTORY	INJURIES	WEIGHT POUNDS	BLOOD PRESSURE Heart and Lungs	EENT	ABDOMEN GU or Gyn	SKIN
1 62 F	Obesity, Deafness Mild hypertension	Fixation, left elbow, trauma		155/90 Hypertension	Macular degeneration and lenticular opacities OS	Mild cystocele	Slight roughening and pig. back neck
4 46 M	Substernal pain, 1 year; slight hypertension	Traumatic Amputation, distal phalanx, lt. 2nd finger	154	120/70	Pterygium and lenticular opacities bilateral		
7 44 M			125	110/64	Pterygium bilateral		
9 31 M			122/80	Strabismus	Trichomonas of urinary tract		
10 32 M	Chronic lower abdominal pain		143	120/70	Pterygium left eye		
11 58 M	Kahn 3+	Scarring due to burn, rt. shoulder, lt. chest; fracture-rt tibial head (old)	115 thin	185/110 hypertension arteriosclerosis	3+4 retinal arteriosclerosis, strabismus Argyll-Robertson pupils	Prostate sl. enlarged	Sl. roughening and pig. lt. A.C.F., Few moles inferior to axilla on chest.
12 26 F	Hydatiform mole, bleeding, 1957		123	110/70	Old, traumatic choroiditis right eye	Endocervicitis	Biopsy nevus Histopath. Benign
13 66 F	Cough, sputum backache, "broke back" '54			120/66 Exp. rales rt. chest difficult resp.	Anterior Staphyloma and intraocular infection left eye		
14 33 F		Scar, rt. antecubital space	132	98/62		Liver palp. 1 cm down History of cystocele Preg. 7 mos.	back neck- pig. hyperkeratotic oval inf. scars?
16 47 M			125	110/68	Pterygium rt. eye. Thin eyebrows		Biopsy face, non spec., chr., inflamed. No acid fast bac.
18 29 F			106	108/62	Pinguecula rt. eye	Smear neg. for. Trich. Vag. discharge - cervical erosion	Biopsy scars back neck
22 25 F	Cough Sputum		101	95/60			
24 21 F	Cough Sputum		98	90/58			Dorsum feet pig variation sl. hyperkeratosis
26 21 M			166	110/70			
27 34 M			135	90/50 Brady Cardia	Dent. caries Tonsilar hyp. Pain-lft. eye, dental?		

Miscellaneous Neurological Tumors, etc.	BLOOD COUNTS WBC	HCT	PLAT.	LAB DATA, Urine, Pap., X-Ray, etc.	COMMENTS
	6.8	42	265	Pap, superficial squamous. Dry smear; Neg. for Ca; acc. lung marking rt. mid. chest cardiac enlargement (' 61)	
Fibroma mid-line D_6	6.4	48	195	Chest X-ray neg. ('61)	R_x trial of nitroglycerine
Small tumor lt. buttock ? lipoma	6.5	43	265	Small shadow left lower lung recheck	
	7.3	44	160		
Lipoma 5 x 5 cm. left shoulder	6.0	51	160	Chest X-ray neg.	R_x antipar
CNS Lues (?) Treated with Penicillin in 1957 Rhemberg pos.	4.5	40	340	Chest X-ray neg.	
	5.8	42	275	Pap: good estrogen level; Neg. Ca; Trichomonas Chest X-ray neg.	
2 x 2 cm. hard mass, left labia, kraurosis vaginae arteriosclerosis	6.6	40	525	Pap: scanty smear, Neg. Ca Chest X-ray - Scoliosis ('61)	R_x Vitamins, Resp. difficulty due to osteoarthritis Enucleation recommended
Wrist abn. wide distal epiphyses		36	420	Serum Iron 187 mg. Chest X-ray neg.	R_x Iron and Calcium
Vib. sense absent, lower extremities ? early leprosy	5.2	39	335	Chest X-ray neg. ('61)	R_x Skin check 3-6 mos.
Osteoarthritis	5.8	37	405	Pap. neg. for Ca. Severe inflammation	
	5.3	37	530	Chest X-ray neg.	
	5.4	36	350	Chest X-ray neg.	
	7.6	46	305	Chest X-ray neg.	
External hemorrhoids.	6.2	41	420	Chest X-ray neg.	R_x dental consultation EKG

NO. AGE SEX	PAST HISTORY	INJURIES	WEIGHT POUNDS	BLOOD PRESSURE HEART AND LUNGS	EENT	ABDOMEN Gu or Gyn	SKIN
28 76 F	–		111('61)	180/90 Tachycardia hypertension	4+ ret. arterios. bil. lens opac. Cat. sen.,mature both eyes		
29 73 M	Occ. asthmatic attack. History stroke, 1956		191	110/70	4+ ret. arteriosc. bil. lens opac. aphakia OS, sen. cat. rt. eye, Pterygium OD	Prostatic hypertrophy	
34 53 F	Pan-hysterectomy 1954		114	96/56	Lenticular opacities both eyes	Cervix bled easily	Back neck roughening, moles lt.
35 22 M			125	110/60			
39 23 F			109	90/60	Corneal Scar left eye		S.I. rough; pig back nk;dorsum rt,ft. pg.variat. w/slight hyperkeratosis
40 37 M		Deformity rt. forefinger	124	110/62	Pterygium OS Pinguecula OD		Mole rt. back neck; si. tenia versicolor.
41 52 M		Scar lt. inguinal area	120	110/70 Marked arteriosclerosis	Pterygium both eyes. Retinal arteriosclerosis 1+		Mole upper back rt.
43 75 F				118/60 Arteriosclerosis	Pterygium OS lenticular opacities OS		
45 40 F	Asthma		99	120/70		Cervical laceration endocerv.	
49 23 F	3 previous abortions	Scar rt. supraclavical area	134 obese	110/70			Pig.mac. neck, particulary rt; few lt. A.C.F.
50 42 M	Bilateral hallux valgus		175 obese	116/70			
51 33 F			94	118/60			
52 64 F	Pain rt. shoulder		113	110/76 Arteriosclerosis	Pterygium or lent. opac. or 2+ ret. arteriosclerosis		Moles side and front neck
55 83 M			134 ('61)	110/64 Arteriosclerosis	Senile cat. OD Aphakia OS Pterygium OS	Mod. gynecomastia.	
57 108 F				134/70 Arteriosclerosis	Vis. L.P. Cataracts OS		

MISCELLANEOUS NEUROLOGICAL TUMORS, ETC.	BLOOD COUNTS WBC	HCT	PLAT	LAB DATA, Urine, Pap., X-Ray, etc.	COMMENTS
Prominent heads of ulnae sl. fever	6.1	39	255	Chest X-ray neg. Pap: Some endocervical atypia	Light percep. only, due to cataracts, almost blind. Rec: Cat. removal
	6.9	41	235	Chest X-ray neg. ('61)	Light perception only due to cataracts. Senile cerebral arteriosclerosis.
	5.4	37	190	Chest X-ray Neg. Pap. neg. for ca., dry smear	R_x Biopsy Cervix
	10.0	49	280	Chest X-Ray neg.	
	4.9	39	435	Chest X-ray - Negative	
	6.7	43	445	Chest X-ray - 5 mm shadow lt. lower lung - Recheck	
Congenital dislocation of hips	4.4	44	320	Chest X-ray Neg.	
	7.4	40	200	Chest X-ray neg.	Marked Kypho-scoliosis.
	4.2	35	245	Pap: neg Ca: scanty smear. Chest X-ray: heart upper norm. Pl. ad. rt. diaph. area - recheck	
	6.4	40	320	Pap: - neg QNS	
	7.6	46	255	Chest X-ray neg.	
				Pap: neg. inflam.	Refused exam. 1962
Heberden's nodes	9.7	42	300	Chest X-ray neg. Osteoarthritis, scoliosis to right. Pap: neg., dry smear	
Osteoarthritis, Positive Romberg ? tabes dorsalis	4.9	30	260		R_x Removal of cat. Penicillin Vitamins
Kyphoscoliosis to right. Severe osteoarthritis. Subcutaneous masses both hips. Shortened left thumb.	5.1	40	225	Chest X-ray neg. ('61)	Light perception only, X-ray of hip masses not diagnostic ('61).

NO. AGE SEX	PAST HISTORY	INJURIES	WEIGHT POUNDS	BLOOD PRESSURE HEART AND LUNGS	EENT	ABDOMEN Gu or Gyn	SKIN
58 67 F			101	132/70	Cataract lt. eye mature; lent. opac. rt. eye	Lac. cervix, blood in smear, Fundal Ca?	Moles sides and front neck
59 42 F			88	124/80			Hyperkeratosis back neck, sl. itching
60 64 F			Obese	138/60 sys. M, art. heart dis. cardiac enlargemt.	Pterygium OS Lenticular opac.		
63 44 F			110 ('61)	110/70	Ac. Sinusitis	Cervical eros. Prolapse, vag. wall	
64 38 F			126	94/60	Double pterygium rt. eye	Cervical eros. Prolapse, vag. wall	Raised mole back of neck
66 38 F			133	100/60	Pterygium lt.eye Corneal scar rt. eye		Few pig. mac. back neck
68 52 M			127 ('61)	128/70			
70 25 F	Anemia		114	104/58			
71 36 F			121 sl. obese	116/70	Corneal Scar rt. eye	Cervical lac.	
73 26 M			162	110/64			
74 24 F			167 obese	108/70		Cervical eros.	
75 19 F	Lost baby 4 mos. congenital heart?		107	98/68 Sys. M Aortic ('61) None heard ('62)			
76 20 M	Rheumatic heart dis. ('56) chest dermatitis ('56)		137	120/70 Valvular ht. disease with D⧫S m	Tonsils enlarged		
77 34 M			113 ('61)	130/80	Pterygium lt.eye		
78 45 F			152 ('61) mod.obesity	105/70	Pterygium lt. Lent.opacities lt. retinal arteriosc.	Sm. laceration Cervix	

MISCELLANEOUS NEUROLOGICAL TUMORS, ETC.	BLOOD COUNTS WBC	HCT	PLAT	LAB. DATA, URINE, PAP., X-RAY, ETC.	COMMENTS
	5.3	36	190	Pap:? Neg, Trichomonas marked inflammation ('61)	Old pleuritic thickening diaphragmatic area. Rec. D. and D.
	8.8	37	515	Chest X-ray neg. Pap: Neg. Ca, Si. atrophia	
Mild Osteoarthritis	8.2	36	230	Pap: Blood inflammation	Rec. glasses
	5.1	41	305	Chest X-ray neg.-('61); Pap: neg.CaR$_x$ Ac. Sinusitis blood, inflammation	
Fibroma (?) Rt. finger 4th	5.8	37	340	Chest X-ray neg. Pap: Neg. Ca, marked inflammatory changes,dry smear	[illegible]: Biopsy tumor - "Giant cell tumor".
	6.4	40	265	Chest X-ray neg. Pap: neg. urine sugar 2+ ('61),fasting blood sugar neg.	
Sm.rt axillary node	4.2	41	205	Chest X-ray. neg. ('61)	
	5.5	29	300	Chest X-ray neg. Pap: neg. Ca., ?Trichomonas	Rec. Serum iron, bone marrow
	7.7	42	370	Chest X-ray-Calcif. neck,recheck Pap: neg. Ca.; marked inflammation	
	5.5	49	165	Chest X-ray neg.	
	13.3	50	295	Chest X-ray neg. Pap: neg. Ca. Atypia, clusters of benign endometrial cells	Constipated
Multiple skin infections, Yaws? Short 5th fingers, bilateral	7.9	46	250	Chest X-ray neg. Pap: neg.	
	5.9	43	310	Chest X-ray-heart normal size; few fibrotic markings extending upward from hilar region;recheck	Rheumatic heart disease-compensated.
Absent fingers and toes w/ ulcers of extremities and soles of feet. leprosy arrest.	5.8	46	450		
Bilateral short 5th finger	5.7	40	315	Pap? neg. Ca cervical cells, atypia, inflam.	

NO. AGE SEX	PAST HISTORY	INJURIES	WEIGHT POUNDS	BLOOD PRESSURE HEART AND LUNGS	EENT	ABDOMEN Gu or Gyn	SKIN
79 47 M	Bilateral incis. scars, groin		129	138/90	Lenticular- opac. enlarged parotid rt.		Scar lt. ear, pig. changes, 2 white nodules
80 54 M		1st lt. toe deformed	135	110/70 NSR/ fibrillation, pulse irreg. ('53)	Pterygium lt.		
82 58 M	Old facial paralysis		122	110/68	Pterygium lt. lenticular opac. lt. left facial nerve impaired	Prostate 2X normal; lt. testis 3 X rt.	

MISCELLANEOUS NEUROLOGICAL TUMORS, ETC.	BLOOD COUNTS WBC	HCT	PLAT.	LAB. DATA, URINE, PAP., X-RAY, ETC.	COMMENTS
Lt. inguinal nodes enlarged	8.7	49	175	Chest X-ray - recheck	
	9.1	44	275	Chest X-ray - sl. enlarged heart; mod. gen. emphysema	Heart block, auric, fib.
	6.8	38	450	Chest X-ray - neg. ('61)	R_x - glasses

NO. AGE SEX	PAST HISTORY	INJURIES	WEIGHT POUNDS	BLOOD PRESSURE HEART LUNGS	EENT	ABDOMEN Gu Gyn	SKIN
823 20 M			125	116/60	Exophoria		
825 20 F			111	82/50		Liver edge down 1cm;Sl. erosion cervix	
826 25 F	Stillbirth 1959		92	90/56	Large Tonsils	Mild cervical erosion	
827 22 M							
828 21 M		Scars around knees	106	106/58			Pig. back neck
829 23 F			109	90/60	Corneal Scar, lt.	Cerv. Lacerations	
830 23 M			151	120/80			
831 22 M			121('61)	110/56('61)	Tonsils enlarged	Guarding RLQ (61) Irritated intestine?	
832 24 F	Left ovarian cyst '58		99	80/40		Mass LLQ, sl. tender;? cyst	
833 29 M	Uniform Pig. hands & feet.		131	110/50 sys m,aortic area (61)not noted(62)	Tonsils enlarged		
834 28 M			120	120-68	Pterygium,lt.		
835 28 F	Constipation		100	100-48		Preg-'62;lt. breast>rt;ovary palp; thickened cervix ?	
836 29 M			122	104/64	Foreign body lt.cornea,coral?		
838 30 M			137	110/60			
839 34 F	Fibroma Uterus				Congenital Nystagmus('59)		

MISCELLANEOUS NEUROLOGICAL TUMORS ETC.	BLOOD COUNTS WBC	HCT	PLAT	LAB DATA, Urine, Pap, X-Ray, Etc.	COMMENTS
Inguinal Adenopathy	5.6	41	260	Chest X-Ray - Bil. apical Pleur. thickening - not signif.	
	9.1	38	315	Chest X-Ray - neg Urine prot. 100 mg.	
Patella moves lat.' flexion def. fingers	7.2	35	520	Chest X-Ray - recheck infilt. lt base; pl. adhesions both bases, Pap- neg	
					Not examined '62
Tiny axillary nodes	7.3	47	220		
	5.3	35	380	Chest X-Ray - neg. Fe 70.mpg Serum Iron 70.6 mpg	Rec. Price-Jones; Vit A on sera Anemic tendency
	6.2	47	350	Chest X-Ray - neg.	
					Not examined '62
Short 5th fingers	6.6	37	380	Chest X-Ray - recheck Pap - neg	
	4.8	37	380	Chest X-Ray - neg	
	8.8	46	255	Chest X-Ray - recheck, Shadows over heart	
	10.4	38	270	Chest X-Ray - neg Pap - neg Ca; marked inflam.	
Bilat. shortening 5th fingers ('61)	6.6	46	320	Chest X-Ray - neg	See chart for eyes R_x remove coral from eye
	5.7	49	265	Chest X-Ray - neg	
					Not examined since '59

NO. AGE SEX	PAST HISTORY	INJURIES	WEIGHT POUNDS	BLOOD PRESSURE HEART LUNGS	EENT	ABDOMEN Gu Gyn	SKIN
840 32 M			139	102/60	Pterygium, rt.		
841 29 F	-		153	110/70			
842 38 M		5th missing finger, rt; 2nd finger lt.	146	108/60			
843 33 F	History yaws, old retinal hemorrhage ('58)		102	90/58			
844 43 F	Myocardial damage EKG?		109	100/70	Pterygium, rt.	Liver + 3 cm	
845 32 M			140(61)	114/70 (61)			
846 39 F	?Syphilis		131	110/70		Endocerv., severe	
847 41 M	Hypertension						
848 42 F	Argyll Robertson pupil; neuro-syphilis (?)						
849 43 M	Depig. area, skin		207 obese	118/70 ? Pul. sys m(61) not heard ('62)		Hemorrhoids (61)	
850 52 M	Arterosclerosis mild; hypertension						Acute exfoliative type dermatitis
851 53 F	?Hypertension 150/96('59) ?Cerebral polyp Myocardial damage		159 obese	120/70 Arteriosclerosis mild	Pterygium lt & rt	Cervical laceration and erosion, bleeds easily	
852 58 F	?Hypertension		100 (minus 16 lbs.)	146/70 Spl. 2nd sound; sys. m	Pterygium, lenticular opacities rt,		
853 57 M	Deafness Emphysema ?		152	124/80 Arteriosclerosis	Pterygium lt. Lenticular opacities. Nodule(2cm),lt lobe thyroid	Liver + 2 cm	Vitaligo on trunk
855 58 M	Hemorrhoidectomy ('61)		145	110/70 Arteriosclerosis mild	Pterygium, lt & rt Lenticular Opacities	S1 hypertrophy rt lobe postate	
856 55 M			120	130/80	Lenticular Opacities lt.		

MISCELLANEOUS NEUROLOGICAL TUMORS ETC.	BLOOD COUNT WBC	HCT	PLAT	LAB DATA, Urine, Pap, Rx-Ray etc.	COMMENTS
	7.1	46	305	Chest X-ray- neg.	
	8.8	49	450	Chest X-Ray - rt. hilar prom.?, recheck Pap: neg ca; Prob Trichomonas	
	8.8	49	325	Chest X-Ray - neg	
	6.7	37	405	Chest X-Ray - neg Pap. neg ca; squamous atypia	R_x Fe, '61
	5.5	41	235	Chest X-Ray - neg Pap: neg ca; marked inflam.	
1cm Visicle, rt buttock ? herpes ('61)					Not examined '62
	6.8	43	285	Chest X-Ray - neg Pap:	Gyn consult.
					Not exam. '62
					Not Exam. '62
	7.1	47	465	Chest X-Ray - neg	
					no phy. exam. '62 R_x hydrocortone oint.
	12.7	36	295	Chest X-Ray - neg Pap: neg ca; inflam	
	10.1	37	465	Chest X-Ray - neg ('61) Pap - neg. ca	
Lipoma rt thigh (6x6in)	7.4	43	340	Chest X-Ray - rt hilar prom? Recheck Urine sugar 4+ FB sugar 248	Diabetes R_x diabetes
node rt axilla	6.0	43	340	Chest X-Ray - neg Proternuria ('59)	Check Urine protein
Bil. node 1cm axilla	11.2	38	410	Chest X-Ray - neg (61)	

NO. AGE SEX	PAST HISTORY	INJURIES	WEIGHT POUNDS	BLOOD PRESSURE HEART LUNGS	EENT	ABDOMEN Gu Gyn	SKIN
858 67 F	hypertension, osteoarthritis, cervical discharge, goitre, fixed chest		98	170/80 ?cardiac enlargement myocardial damage(?)	pterygium, rt. lenticular opacities goitre(?)	cervical discharge('61	
859 69 F	mild hypertension		124	140/80 cardiac enlargement; arteriosclerosis mild	healed corneal scar; lenticular opacities		
860 72 M	acromegaly? ext. hemorrhoids,	Burn scar rt arm; old fracture rt lower arm	121(61)	120/62 arteriosclerosis	Corneal scar lt; aphakia, rt.		
862 89 M				110/70 Card.enlargement sys m., severe arteriosclerosis	pterygium lt&rt lenticular opacities;retinal arteriosl B+,choroidal scar rt.		
864 37 M			150	120/80	enlarged tonsils	Sl. enlarged prostate	
865 29 F	7 children; unilateral exophathalmus		100	90/40		Liver 1cm↓	Bl. mole chest
867 35 F			102	115/80	sl. hyper. tonsils		discoloration skin rt shoulder,birthmark?
868 39 M	Hist. pain legs		154 Steady loss 182 ('61)	102/62			Face pocked
871 50 F	Arteroisclerosis mild,pinguecula corneal pig.						
872 20 M				110/55('59)			
873 44 M	Arteroisclerosis mild;prostatic hypertrophy;URI						
875 45 M	occas.convulsions?		132	100/70	pterygium rt, corneal scar, rt		ulcers lower legs
876 24 F	Corneal pig.						
877 24 M	Keloid rt. shoulder		133 ('61)	124/80('61)			
878 62 M	Hypertension? emphysema, ?prolaped disc '59myocordial damage (61)		175	130/70 arteriosclerosis mild	pterygium lt, lenticular opacities,		hyperkeratotic areas on neck

MISCELLANEOUS NEUROLOGICAL TUMORS, ETC.	BLOOD COUNT WBC	HCT	PLAT	LAB DATA, Urine, Pap X-Ray, Etc.	COMMENTS
Osteoarthritis; bil. enlargement ulnae	6.9	39	235	Pap- neg Ca	R_x glasses
Short left 4th toe; Kyphosis; Osteoarthritis rt shoulder & fingers	6.4	39	295	Chest X-Ray - mild cardiac enlargement. node Hilar; recheck	
Osteoarthritis Kyphoscoliosis, enlarged knees, difficulity walking, ? tabes				Chest X-Ray - neg ('61)	R_x - Fe ('61)
Severe arteriosclerotic heart disease, paralysis	6.2	40	335		
Sl. enlarged nodes axilla, lipoma behind lt. ear	7.8	48	380	Chest X-Ray - neg	
	4.9	34	345	Chest X-Ray - neg Fe 85.4 Serum Iron 85.4 mμg	Rec - Price-Jones anemic tendency
	8.0	38	415	Chest X-Ray - neg	
	4.9	44	350	Chest X-Ray - neg	Weight loss
					Not exam. since '59
					Not exam. since '59
					Not exam. since '59
Lipoma, 2x3in.lt thorax	7.3	49	315	Chest X-Ray - pl. thick lt base	
					Not exam. since '59
					Not exam. in '62
Rheumatoid Arthritis?	4.9	39	125	Chest X-Ray - neg	

NO. AGE SEX	PAST HISTORY	INJURIES	WEIGHT POUNDS	BLOOD PRESSURE HEART AND LUNGS	EENT	ABDOMEN GU or GYN	SKIN
881 31 M	Varicose veins lt. leg; back strain		197	90/50			
882 30 M	Tumor lt parotid, removed 1950 (benign)		125	90/60			
883 51 M	Facial asymmetry, corneal opacity lt.						
884 67 M	Scar lt. groin, old TBC?; spondylitis, RCN '50 ?		140	110/70 Arteriosclerosis mild	lenticular opacities	hemorrhoids, [illegible] [illegible], testicles	
885 22 M			133	130/70		Rt. tender RLQ?? worms.	
886 56 M	Osteoarthritis						
888 34 F	Cong. shortening 5th metacarpal both lt. eye - [illegible], 10 [illegible]			100/60			
890 37 F			100('61)	100/62 ('61) A2P2		Hard cervix, / indur. rt adnexa; Bl. prolapse	
893 64 F	Diabetes myocardial damage ('61)		100 112('61)	90/50 Wheezing both lungs ('61)	Pterygium lt; corneal scar lt. (Arterio-sclerosis, retinal)	Thrombosed hemorr.	
894 63 F	Chronic Cough		104	130/72 Rales, rt base, few lt base arteriosclerosis, mild	lenticular opacities,		
895 33 F	6 miscarriages; last Feb. '62 3 mo.		122	140/64	Pinguecula rt & lt	cervical erosion	linea vert., upper chest
896 22 F	Backache, worms		94	80/40			
897 64 M	Pain lt shoulder weakness		154	120/60	Pterygium lt, lenticular opacities; Arcus sen. lt.		
898 64 F	Arthritis lt knee; Vaginitis		169	110/80 Arterio-sclerosis	Pterygium lt. lenticular opacities enlarged tonsils	cervix bleeds easily	
899 66 M	Dupuytrens contr. both hands		124(59)	136/70 (59) Arterio-sclerosis			

MISCELLANEOUS NEUROLOGICAL TUMORS ETC.	BLOOD COUNT WBC	HCT	PLAT	LAB DATA, URINE, Paf., X-Ray, etc.	COMMENTS
No Vericose veins noted, back ache lt lumbar region;	6.3	43	275		Back strain
Enlarged distal ulna?	7.4	40	155	Chest X-Ray - neg	
	6.0	43	455		Not exam since 1959
Arthritis, spine; Dupuytrens contr., bilat,	7.3	43	190	Chest X-Ray - neg Urine sugar 3+ FB sugar 132 non FB sugar 212	Mild diabetis
Sm. axillary nodes nodule, ½ cm below typhoid	7.8	45	365	Chest X-Ray - neg	
	8.9	42	425		Not exam since '59
Few sm. nodes neck	5.6	40	380	Pap - neg	
	4.5	41	490	Pap- neg	Gaining wt '61 Not exam. '62
Sm. axillary nodes,	10.5	41	235	Chest X-Ray - neg ('61) Pap. neg ca, Trichomonas Urine sugar 4+; FB sugar 281	R_x diabetes
Nodes, lt axillary Osteoarthritis ('61)	10.5	41	320	Chest X-Ray - heart upper limits normal ('61) Pap - neg	
	8.6	42	510	Chest X-Ray - neg Pap - neg for ca; inflam; atypia Urine prot. 100mg	R_x Vit B6
Arthritic Changes knees?	11.1	38	450	Chest X-Ray - neg Pap - neg for Ca, cellular atypia	
	8.1	43	210		
Arthritis	7.3	37	310	Chest X-Ray - neg Pap - neg	
Osteoarthritis Dupuytren's Contracture? '59					Not exam. since '59

AGE	NO. SEX	PAST HISTORY	INJURIES	WEIGHT POUNDS	BLOOD PRESSURE HEART LUNGS	EENT	ABDOMEN Gu Gyn	SKIN
73	908 F	Arteroisclerosis mild;heberden's node;hypertension U.R.I.;		104	180/90 hypertension arteriosclef s	penguicula, rt lenticular opacities;retinal arteriosc1.1*		
60	910 M	arteriosclerosis mild;deafness; prostatic hypertrophy; hosp. majuro abd.pain '61.		102	120/60		Prostate sl. enlarged	
27	914 F	Worms		102	90/48			
65	915 M	thickened rt cord;		127	110/70	pterygium rt; lenticular opacities;retinal arteriosc1.	Prostate 2x normal	
38	916 F	Deafness?		130 143 ('61)	100/70	Pterygium lt;		Ulcer lt. lower leg. few mac. back neck
43	917 M	Myocardial damage EKG ('59)		176	110/76 no abnormal sounds		Bilat. tenderness	
64	918 M	Diabetes	Scars groin & rectum		130/76('61) Wheezes lt. chest ('61)	Arcus senilis		
30	920 M	Healed burn scar buttocks		132(59)	108/72('61)			
38	922 F			94	90/60			
49	928 F		Burn scars lt. chest	128	106/60	pterygium lt;		
64	929 F	Never preg.		134	130/80	pterygium rt; cataract lt.;		
27	932 F	Anencephalic baby		98	90/58	Corneal scar,rt.		
27	934 F			121	100/70	sl. enlarged tonsils	tenderness RLQ	
65	935 M	Osteoarthritis U.R.I.			100/62	penguicula lt & rt, poor vision, lenticular opacities		hypo pig. ant. shins
72	936 F				130/62 diastolic m grade 2 base of heart		Tenderness lowr abdomen; inflam cervix.	

MISCELLANEOUS NEUROLOGICAL TUMORS, ETC.	BLOOD COUNTS WBC	HCT	PLAT	LAB DATA, Urine, Pap, X-Ray, Etc.	COMMENTS
	6.2	44	205	Chest X-Ray - recheck Pap - neg.	
Sm. bilat. axillary nodes; shortened 5th fingers;					
	6.5	36	430	Chest X-Ray - lt. substern. thyroid?, recheck Pap - neg F.B. sugar 83	
Enlarged rt Ulna; Osteoarthritis '61?	5.2	42	330	Chest X-Ray neg ('61) Pap - neg	R_x refraction
	7.4	35	615	Chest X-Ray - neg FB sugar 83 Urine sugar 2+ ('61) Pap - neg ca; poor smear	R_x Fe ('61)
?Arthritis lt. wrist	5.6	47	245	Chest X-Ray - rt apical density, recheck urine sugar 1+ non FB sugar 109	R_x aspirin; uric acid det.
				Chest X-Ray - neg ('61) Urine sugar 4+ ('61)	Rec - fasting blood sugar Not exam. '62
Sm. cervical nodes				Urine sugar 3+	Rec - fasting blood sugar Not exam. '62
Osteoarthritis	5.6	38	355	Chest X-Ray - neg Pap - neg Ca; inflam.	Rec Checkup Tbc.
Osteoarthritis Heberden's nodes ('61)	5.4	36	370	Chest X-Ray - heart upper normal Pap - neg Ca; inflam	R_x Fe ('61)
	5.5	38	360	Chest X-Ray - neg Pap - neg Ca, endocervical atypia	
	6.9	37	435	Chest X-Ray - neg Pap- neg non FB sugar 210	R_x Fe ('61) Diabetes? fasting blood sugar
	8.4	41	285	Chest X-Ray - neg Pap - neg	
Osteoarthritis mod. arteriosclerosis	5.8	43	350		R_x refraction
Osteoarthritis diminished reflexes	8.1	38	150	Urine sugar 1+ non FB sugar 207	Diabetes? fasting blood sugar

NO. AGE SEX	PAST HISTORY	INJURIES	WEIGHT POUNDS	BLOOD PRESSURE HEART LUNGS	EENT	ABDOMEN Gu Gyn	SKIN
938 23 F	6 toes lt foot, webbed		95	108/70		Cervicitis, severe erosion	
941 61 F	Hypertension		114(61)	140/90 ('61)			
942 47 F	Cardiac Enlargement(61)		121	100/64		Sl. tenderness rt	Furuncles, legs
943 34 M			143	110/70			
944 37 M	Hist. of backache		176	110/80			Pig. mole RLQ
945 37 F	Hist. of productive cough		83	110/80 ?Sys. m Gr. 2 Pul. area		Tend. RLQ? Cervix lacerated	
947 54 M	Hypertension Arthritis Myocardial Damage EKG(59)		108	198/92 Advanced arteroisclerosis Wheezes both lungs	Pterygium lt & rt, Cataract, rt; Old choreo-retinitis, lt.	Enlarged Prostate	Depig. Areas
951 29 F	Hist. night blindness		137	110/70		Cervicitis	
956 53 F	1 baby died at birth, 1 stillborn; myocardial damage, EKG(59)		127	120/70 mild arteriosclerosis	Pterygium rt		
957 52 F	Loss hearing Myocardial damage ('59)		152	106/72 mild arteriosclerosis	pterygiae lenticular opacities		
958 31 M	GI symptoms, ?amoebia; ?perineal cyst?		126	142/88 Wheezing both lungs ('61)		Liver 3cm↓, Prostate lt lobe enlarged	
961 69 M	Paresthesias leg, diabetes?		143	110/70	pterygium lt.	Hilat. Hydrocode	
963 44 M			143	100/68	pterygiae	Liver 1 cm↓	Scar from tumor removed lt breast
964 86 M	Deafness (?)		123	130/70 arteroisclerosis	Cataracts	Sl. enlarged prostate	
966 31 M			144	102/72	Corneal pig. rt		

MISCELLANEOUS NEUROLOGICAL TUMORS, ETC.	BLOOD COUNT WBC	HCT	PLAT	LAB DATA, Urine, Pap, X-Ray, Etc.	COMMENTS
Enlarged Cervical nodes Polydactylism	7.9	40	210	Chest X-Ray, neg Pap - neg	R_x GYN checkup
				Pap - neg Ca, inflam, trichomonas	Not exam '62
	7.0	40	300	Chest X-Ray - recheck Pap- neg Ca; some keratinization; trichomonas	
	7.8	46	355	Chest X-Ray - neg	
	6.7	48	230	Chest X-Ray - neg	
	11.4	45	340	Chest X-Ray - neg Pap - neg	
	14.4	41	160	Chest X-Ray - neg	
	8.6	41	185	Chest X-Ray - neg Pap - neg Ca, inflam.	
	7.0	37	410	Chest X-Ray - neg Pap - neg Ca, inflam. P.B. sugar 89	R_x removal pterygium
	8.7	39	370	Chest X-Ray - neg Pap - neg	
Pilonidal cyst	10.2	41	450	Chest X-Ray - neg	Rec. liver function test
Osteoarthritis, knees	5.5	43	395	Chest X-Ray - neg Urine sugar 2-3+ P.B. sugar 99	
	6.1	45	280	Chest X-Ray - neg	
Severe kyphosis, Osteoarthritis, Lipoma lt knee	5.8	37	355	Chest X-Ray - neg	R_x Cataract removal
	6.2	46	360	Chest X-Ray - neg	

NO. AGE SEX	PAST HISTORY	INJURIES	WEIGHT POUNDS	BLOOD PRESSURE HEART LUNGS	EENT	ABDOMEN Gu Gyn	SKIN
967 20 M			144	120/80	pterygium, lt		
969 44 M	Myocardial damage EKG ('59)		124 ('61	Arterio-sclerosis 116 mild 90/58 Coarse rales	pterygium, lt.		
970 48 F	Myocardial damage EKG ('59)		103	100/60 Arteriosclerosis mild	pterygium lt.	liver↓1cm('61) Endocervicitis	Impetigo, legs
971 20 M			126	125/70	Dental caries		Vitaligo spots back & chest
973 53 M	mild hypertension	Burns lt lower leg, inguinal incisions	133('61)	Arteriosclerosis mild, 140/90('61) Frothy sputum, wheezing both lungs		Sl. enlarged prostate	
975 40 M	Traumatic Arthritis		146	102/70	pterygiae		
982 41 F	Hypertension		140	170/108 Hypertension Arteriosclerosis mild	pterygium lt.	endocervitis	Impetigo
984 30 F				114/74 ('61)		Liver 1cm↓ mass RLQ, probably feces; prolapse rectum	
991 54 F	? Diabetes		173('61) obese	120/78		Lacerated cervix ('61)	
1001 28 F			134	110/70		Endocervicitis	
1005 29 M	Hist. of sm. inguinal herniae, rt. indirect, easily reduced		170 obese	125/70	Ocular protrusion, lt,old; geographic tongue;		
1007 51 M		rt. inguinal scar	153 170 ('59)	120/70	pterygium, lt.	Sm. testis	
1041 58 M			190 obese	130/90	pterygium, rt. lenticular opacities,corneal pigmentation, mild arcussenilis 4+		
1042 46 F		Midline abd. scar from cesarean scar 1958	132 Sl. obese	110/72		Uterus Sl. enlarged cervicitis	
1043 28 F			91	100/50	Pinguicula		

MISCELLANEOUS NEUROLOGICAL TUMORS, ETC.	BLOOD COUNT WBC	HCT	PLAT	LAB DATA, Urine, Pap, X-Ray, Etc.	COMMENTS
				Chest X-Ray - neg	
	8.7	44	470	Chest X-Ray - ? Plural thickening rt cortal sinus recheck	Resp. eval
Ganglion lt. mid. wrist	7.6	36	380	Chest X-Ray - neg Pap - neg	
Adenopathy, axillary inguinal	7.2	46	365	Chest X-Ray - neg	
Lipoma lt. shoulder, 8x5 cm				Chest X-Ray - Fibrotic lung markings lt base	Not exam '62 R_x dental ('61) Rec - recheck X-Ray
	4.8	43	155	Chest X-Ray - neg	
Several sm. nodes lt. neck	5.1	41	245	Chest X-Ray - neg ('61) Pap - neg	
					Not exam. '62
	7.4	45	420	Pap - neg Urine sugar 4+ F.B. sugar 430	R_x diabetes
	7.0	39	140	Chest X-Ray - neg Pap - neg Ca, inflam.	
	6.3	51	365	Chest X-Ray - neg	Check for hernia
Reflexes depressed fibroma wrist?	6.8	39	265	Chest X-Ray - neg.	
	5.5	44	215	Chest X-Ray - neg Urine sugar 2-3+ non FB sugar 135	Urine and blood sugar
	8.4	42	310	Chest X-Ray - neg Urine sugar 2-3+ Urine prot. 100 mg non FB sugar 169	GYN checkup check diabetic tendency
	5.1	40	240	Chest X-Ray - neg	

APPENDIX 6

Pathologic-Anatomical Diagnoses* on 78-yr-old Woman in Exposed Group (No. 56)

Main Diagnoses

Fracture of vertebral body L_1. (No mention is made of the spinal cord.)
Fractures of the left 5th to 8th ribs.
Retroperitoneal hematoma.
Ecchymoses in the vicinity of the rib fractures.
Focal fibrosis of the myocardium.
Stasis and edema of the lung and liver.

Other Diagnoses

Patchy endocardial calcification of the heart.
Brown atrophy of the myocardium.
Generalized arteriosclerosis with
(a) atheromatosis, in particular of coronary arteries and aorta,
(b) Moenckeberg's media sclerosis,
(c) arteriolar hyalinosis in many organs.
Atelectasis of the middle lobe of the right lung.
Patchy atelectasis, emphysema, focal fibrosis, and coniosis of the lungs.
Pleural adhesions on the right side.
Bronchitis, chronic, mucopurulent. Bronchiectasis.
Atrophy of bronchial mucosa.
(Fat embolism of the lung?)
Atrophy, fibrosis, and stasis of the spleen. Atrophy and fibrosis of lymph nodes.
(Hemosiderosis of the spleen?) Atrophy of the thymus.
Lipomatosis of the bone marrow, particularly in the vertebral body, with decreased mass of blood-forming tissue.
Osteoporosis. Scoliosis of the vertebral column.
Arteriolosclerotic scars in the kidney (benign nephrosclerosis).
Capsular fibrosis of the kidney.
Small kidney cysts.
Calcifications in the kidney. (Fat embolism of the kidney?)
Small papillary cystadenoma of the kidney.
Atrophy of the pelvic mucosa of the kidneys.
Atrophy and fibrosis of the ovaries.
Atrophy and fibrosis of the uterus.
Telangiectasis in the uterus.
Brown atrophy of the liver.
(Hemosiderosis of the liver?)
Slight portal fibrosis of the liver.
Subserosal fibrosis of the gall bladder.
Atrophy and slight fibrosis and lipomatosis of the pancreas.
Peritoneal adhesions between hepatic flexure of colon and the liver.
Atrophy and nodular hyperplasia of the adrenal glands.
Atrophy of the thyroid gland.
Small cysts of the thyroid gland.
Atrophy of the skin.
Atrophy of epidermal appendages of the skin. Greying of hair.
Epidermoid cyst of the skin.
Sweat gland cyst of the skin.
Nevus pigmentosus of the skin.
Elastosis of the skin.
Atrophy of the mammary gland.
Osteoarthrosis (noted for fingers).
Corneal opacities.
Arcus senilis.
Loss of teeth.

*Summary prepared by Dr. Hans Cottier.