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9/29/86

HANFORD HEALTH EFFECTS PANEL MEETING

SEPTEMBER 22-26, 1986

RIVERSHORE MOTOR INN - RICHLAND, WASHINGTON

A G E N D A *

<u>Sunday</u> 9/21	6:00 p.m.	Registration and Social	
<u>Monday</u> 9/22	8:00 a.m.	Welcome	Nancy P. Kirner
	8:05	Introduction	Curt Eschels representing Governor Gardner
	8:15	Overview and Procedural Rules	Dr. Glyn Caldwell, Chair
	8:45	State of Oregon	Oregon Division of Health
	9:00	State of Idaho	Idaho Division of Health
	9:15	Confederated Tribes of the Umatilla Indian Reservation	Bill Burke or Designee
	9:30	Nez Perce Tribe	Ron Halfmoon or Designee
	9:45	Yakima Indian Nation	Russell Jim or Designee
	10:00	Break	
	10:15	History and Current and Future Status of Hanford Operations	U.S. Department of Energy
	10:30	Environmental Monitoring at Hanford	U.S. Department of Energy
	11:00	Public Comment	
	12:00 p.m.	Lunch	

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Hanford Health Effects Panel Meeting
Page Two - Agenda

1:30	Survey of Health Concerns	Advisory Council	
1:45	Hanford Effluent Monitoring and Controls	U.S. Department of Energy	
2:15	DSHS Environmental Monitoring	John Erickson, DSHS	
2:30	Regional Monitoring and Quality Assurance Task Force	Robert R. Mooney, DSHS	
2:45	Hanford Monitoring Needs	Tim Conner, HEAL	
3:00	Break		
3:15	Review of Historical Documents and Dose Assessment	Allen W. Conklin, DSHS; and Dr. Jim Rutenber, CDC	
4:00	Hanford Historical Document Review Committee Goals	Dr. Royston Filby	
4:15	Public Comment		
7:00	Public Comment Session		
<u>Tuesday</u> <u>9/21</u>	8:00 a.m.	Introduction and Summary of Monday's Activities	Dr. Glyn Caldwell
	8:30	Hanford Worker Study	U.S. Department of Energy
	9:15	Mancuso's Worker Study	Dr. Alice Stewart
	10:00	Break	
	10:15	DSHS Epidemiological Work	Dr. Sam Milham
	11:00	Public Comment	
	12:00 p.m.	Lunch	
	1:00	Panel to Begin to Address or Prioritize Specific Questions and Answers	
	3:00	Aerial Tour of Hanford for Panel (two trips - up to one hour each)	
	7:00	Evening Session - Yakima Room	

Hanford Health Effects Panel Meeting
Page Three - Agenda

Wednesday All Day Deliberations by Panel - Yakima Room
9/24

Thursday All Day Deliberations by Panel - Yakima Room
9/25

Friday 9:00 a.m. Summary Report of Deliberations
9/26 Results to Public - Ballroom 1

* *All sessions, unless otherwise noted, are in Ballroom 1.*

MEMBERS OF THE HANFORD HEALTH EFFECTS REVIEW PANEL

- Glyn Caldwell, M.D.
Panel Chair Assistant Director, Arizona Department of Health Services. He is a cancer epidemiologist who has conducted research on health effects of nuclear weapons testing and was formerly with the Centers for Disease Control.
- Robert Alvarez Director of the Radiation and Health Project for the Environmental Policy Institute, Washington, D.C. He has conducted and sponsored studies of the environmental impacts of DOE facilities and has been an advocate for stricter regulation of the nuclear power and weapons industry.
- Henry Anderson, M.D. Chief, Section of Environmental and Chronic Disease Epidemiology, State of Wisconsin Division of Health. He is board-certified in occupational medicine and has conducted numerous epidemiologic studies of occupationally-related diseases.
- Allen Benson, Ph.D. Instructor of Chemistry, Spokane Falls Community College. He has studied the environmental effects of the Hanford facility and has provided technical consultation to the Hanford Education Action League, (H.E.A.L.).
- Steven Blum, Ph.D. Assistant Director, Division of Environmental Epidemiology, New York City Department of Health. He is trained in environmental and occupational epidemiology and has conducted research on the health effects of ionizing radiation at the Oak Ridge Associated Universities.
- Donald Hendricks Private health physics consultant to entities such as the State of Washington and the Council of Energy Resource Tribes. He is the former director of the Environmental Protection Agency Office of Radiation Programs, Las Vegas Facility.

Vilma Hunt, B.D.S

An anthropologist and epidemiologist who has studied the effects of occupational hazards upon women. She was Professor of Environmental Health at Pennsylvania State University and Deputy Assistant Administrator for Health Research, Office of Research and Development, Environmental Protection Agency.

Vietchau Nguyen,
Ph.D.

President of Environment and Water Resource Management, Minneapolis, Minnesota. He is a civil engineer with extensive consulting and research experience in the field of hazardous and nuclear waste management. He serves as a consultant to the Yakima Nation for its Columbia River environmental monitoring project.

Lincoln Polissar,
Ph.D.

Associate Professor, Department of Biostatistics, University of Washington. He conducts epidemiologic research in the field of environmental epidemiology and is an Associate Member of the Fred Hutchinson Cancer Research Center.

James Smith, Ph.D.

Chief, Physical Agents Effects Branch, Division of Biomedical and Behavioral Science, National Institute for Occupational Safety and Health, Centers for Disease Control. He is a physicist and health physicist with extensive research experience in assessing internal exposure to radionuclides.

David Willis, Ph.D.

Professor of Radiation Biology, Oregon State University. He conducts research and is a consultant in the field of radiation biology and radioecology.

Harold Wyckoff,
Ph.D.

Radiation physicist and Chairman of the International Commission on Radiation Units and Measurements. He was formerly with the National Bureau of Standards, the Armed Forces Radiobiology Research Institute and the Bureau of Radiological Health.

Bernard Shleien,
Pharm. D.

Certified health physicist, formerly with the U.S. Food and Drug Administration. He studied extensively the effects of Iodine-131 on the thyroid and radiation dosimetry. The past two years he was head of the Radiation Protection and Isotopes Department, Center for Environmental Research, Tel Aviv University, Israel.

ANDREA BEATTY RINKER
Director



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FOR IMMEDIATE RELEASE:

September 18, 1986

**HANFORD HISTORICAL DOCUMENTS REVIEW COMMITTEE
NAMES PEER REVIEW PANEL**

Olympia, WA -- Dr. Royston Filby, Chair of the Hanford Historical Documents Review Committee, (HHDRC), announced the members of the Peer Review Panel today. The Panel represents national expertise in radiochemistry, the nuclear fuel cycle, health physics, radioecology, nuclear waste management, nuclear engineering and epidemiology.

The Hanford Historical Document Review Committee is composed of representatives from the states of Washington and Oregon, the Yakima Nation, Nez Perce Tribe and the Confederated Tribes of the Umatilla Indian Reservation. The HHDRC will be responsible for the evaluation of environmental impacts associated with releases from Hanford operations. These releases are described in part in the 19,000 pages of information recently declassified by the U.S. Department of Energy. The Peer Review Panel's duty will be to assist the HHDRC in its evaluation process.

The Peer Review Panel members include: Dr. W.E. Kreger, Washington; Dr. John A.H. Lee, Washington; Dr. Edward S. Macias, Missouri; Dr. John Poston, Texas; Dr. Allan H. Seymour, Washington; Mr. Robert D. Siek, Colorado; and Dr. C.H. Wang, Oregon. Information on panel members is attached.

#

MEMBERS OF THE PEER REVIEW PANEL

W.E. Kreger, Ph.D., Consultant, U.S. Nuclear Regulatory Commission. Dr. Kreger has worked for several federal radiation agencies including the U.S. NRC as Assistant Director for Radiation Protection, and the U.S. Naval Radiological Defense Laboratory as Head of the Physical Sciences Division. He has participated in panels and committees for the International Atomic Energy Agency, National Academy of Sciences and the National Council on Radiation Protection and Measurements.

John A.H. Lee, M.D., Professor of Epidemiology and International Health, University of Washington. Principal investigator for a consultant agreement to develop the technology for a statistical health surveillance system dealing with impacts to Hanford workers. Dr. Lee has served as a consultant to the World Health Organization, U.S. Department of Health, Education and Welfare, and National Academy of Sciences.

Edward S. Macias, Ph.D., Professor and Chairman of Chemistry, Washington University. Dr. Macias is involved with research on the chemistry and physics of atmospheric aerosols. He has also served as Chair of the National Academy of Sciences, National Research Council, Committee on Nuclear and Radiochemistry.

J.W. Poston, Ph.D., Professor, Department of Nuclear Engineering, Texas A and M University. President, Health Physics Society, 1986-1987. Dr. Poston has been involved with many groups involved with radiation protection, including the International Commission on Radiological Protection, National Council on Radiation Protection and Measurement, and the International Atomic Energy Agency.

Allan H. Seymour, Ph.D., Professor Emeritus, College of Fisheries, University of Washington. Dr. Seymour is a radioecologist who has conducted extensive research on radiation in the aquatic environments. He has been associated with the Nuclear Waste Regulatory Commission, International Atomic Energy Agency, Smithsonian Institution and National Academy of Sciences.

Robert D. Siek, M.P.H., Chief Technical Advisor, Council of Energy Resource Tribes. Mr. Siek has directed the Division of Radiation and Hazardous Materials and the Environmental Protection Programs for the state of Colorado. Mr. Siek has also served as Chairman of the National Conference of Radiation Control Program Directors.

C.H. Wang, Ph.D., Professor Emeritus, Department of Nuclear Engineering, Oregon State University. Dr. Wang has served as Director of the Radiation Center at Oregon State University and as Head of the Department of Nuclear Engineering. He has also served as a consultant to the National Science Foundation and the National Institute of Health.

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A note on "Job related mortality risks of Hanford workers and their relation to cancer effects of measured doses of external radiation"

ETHEL S GILBERT AND G R PETERSEN



British Medical Association, Tavistock Square, London WC1H 9JR



Short report

A note on "Job related mortality risks of Hanford workers and their relation to cancer effects of measured doses of external radiation"

ETHEL S GILBERT¹ AND G R PETERSEN²

From the Pacific Northwest Laboratory¹ and the Hanford Environmental Health Foundation,² Richland, Washington 99352, USA

Although it is not our intention to provide a complete critique of the latest analyses of the Hanford data by Kneale *et al*,¹ we wish to comment briefly and to present some results of our own analyses of the Hanford data for comparison. In an earlier paper of Kneale *et al*² internal monitoring levels played a key part as a control variable. In the latest paper¹ this variable has been replaced with a measure of job hazard developed by the same authors in a companion paper.³ We will not comment on the construction of this variable, but we do wish to note that the results of analyses using this new variable clearly differ from those in the earlier paper. The *t* value for the analysis of cancers of radiosensitive tissues (A-series) with control for job hazard and other "obvious" factors based on untransformed doses and no allowance for cancer latency or exposure age (table 9¹) is 0.77. This may be compared with a *t* value of 2.47 for a comparable analysis presented in the earlier paper (using internal monitoring levels instead of job hazard). Kneale *et al* do not comment on the implications of this change, but it is clear that the strength of the association of cancer mortality and exposure to radiation has been weakened with the use of the job hazard variable and the inclusion of recent deaths. No estimates of radiation risks are offered in the most recent paper, but they would probably differ substantially from those presented earlier.

Kneale *et al* obtain statistically significant results only by analysing ranks instead of doses, or by allowing for an age effect as well as by restricting the analyses to a subgroup of cancers identified as cancers of radiosensitive tissues (A-series). The use of

ranks (which in this application is similar to analysing the logarithms of doses) involves searching for the measure of dose that correlates best with A-series cancer mortality. Since there is no a priori reason (based on data from populations other than Hanford workers) to think that the dose response curve will be logarithmic, results of the *t* test presented must be interpreted with caution. In fact, if a parameter describing the shape of the dose response curve had been estimated by maximising the log likelihood an additional degree of freedom would be needed in assessing the statistical significance of the result—that is, a higher value than those attained would be required to achieve statistical significance.

With regard to the age at exposure adjustment, studies of the Japanese A-bomb survivors⁴ and other populations⁵ suggest that relative risks decrease rather than increase with increasing age at exposure. Since there is no a priori justification for the adjustment applied by Kneale *et al* in table 11,¹ it is again inappropriate to evaluate statistical significance using a simple *t* test. There is, however, an additional problem with the adjustment for age. Kneale *et al* have controlled for age by using fairly broad age at hire groups (<25, 25-34, 35-44, 45-54, and >55). The age at exposure adjustment consists of "increasing dose by 10% for each year after age 40" with the result that those at the upper end of the 10 year age intervals have their doses increased by more than those at the lower end. Since spontaneous rates for cancer increase appreciably with age, those at the upper ends of intervals also have higher death rates than those at the lower end. The result is a spurious correlation of cancer mortality and exposure that accounts at least in part for the increased correlation produced by the adjustment for age at exposure.

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We present here some results of our own analyses of the Hanford data. Our data base is similar to that analysed by Kneale *et al*, but there are some differences, especially for mortality data in recent years when our ascertainment of deaths and certificates has been conducted independently of Kneale *et al*. Ascertainment of deaths for our analyses is based on information provided by the Social Security Administration (up to September 1983), the US National Death Index, the Washington State Occupational Mortality Surveillance System, and the California (State) Automated Mortality Linkage System. Cause of death coding for all deaths was validated by the US National Center for Health Statistics staff in 1983.

The fact that the two data sets are reasonably comparable is shown in table 1. Because we do not yet have certificates for over 20% of the deaths occurring in 1979 and 1980 (fewer than 2% of the certificates are missing for deaths occurring earlier), we have presented deaths both up to 1 January 1979 and up to 1 January 1981 (only deaths with certificates are presented). Kneale *et al* indicate that their analyses include deaths up to March 1980; we are given no information on the completeness of their mortality data, or the procedures used to obtain it.

Because of the possibility of bias resulting from incomplete ascertainment (those deaths without certificates show evidence of a negative correlation with radiation exposure), the analyses presented in table 2 include only deaths up to 1 January 1979. The general method used in conducting these analyses is similar to that used by Kneale *et al*, and has been described by Gilbert⁶ and by Gilbert and Marks.⁷ Our analyses were controlled for single years of age (age plays the part of follow up period in the Kneale *et al* analyses), calendar year (five year intervals except for 1975-8 when single year intervals were used to account for possible delays in mortality ascertainment), sex, and duration of employment (less than two years, and two or more years). Exposures were lagged for a 10 year period as in the results presented in tables 10 and 11 of Kneale *et al*. The A-series cancer group is that specified in Kneale *et al*⁶—namely, ICD codes 146-159, 162-163, 174, 193, 200-209. We have analysed the A- and B-series separately for the purpose of comparing results, not because we believe that this represents an appropriate grouping. The trend test statistics may be compared with a standard normal distribution to assess statistical significance.

There is no evidence of a negative correlation with exposure for all causes of death, although a negative correlation was found (trend test statistic = 1.77) when the data were analysed with no allow-

Table 1 Number of certified deaths by exposure category at death. (Number of deaths due to cancer given in parentheses)

Dose (rem)	Kneale <i>et al</i>	Our files to 1 January 1979	Our files to 1 January 1981
0-00	1060 (208)	998 (209)	1068 (229)
0-01-	555 (109)	522 (107)	565 (115)
0-08-	869 (164)	819 (154)	878 (174)
0-32-	647 (146)	600 (135)	671 (156)
0-64-	689 (147)	652 (143)	711 (158)
1-28-	530 (115)	491 (109)	551 (121)
2-56-	231 (60)	218 (55)	243 (64)
5-12-	150 (35)	143 (32)	164 (40)
10-24-	96 (23)	92 (22)	107 (26)
>20-48	112 (26)	100 (25)	126 (30)

ance for latency (comparable with the analyses presented in table 6 of Kneale *et al*⁶). At least a part of the explanation for the negative correlation observed with no allowance for latency is that in many cases workers who die will have been ill for some period preceding their deaths, and thus will not be reporting for work and having their dosimeters read. This source of bias will be reduced when the last 10 years of dose is discarded as in the analyses in table 2. The fact that our control variables differ from the "obvious" factors used by Kneale *et al* may also be a factor in the reduction of the negative correlation reported by these investigators. Although the possibility of exposure related bias can never be ruled out in an epidemiological study, there is no strong evidence of such bias in our data for either cancer or all cause mortality.

Table 2 Observed and expected deaths for all certified deaths, A-series ("radiosensitive") cancers, and B-series cancers with allowance for 10-year latency

Dose (rem)	All certified deaths	A-series cancers	B-series cancers
	Obs/Exp*	Obs/Exp*	Obs/Exp*
0-00	870/835.5	123/125.0	63/49.2
0-01-	499/493.4	79/75.5	21/29.1
0-08-	783/777.0	117/119.1	43/45.8
0-32-	617/593.7	94/89.1	29/35.3
0-64-	533/570.2	84/87.9	38/35.0
1-28-	307/331.0	48/55.6	23/20.5
2-56-	142/149.3	31/25.7	9/9.4
5-12-	93/91.9	16/16.4	5/5.9
10-24-	65/65.7	15/11.4	4/4.4
20-48-	33/34.2	5/6.2	2/2.4
Trend test statistic	-0.67	0.28	-0.43
Probability of trend arising due to chance†	0.75	0.39	0.67

*Expected deaths are calculated from experience of all monitored workers (including survivors) allowing for age, calendar year, sex, and duration of employment.

†Significance levels are for a one tailed test and are calculated using a normal approximation.

The results in table 2 show little evidence of a positive correlation of A-series cancers with radiation exposure. Repeat analyses with ranks (trend test statistic = 0.16) and with the 1979-80 deaths included (trend test statistic = -0.07) also failed to yield significant correlations. There is no evidence of a significant negative correlation for the B-series cancers.

Because of the limited power of statistical tests based on data on Hanford workers, the possibility of radiation induced cancer in the Hanford population cannot be ruled out. Nevertheless, in view of the results presented in the latest paper of Kneale *et al* and our own analyses there does not seem to be any compelling reason to believe that the Hanford data are inconsistent with current estimates of radiation risks.

Work supported by the US Department of Energy under contract DE-AC06-76RLO 1830.

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- ⁷ Gilbert ES, Marks S. An analysis of the mortality of workers in a nuclear facility. *Radiat Res* 1979;79:122-48.

ACUTE LEUKEMIA IN AN ISOLATED WESTERN COMMUNITY

By

S. K. McIlvanie, M.D. and William A. Dittman, M.D.

From the Department of Medicine, Rockwood Clinic, Spokane, Washington.

Acute Leukemia in an Isolated Western Community

S. K. McIlvanie, M.D. and William A. Dittman, M.D.

2.

Our attention was attracted to the Lewiston-Clarkston area in 1957 when one of us (SKM) saw four cases of acute leukemia from that community. Correspondence with United States Public Health Service officials at that time indicated no similar reported pattern elsewhere. Since then considerable interest in the geographic distribution of leukemia has developed,¹⁻³ and particular attention has been paid to "leukemia clusters".^{4, 5}

It is our purpose to present data on such a cluster and to comment on some of the epidemiological aspects previously explored.⁶⁻¹⁴

Results

Description of Communities.--The twin towns, Lewiston, Idaho, and Clarkston, Washington, had a population of 22,371 and 6,209 respectively (1960), and are separated by the Snake River. The Lewiston population includes a suburban subdivision of Lewiston Orchards with a 9,680 population. The elevation is approximately 738 feet above sea level. The towns are in a deep valley, bounded on the north by the high southern plateau of Washington with an abrupt rise to approximately 2,000 feet, and on the south by the Blue Mountains, and the breaks of the Snake and Clearwater Rivers. There are no large adjacent population centers.

Lewiston, Idaho is located on the banks of the Snake and Clearwater Rivers. It has a modern sewage disposal system. The

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Methods

Data was collected from direct referrals, a systemic survey, by letter, of all the doctors in the region, and by an analysis of vital statistics provided by the Idaho Department of Vital Statistics and the Washington Department of Vital Statistics. A field survey was carried out during the summer of 1962 by a senior medical student. Detailed questionnaires were filled out with help from patients and their families. Doctors concerned were interviewed. Soil and water samples were collected, where possible, from the home or immediately adjacent to the home of each patient. Populations of the areas involved, i.e., Lewiston Orchards, Lewiston, Idaho, and Clarkston, Washington, were obtained from the 1950 and 1960 census. It should be noted that the population of Lewiston and Clarkston were relatively stable, and that the population of Lewiston Orchards did increase during this interval. (Fig 1) The estimated number of children fifteen years of age and under was obtained with the cooperation of the principals of the public schools in the three communities. The birth rate in Nez Perce County, of which Lewiston is the county seat, was relatively constant throughout the twelve-year period averaging about 650 per year with the exception of 1954 and 1955 when it reached 724 and 766 respectively.

Distribution of Cases.--Figure 2 demonstrates a clustering of the cases in Lewiston Orchards and the Clarkston area as

Acute Leukemia in an Isolated Western Community

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of 62 cases shown in 1958. Public Health officials commented on the apparent large number of cases of hepatitis in the Lewiston Orchards area in the last half of the decade, beginning in 1957. Large amounts of gamma globulin were given during this time.

Results of Questionnaires.--Sixteen of the 24 questionnaires were completed satisfactorily. Analysis of this series shows that 16 of the 24 cases were childhood leukemia. These were classified as 14 lymphoblastic and 2 myeloblastic. The sex ratio was 1:1. (Table I) Seven of these patients had had either prenatal or neonatal irradiation exposure, usually only diagnostic in form of pelvimetry or chest x-rays. There was no discernible predisposing illness or prenatal illness in the mothers. Animal or pet exposure was almost universal in this group.

A family history of leukemia was obtained in two of the 16 cases. In one, a child of 14 months, the mother's maternal grandfather died of leukemia. In one adult patient, a nephew died of leukemia. There was no other family history elicited.

Two specific histories of possible contact or exposure between patients were noted. In one, a young child accompanied his mother to the meat market where a 19-year-old boy with acute lymphoblastic leukemia worked. The 19-year-old patient's disease was diagnosed on September 16, 1959. The child's disease was diagnosed on December 31, 1959. There was no other specific contact besides this association. In the other instance three small children were involved. Child one (not included in series because of onset in 1963) visited child two who in turn took a present to child number three.

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States. If one divided the total population of the United States into 6,878 communities of 28,000 population, our peak incidence of six cases/year/28,000 population would occur in only 11 communities in the same year. Unlike the reports of Heath,⁴ Hayes,¹⁰ and Lee,¹¹ we along with Bjelke,¹² were unable to detect a seasonal variation. The latter noted a bimodal yearly distribution.

The very large number of cases of infectious hepatitis occurring in the area, particularly when viewed in the light of the known problem of drainage and septic tank sewage disposal in the Lewiston Orchards area, raises the question: Is there a common route of transmission of an agent in the case of acute leukemia and hepatitis?

The high incidence of hepatitis suggests epidemiological factors that were optimum for spread of an enterovirus. Both enteroviruses and adenoviruses are known to be ubiquitous and are harbored in the gastrointestinal tract and nasopharynx. The possibility is suggested that exposure to a similar viral agent of leukemia could have been widespread yet, as in poliomyelitis, only a few patients, because of hereditary and environmental factors, developed overt disease. Types 12 and 18 adenovirus taken from human subjects^{13, 14} are now shown to produce tumors in hamsters adding further credence to this hypothesis. We were unable to note any relationship between other factors in the community. Specifically, there is a large paper mill located on the east side of the community on the Clearwater River. The smoke

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The assistance of Dr. Thomas S. Russell and Dr. Thomas P. Bogyo, Assistant Professor of Agriculture, and Assistant Statistician, Statistical Services, Washington State University, Pullman, Washington, who did the statistical analysis, is gratefully acknowledged.

We wish to thank the physicians of Lewiston, Idaho and Clarkston, Washington for their generous cooperation and interest, and the Public Health and School Officials for their help.

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Table I

Age At Diagnosis	Sex		Type		Total
	M	F	Myeloblastic	Lymphoblastic	
0 - 1		1	1		1
1		2		2	2
2	1	1		2	2
3	1			1	1
4	2			2	2
5 - 9	2	4		6	6
10 - 14	2			2	2
15 - 19	1		1		1
20 - 24		1	1		1
> 24	4	2	2	4	6
Totals	13	11	5	19	24

Acute Leukemia in an Isolated Western Community

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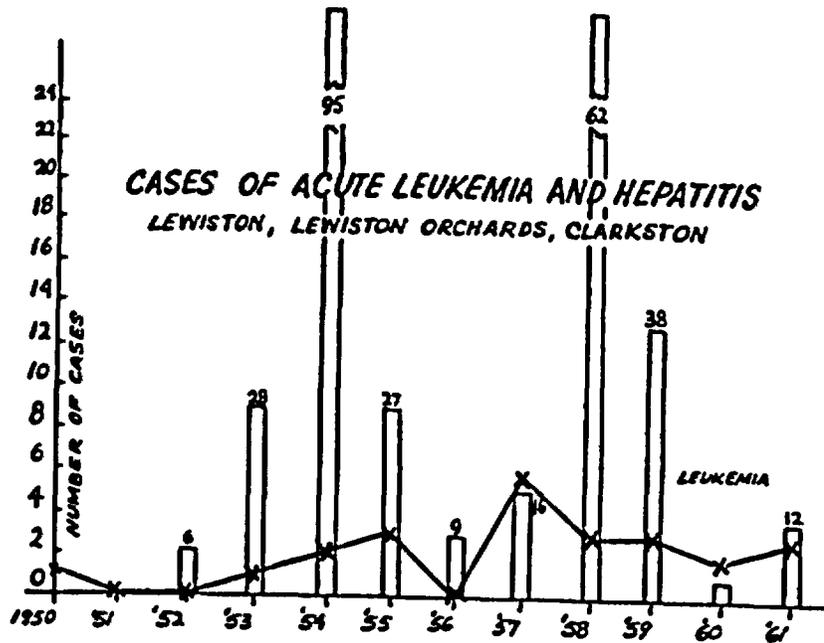


Fig 3.

Fig 3. Incidence graph of acute leukemia superimposed on the incidence of hepatitis as obtained from Public Health Department of Idaho 1950 - 1961.

Acute Leukemia in an Isolated Western Community

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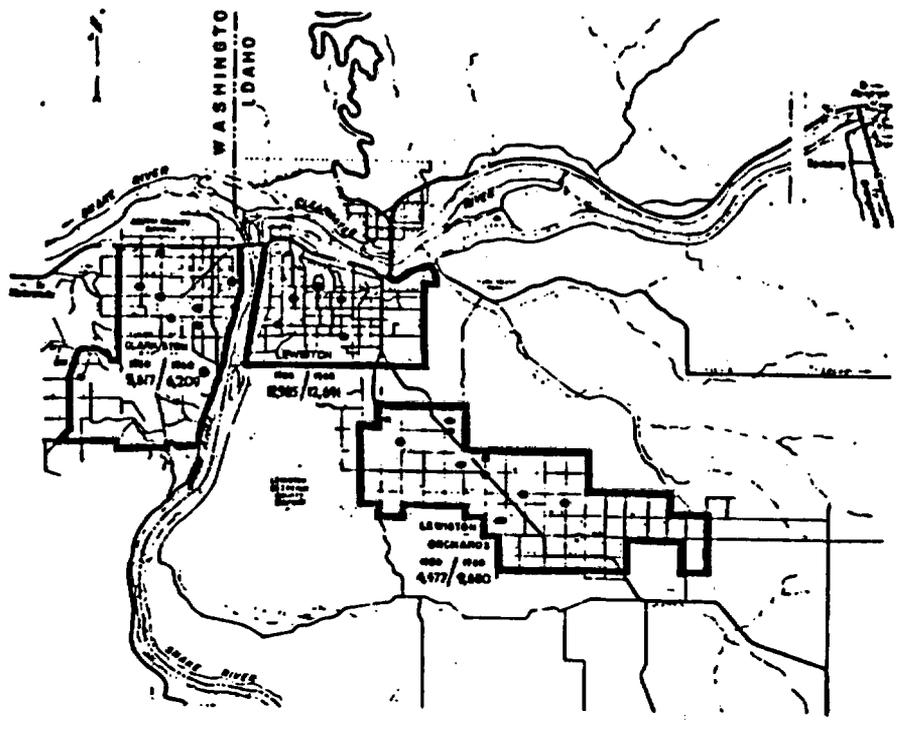


Fig 2.

Fig 2. Acute leukemia in Lewiston, Lewiston Orchards, Idaho, and Clarkston, Washington 1950-1961. The solid dots indicate the cases of acute leukemia.