Epidemiology of Hairy Cell Leukemia in Los Angeles County

Leslie Bernstein, Patricia Newton and Ronald K. Ross

Cancer Res 1990;50:3605-3609.

[CANCER RESEARCH 50, 3605–3609, June 15, 1990]

Epidemiology of Hairy Cell Leukemia in Los Angeles County[1]

Leslie Bernstein,[2] Patricia Newton, and Ronald K. Ross

Department of Preventive Medicine, University of Southern California School of Medicine, Los Angeles, California 90033

ABSTRACT

The descriptive epidemiological characteristics of hairy cell leukemia (HCL), a rare chronic lymphoproliferative disorder, were examined by using incidence data collected from 1972 to 1987 by the Cancer Surveillance Program, the population-based cancer registry for Los Angeles County. During the study period, 208 incident cases of histologically confirmed HCL were diagnosed. HCL comprised 2% of all leukemias diagnosed in Los Angeles County during the study period. HCL risk was concentrated in white males; there were few black and Asian patients for analysis. Overall, the age-adjusted incidence rate of HCL for men (2.9/million population) was 4.8 times greater than that for women (0.6/million population). Using data from all cancer patients diagnosed during the study period, Jewish men had significantly greater risk of HCL than Protestant men (odds ratio (OR) = 3.0, $P < 0.0001$); there was no significant variation in risk of HCL by religion for women. For men, the OR was significantly elevated for professional and technical workers (OR = 2.1, $P = 0.001$); within this category of occupations, risk was significantly elevated for engineers (OR = 3.3, $P < 0.0001$) and university faculty and school teachers (OR = 4.0, $P = 0.0008$). HCL patients were more than twice as likely to have multiple primary cancer diagnoses as other cancer patients. Since the majority of the other primary cancer diagnoses occurred prior to (>1 year) or concurrent with (≤1 year) the HCL diagnosis, this greater frequency of multiple primaries in HCL patients may be due to impaired immune function.

INTRODUCTION

HCL[3] is a rare, chronic B-cell lymphoproliferative malignancy that is reported to comprise 2% of all leukemias (1). It was first described as a distinct entity in 1958 by Bouroncle *et al.* (2). Variants of HCL with T-cell features and both T-cell and B-cell features have also been described (3, 4) and the human lymphotropic virus HTLV-II has been detected in several patients with the T-cell phenotype (5, 6). Patients with HCL typically present with nonspecific complaints of fatigue or weakness. Pancytopenia and splenomegaly are present in the majority of patients; lymphadenopathy and hepatomegaly are uncommon (7). HCL is morphologically characterized as a circulating leukemic cell with cytoplasmic projections ("hairy cells") which express the tartrate-resistant isoenzyme of acid phosphatase (8).

Our interest in the descriptive epidemiology of HCL was prompted by our recent study of AIDS-related cancer trends in Los Angeles County men in which we examined cancer risk by marital status under the assumption that the category "never-married" men would include the majority of homosexual men (9). In that study, we noted a statistically significant increase in the secular trend of "other leukemias" (which excludes acute lymphocytic, acute myelogenous, chronic lymphocytic, and chronic myelogenous leukemia) for never-married men. Although only two such cases had been diagnosed prior to 1983 in never-married men, during the interval 1983 through 1985, seven cases of other leukemias were diagnosed, of which five were HCL. No similar increasing trend was apparent for ever-married men.

There are few data on the epidemiological characteristics of HCL as no population-based studies of HCL have been conducted. Based on several case series, the disease is diagnosed most frequently in males, with the male:female case ratio ranging from 2.7 to 5.0; the median age at diagnosis is typically the mid-fifties with patients ranging in age from 20 through 85 (10–14). Here, we describe the incidence of HCL in the population of Los Angeles County from 1972 through 1987.

MATERIALS AND METHODS

The Los Angeles County/University of Southern California Cancer Surveillance Program is a population-based cancer registry that identifies all newly diagnosed cancer cases occurring among the more than 8.5 million residents of Los Angeles County (15). Only patients with basal cell and squamous cell carcinomas of the skin are not included in the registry data base. Since 1972, well over 95% of the patients diagnosed with incident cancer of all other sites and histologies who are residents of Los Angeles County have been identified. A detailed description of the methodology, organization, and administration of the CSP has been published elsewhere (16). As of May 1987, the CSP became part of the statewide California Tumor Registry, and the methodology for case ascertainment was modified somewhat.

Cancer patients are identified from hospital, clinic, and pathology records as well as from death certificates. For each cancer patient, address, date of birth, race, sex, religion, marital status, and other pertinent data are abstracted from medical records. The CSP divides whites into "Spanish surnamed" and "non-Spanish surnamed" white categories by means of the detailed Spanish surname list prepared by the United States Bureau of the Census. Allocation of race to patients of unknown race not bearing a Spanish surname is done in a manner corresponding to the method used by the United States Bureau of the Census (17). No such allocation was required for any HCL patient in this study. Occupation at diagnosis is coded into one of 417 occupational codes using the 1970 United States Bureau of the Census classification system (18). For this study, patients with known religious preference have been broadly classified into six categories: Protestant, Catholic, other Christian (includes Mormons, Seventh Day Adventists, Jehovah's Witnesses, and various Eastern Orthodox denominations), Jewish, a combined category of other religious preferences, and none.

Within the CSP, cancers are coded by ICDO topographical and morphological (M) codes (19). Data presented here are for HCL (ICDO M-9940), acute lymphocytic leukemia (ICDO M-9821, M-9822), acute nonlymphocytic leukemia (ICDO M-9840, M-9860 to M-9862, M-9865, M-9866, M-9870, M-9880, M-9890 to M-9892, M9894), chronic myelogenous leukemia (ICDO M-9863, M-9893), chronic lymphocytic leukemia (ICDO M-9820, M-9823, M-9825), and other leukemias (ICDO M-9800 to M-9804, M-9810, M-9824, M-9830, M-9841, M-9842, M-9850, M-9864, M-9900, M-9910, M-9920, and M-9930).

Multiple primary cancer diagnoses for an individual are identified by a combination of computerized record linkage and manual screening methods. Multiple hospital reports of the same tumor and reports of metastases are designated as such as are multiple interpretations of

Received 10/12/89; revised 3/6/90.

The costs of publication of this article were defrayed in part by the payment of page charges. This article must therefore be hereby marked *advertisement* in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

[1] This work was supported by Grant CA17054 from the NIH and by Subcontract 050E-8709 with the California Public Health Foundation which is supported by the California Department of Health Services as part of its statewide cancer reporting program, mandated by Health and Safety Code Section 210 and 211.3. The ideas and opinions expressed herein are those of the author, and no endorsement of the State of California, Department of Health Services, or the California Public Health Foundation is intended or should be inferred.

[2] To whom requests for reprints should be addressed, at Department of Preventive Medicine, USC School of Medicine, 1420 San Pablo St., PMB A-202, Los Angeles, CA 90033.

[3] The abbreviations used are: HCL, hairy cell leukemia; AIDS, acquired immune deficiency syndrome; CSP, Cancer Surveillance Program; ICDO, International Classification of Diseases for Oncology; OR, odds ratio.

morphology from the same tumor. Only primary tumors are considered as analytic cases by the CSP and they are identified as such and are sequenced by date of diagnosis. Each CSP report for a particular person has a unique identification number as well as a cross-reference number which links all reports for an individual so that multiple primary cancer diagnoses are readily identified. For the purposes of this report, only multiple primary diagnoses occurring during the study period, 1972–1987, are considered.

For the estimation of incidence rates, we have developed a population-at-risk model which is based on the 1970 and 1980 United States censuses of population (20, 21). To classify the 1980 white population into Spanish surnamed whites and non-Spanish surnamed whites, data on the relationship of Spanish surname to the designation of Spanish origin from the 1980 Public Use Microdata Sample (5% sample) for Los Angeles County were used (22). Individuals of Spanish origin who were designated either "whites" or "other races" were allocated to the Spanish surnamed white ethnicity group on an age-specific, sex-specific percentage basis. Year-specific population estimates were obtained individually by racial/ethnic group, 5-year age group, and sex. Intercensual estimates were obtained by interpolation assuming a constant rate of growth (decline). For the postcensual period, estimates were obtained by extrapolation assuming the same rate of growth (decline). Age-adjusted incidence rates per 10^6 population were calculated by direct standardization, using 5-year age groups with weights derived from the 1970 United States population (20). For the analysis of secular trends, cases of HCL were classified by year of diagnosis (1972–1975, 1976–1978, 1979–1981, 1982–1984, and 1985–1987) and sex.

Because census data are not available for religion and because they cannot be used reliably to produce population denominator figures by occupation, religion-specific and occupation-specific estimates of risk were determined by comparing the number of patients with HCl in a particular religion or occupation category to the total number of patients with other types of cancer in that particular category on an age-specific basis (using 5-year age groupings). This approach is essentially a case-control study using cancer patients with diagnoses other than HCL as controls. This design is similar to that of a hospital-based case-control study where controls are patients with diagnoses thought not to be associated with the risk factors under study. Since we had no prior hypotheses regarding occupational or religious groups at high risk of HCL, all other cancer diagnoses were included as controls.

Age-adjusted ORs were estimated by using the Mantel Haenszel method and 95% confidence limits were calculated by using the Cornfield method (23). To evaluate proportional risk by religion, Protestants were used (arbitrarily) as the baseline comparison group. For risk estimates by occupation, the base-line comparison group was comprised of all patients in the remaining occupation categories. All P values reported are two sided.

RESULTS

Average annual age-adjusted incidence rates of HCL for men and women residing in Los Angeles County are presented in Table 1 for the years 1972 through 1987. Overall, 208 incident cases of histologically confirmed HCL were identified during the study period. HCL risk is concentrated in white males. There are few black or Asian cases and incidence rates for males overall are nearly 5 times greater than those for females.

Incidence rates for other major categories of leukemia in men and women aged 20 and older are provided in Table 1 as a point of reference for the HCL rates. The pattern of occurrence of HCL by sex and race/ethnicity does not resemble the patterns of these more common leukemias. For all races and ethnicities combined, the male:female rate ratios range from 1.5 for acute nonlymphocytic and acute lymphocytic leukemia to 1.9 for chronic lymphocytic leukemia. For whites and blacks, no male:female rate ratio is greater than 1.9.

During the study period, there were 10,605 persons who were diagnosed with some form of leukemia in Los Angeles County. Overall, HCL comprised 2.0% of these diagnoses. For adult males (aged 20 and older), 3.9% (166 of 4303) of all leukemias were HCL, whereas, for adult women, the figure was 1.2% (42 of 3556).

Male and female age-specific incidence rates of HCL for all racial/ethnic groups combined are shown in Fig. 1. A male excess is present in all age categories except the 20- to 24-year-old age group. Average annual age-adjusted incidence rates by calendar period of diagnosis and sex are shown in Table 2. The rates prior to 1976 are lower than later rates. After 1976, there is no trend in rates for either males or females. Over all time periods, the male:female excess has remained constant.

Overall, 91.6% (152 of 166) of male HCL patients had known marital status. Of these, 12 had never been married. For all other male cancer patients aged 20 and older, marital status was recorded for 95.1%. Never-married men had lower risk of HCL than ever-married men; however, this result was not statistically significant (OR = 0.6, $P = 0.11$).

Because of the few blacks or Asians with HCL, assessment of risk by religious preference was restricted to white patients. Data on religion were available for 73.4% of white males and 78.1% of white females diagnosed with cancer during the time period of interest and for 75.3% (119 of 158) of white males and 90.0% (36 of 40) of white females with HCL (Table 3). Based on all cancers diagnosed in Los Angeles County white men with known religious preference during the years 1972–1987, Jewish men had three times greater risk of HCL than did Protestant men ($P < 0.0001$). For men with other religious preferences, the risks were not appreciably different from that of Protestant men. For white women, the risk of HCL was similar in all religious preference categories.

Information on occupation at the time of diagnosis was available on 75.9% of white males with HCL (120 of 158, including 11.4% who were retired) and 84.8% of all white males with cancer (including 27.8% who were retired). The ORs for broad census classifications of occupations were estimated relative to all other nonretired men with known occupation at the time of diagnosis (Table 4). The OR for the category, professional and technical workers, was significantly elevated (OR = 2.1, $P = 0.001$). No other broad category of occupation was significantly elevated. Within these broad categories, only four subgroups had more than three HCL patients: engineers (including aeronautical, chemical, electrical and electronic, industrial, and mechanical engineers); university faculty and school teachers; writers, artists and entertainers; and mechanics. Significantly elevated ORs were observed for engineers (OR = 3.3, $P < 0.0001$) and university faculty and school teachers (OR = 4.0, $P = 0.0008$); results for mechanics (OR = 2.0, $P = 0.09$) and writers, artists and entertainers (OR = 2.0, $P = 0.53$) were not statistically significant. Similar results were obtained for all occupational categories evaluated when the analyses were restricted to white males aged 20 to 64.

The frequency of other malignancies in HCL patients was examined; 30 (14.4%) of these patients had another primary cancer diagnosis that occurred within the study period compared with 6.7% of all patients diagnosed with cancer during the study period. Women were more likely than men to have another primary cancer diagnosis (21.4 *versus* 12.6%); this contrasts with results for all cancer patients showing men and women equally likely to have multiple primary cancer diagnoses. For HCL patients, the other primary cancer diagnoses included a wide variety of solid tumors; no lymphomas or leukemias were included. The most frequently occurring cancer sites were colon (5 men and 1 woman), breast (4 women), and

Table 1 *Average annual age-adjusted incidence rates of hairy cell leukemia and of other leukemias per million population aged 20 and older, Los Angeles County, 1972–1987, by sex and ethnicity*

Type of leukemia	Racial/ethnic group	Males	Females	Male/female rate ratio
Hairy cell	Whites			
	Non-Spanish surnamed	3.5 (134)[a]	0.8 (36)	4.4
	Spanish surnamed	3.4 (22)	0.4 (4)	8.5
	Blacks	0.7 (4)		
	Asians/other	1.5 (6)	0.3 (2)	5.0
	All	2.9 (166)	0.6 (42)	4.8
Acute non-lymphocytic	Whites			
	Non-Spanish surnamed	31.0 (1140)	20.1 (1037)	1.5
	Spanish surnamed	23.9 (173)	18.3 (159)	1.3
	Blacks	21.3 (99)	20.3 (138)	1.0
	Asians/other	18.2 (69)	10.8 (57)	1.7
	All	28.5 (1481)	19.3 (1391)	1.5
Acute lymphocytic	Whites			
	Non-Spanish surnamed	3.9 (149)	2.6 (126)	1.5
	Spanish surnamed	5.2 (69)	4.3 (49)	1.2
	Blacks	2.3 (14)	1.9 (15)	1.2
	Asians/other	3.6 (18)	0.9 (5)	4.0
	All	4.0 (250)	2.7 (195)	1.5
Chronic myelogenous	Whites			
	Non-Spanish surnamed	14.7 (544)	8.2 (424)	1.8
	Spanish surnamed	18.1 (132)	9.9 (88)	1.8
	Blacks	14.6 (75)	10.7 (74)	1.4
	Asians/other	6.9 (28)	2.9 (16)	2.4
	All	14.5 (779)	8.4 (602)	1.7
Chronic lymphocytic	Whites			
	Non-Spanish surnamed	30.8 (1131)	15.8 (892)	1.9
	Spanish surnamed	13.8 (75)	7.5 (50)	1.8
	Blacks	24.8 (110)	14.4 (90)	1.7
	Asians/other	3.8 (11)	1.0 (4)	3.8
	All	26.8 (1327)	14.0 (1036)	1.9

[a] Numbers in parentheses, number of cases.

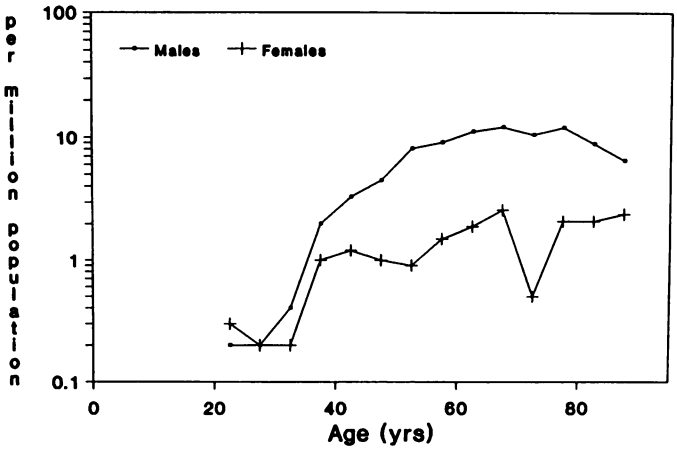

Fig. 1. Age-specific incidence rates of hairy cell leukemia per million population by sex, Los Angeles County, 1972–1987.

Table 2 *Average annual age-adjusted incidence rates of hairy cell leukemia per million population, Los Angeles County, by sex and year of diagnosis, for all racial and ethnic groups combined*

Year category	Males	Females
1972–1975	1.4 (19)[a]	0.3 (5)
1976–1978	3.5 (34)	0.8 (10)
1979–1981	3.0 (30)	0.5 (6)
1982–1984	3.8 (39)	0.7 (10)
1985–1987	3.9 (44)	0.8 (11)

[a] Numbers in parentheses, number of cases.

Table 3 *Age-adjusted OR and 95% confidence limits (CL) for hairy cell leukemia by religion and sex, for whites (non-Spanish surnamed and Spanish surnamed) with stated religious preference*

	Males			Females		
Religion	OR	95% CL	*N*	OR	95% CL	*N*
Protestant	1.0[a]		42	1.0[a]		15
Catholic	1.1	0.7, 1.7	32	1.2	0.6, 2.6	13
Other Christian	1.5	0.5, 4.2	4	0.8	0.1, 5.9	1
Jewish	3.0	1.9, 4.8	30	1.3	0.5, 3.4	5
No religion	1.5	0.8, 2.9	11	0.9	0.2, 4.0	2

[a] Reference group.

Table 4 *Age-adjusted OR and 95% confidence limits (CL) for hairy cell leukemia by occupation at diagnosis, for white males (non-Spanish surnamed and Spanish surnamed) with known occupation*

Occupation	OR[a]	95% CL	*N*
Professional and technical workers	2.1	1.3, 3.0	35
Engineers	3.3	2.1, 5.6	17
University faculty and school teachers	4.0	1.9, 8.3	7
Writers, artists and entertainers	1.5	0.6, 3.7	5
Managers and administrators	0.8	0.5, 1.4	17
Sales workers	0.6	0.2, 1.4	5
Clerical workers	1.4	0.7, 2.9	8
Craftsmen	0.8	0.5, 1.3	18
Mechanics	2.0	1.0, 4.0	8
Operatives, except transportation	0.7	0.3, 1.6	5
Transportation operatives	1.1	0.5, 2.7	5
Laborers, except farm	1.1	0.5, 2.6	5
Service workers, except household	0.5	0.2, 1.4	4

[a] Each occupation category is compared with a reference group of all other occupation categories combined.

prostate (3 men). One male patient was diagnosed with Kaposi's sarcoma after his HCL diagnosis; this patient, who was in his seventies when diagnosed, has previously been reported in the literature (24). Eight (27%) of the other primary malignancies were diagnosed at least 1 year prior to the HCL diagnosis; 12 (40%) were "concurrent" diagnoses occurring within 12 months of the HCL diagnosis; and 10 (33%) were diagnosed at least 1 year after the patient had been diagnosed with HCL.

DISCUSSION

Case reports linking leukemia to the AIDS epidemic have appeared in the literature (25–27). This population-based study of the epidemiological features of HCL was initially motivated by our observation that five never-married men, aged 18 to 54, had been diagnosed with HCL during the period 1983–1985, while no cases of HCL in such men had occurred prior to 1983 (9). Although we found no indication of an association between AIDS and the more frequent types of leukemia in this earlier study, the observed increase in HCL in never-married men suggested a possible link of this cancer with the AIDS epidemic. Since 1985, however, no further cases of HCL in never-married men in this age range have been ascertained by the CSP; moreover, the risk of HCL in never-married men overall appears to be lower than that of ever-married men.

Our population-based data provide the first estimates of the incidence of HCL in a defined population. Our data confirm the descriptive characteristics observed in case-series reports in the literature: a marked excess of HCL in males, median age of diagnosis in the mid-fifties, and a 2% relative incidence in comparison with other types of leukemia. There is no evidence of a secular increase in HCL over the time period studied. The initial increase in incidence rates which is evident after 1975 likely reflects a lack of recognition of HCL by pathologists in the early 1970s.

Compared to Protestant men, Jewish men had significantly greater risk of HCL, but no similar elevation in risk was observed for Jewish women. A number of studies, summarized by Linet (28), have reported elevated risks of all leukemias in Jews compared to non-Jews. Haenszel (29) examined results of several mortality studies in Jewish populations, noting that the male:female ratios of mortality from leukemia appeared to be closer to 1 in Jews than in non-Jews. In an earlier study, we examined patterns of cancer risk by religion in Los Angeles County and found that, compared with the major subgroup of Protestant religions, Jews had significantly greater risk of lymphomas (particularly non-Hodgkin's types) and, although the risk of all leukemias was elevated for Jewish males, the excess was only 20% (30). In this earlier study, we noted that Jewish men had lower risks of lung, larynx, esophageal, oral, prostate, and rectal cancer than the Protestant comparison group. When we eliminated patients with these cancers from the control group, the risk of HCL in Jewish men remained significantly elevated (OR = 2.4, $P = 0.0003$).

Our results showing a significantly elevated risk for professional and technical workers may represent a social class effect since risk was elevated for three different groups within this broad classification. It is unlikely that engineers, university professors and school teachers, and writers, artists, and entertainers would share similar occupational exposures. However, the significantly increased risk for engineers and the number affected (17 or 16.7% of those with known occupation) may reflect some occupationally related etiological exposure. In fact, three of the five cases of HCL diagnosed in never-married young men (aged 20–54) during the period 1983–1985 were engineers.

Several exposures have been proposed in the literature as possible etiological factors for HCL. Oleske *et al.* (31) compared histories of cases from a HCL treatment center with those of population-based controls, and found significantly elevated risks associated with farm birthplace, history of anemia, migraine headaches and infectious mononucleosis, routine use of aspirin, and exposure to organic chemicals in the workplace. Marginal associations were observed with farming and woodworking occupations. Chronic benzene exposure has been anecdotally linked with HCL (32, 33). Stewart and Keating (34) reported a greater frequency of prior occupational, accidental, or therapeutic radiation exposure in HCL patients than in patients with solid tumors.

HCL patients appear to have a greater risk of multiple primary cancers than other cancer patients. This may reflect both lower median age than cancer patients overall (mid-fifties *versus* late sixties) or the greater likelihood of survival. However, neither of these explanations would account for the large percentage of other malignancies that were diagnosed prior to (27%) or concurrent with (40%) the HCL diagnosis in Los Angeles County patients. A more plausible explanation is that patients who develop HCL may have impaired immune function. There have been reports in the literature of decreased natural killer cell activity and defective T-cell activity in HCL (35, 36).

There are many case reports in the literature documenting instances of multiple primary cancer diagnoses in HCL patients. The rate of multiple primary cancers in the large case series of 172 HCL patients being followed at the University of Chicago was 8.7% after 10 years of follow-up with all but one of the second malignancies diagnosed concurrently with or after the HCL diagnosis (37).

Because HCL and chronic lymphocytic leukemia, both chronic lymphoproliferative malignancies primarily of B-cell origin, have similar clinical presentations, one might expect similar patterns of incidence. Neither disease occurs in the young. However, the incidence rate of chronic lymphocytic leukemia rapidly rises in late middle age and continues to increase with age, whereas the incidence rate of HCL appears to plateau after late middle age. In addition, the incidence rate of chronic lymphocytic leukemia are similar in blacks and whites, and the overall male:female rate ratio is less than 2. In fact, none of the four major types of leukemia show the unique racial/ethnic pattern of HCL or the extreme male:female rate ratio observed for HCL.

Although the age-specific and race-specific incidence patterns of classic Kaposi's sarcoma (*i.e.*, Kaposi's sarcoma that is not related to the AIDS epidemic) differ from those of HCL, there are some striking similarities between the two diseases (38). In Los Angeles County, Jewish men of European (especially Eastern and Southern European) origin have excess risk of classic Kaposi's sarcoma (38). Furthermore, the male:female rate ratio for classic Kaposi's sarcoma is greater than 3 for Spanish-surnamed whites, non-Spanish surnamed whites, and blacks. Kaposi's sarcoma patients also appear to have increased risk of second primary malignancies, although in contrast to HCL, the majority of these cancers are lymphoreticular neoplasms (39, 40).

Further studies in other geographic locations are necessary to confirm the epidemiological features of HCL observed here. Although difficult to conduct due to the rarity of HCL, analytic studies will be required to explore the reasons for the marked

excess of HCL in males and among Jews, in particular, and to identify possible nondemographic risk factors for the disease.

REFERENCES

1. Bouroncle, B. A. Leukemic reticuloendotheliosis. Blood, *53:* 412–436, 1979.
2. Bouroncle, B. A., Wiseman, G. B. K., and Doan, C. A. Leukemic reticuloendotheliosis. Blood, *13:* 609–630, 1958.
3. Saxon, A., Stevens, R. H., and Golde, D. W. T-Lymphocyte variant of hairy-cell leukemia. Ann. Intern. Med., *88:* 322–326, 1978.
4. Burns, G. F., Worman, C. P., and Cawley, J. C. Fluctuations in the T and B characteristics of two cases of T-cell hairy-cell leukaemia. Clin. Exp. Immunol., *39:* 76–82, 1980.
5. Kalyanaraman, V. S., Sarngadharan, M. G., Robert-Guroff, M., *et al.* A new subtype of human T-cell leukemia virus (HTLV-II) associated with a T-cell variant of hairy cell leukemia. Science (Wash. DC), *218:* 571–573, 1982.
6. Rosenblatt, J. D., Golde, D. W., Wachsman, W., *et al.* A second isolate of HTLV-II associated with atypical hairy cell leukemia. N. Engl. J. Med., *315:* 372–377, 1986.
7. Golomb, H. M., and Vardiman, J. Hairy cell leukemia: diagnosis and management. Cancer (Phila.), *28:* 265–277, 1978.
8. Yam, L. T., Li, C. Y., and Lam, K. W. Tartrate-resistant acid phosphatase isoenzyme in the reticulum cells of leukemic reticuloendotheliosis. N. Engl. J. Med., *284:* 357–360, 1971.
9. Bernstein, L., Levin, D., Menck, H., and Ross, R. K. AIDS-related secular trends in cancer in Los Angeles County men: a comparison by marital status. Cancer Res., *49:* 466–470, 1989.
10. Golomb, H. M., Catovsky, D., and Golde, D. W. Hairy cell leukemia: a clinical review based on 71 cases. Ann. Intern. Med., *89:* 677–683, 1978.
11. Jansen, J., and Hermans, J. Splenectomy in hairy cell leukemia: a retrospective multicenter analysis. Cancer (Phila.), *47:* 2066–2076, 1981.
12. Katayama, I., and Finkel, H. E. Leukemic reticuloendotheliosis: a clinicopathologic study with review of the literature. Am. J. Med., *57:* 115–125, 1974.
13. Westbrook, C. A., Groopman, J. E., and Golde, D. W. Hairy cell leukemia: disease pattern and prognosis. Cancer (Phila.), *54:* 500–506, 1984.
14. Van Norman, A. S., Nagorney, D. M., Martin, J. K., Phyliky, R. L., and Ilstrum, D. M. Splenectomy for hairy cell leukemia. A clinical review of 63 patients. Cancer (Phila.), *57:* 644–648, 1986.
15. Hisserich, J. C., Preston-Martin, S., and Henderson, B. E. An areawide cancer reporting network. Public Health Rep., *90:* 15–17, 1975.
16. Mack, T. M. Cancer surveillance program in Los Angeles County. Natl. Cancer Inst. Monogr., *47:* 99–101, 1977.
17. United States Bureau of the Census. 1970 Census General Coding Procedures—Manual, Attachment J2. Washington, DC: United States Government Printing Office, 1969.
18. United States Bureau of the Census. 1970 Census of Population: Classified Index of Industries and Occupations. Washington, DC: United States Government Printing Office, 1972.
19. World Health Organization. ICD-O: International Classification of Diseases for Oncology. Geneva: WHO, 1976.
20. United States Bureau of the Census. 1970 Census Second Count Summary Tape. Washington, DC: Bureau of the Census, 1972.
21. United States Bureau of the Census. Characteristics of the Population, General Population Characteristics, California, PC80-1-36. Washington, DC: United States Government Printing Office, 1982.
22. United States Bureau of the Census. Census of Population and Housing, 1980: Public-Use Microdata Samples (A Sample: California). Washington, DC: Bureau of the Census, 1983.
23. Breslow, N. E., and Day, N. E. Statistical Methods in Cancer Research. Vol. I, The Analysis of Case-Control Studies. IARC Scientific Publication No. 32. Lyon, France: International Agency for Research on Cancer, 1980.
24. Hocking, W., Lazar, G., Goldsmith, M., and Foreman, S. Kaposi's sarcoma associated with hairy cell leukemia, immune thrombocytopenia and opportunistic infection. Cancer (Phila.), *54:* 110–113, 1984.
25. Arlin, Z. A., Mittelman, A., Gebhard, D., *et al.* Chronic lymphocytic leukemia in a bisexual male. Cancer Invest., *1:* 549–550, 1983.
26. Gill, P. S., Meyer, P. R., Pavlova, Z., and Levine, A. M. B-Cell acute lymphocytic leukemia in adults. Clinical, morphologic, and immunologic findings. J. Clin. Oncol., *4:* 737–743, 1986.
27. Garavelli, P. L. Acute lymphoblastic leukemia, L3 type, in a HIV-positive patient. Haematologica, *73:* 89, 1988.
28. Linet, M. S. The Leukemias: Epidemiologic Aspects, pp. 66–77. New York: Oxford University Press, 1985.
29. Haenszel, W. Cancer mortality among U.S. Jews. Isr. J. Med. Sci., *7:* 1437–1450, 1971.
30. Mack, T. M., Berkel, J., Bernstein, L., and Mack, W. Religion and cancer in Los Angeles County. Natl. Cancer Inst. Monogr., *60:* 235–245, 1985.
31. Oleske, D., Golomb, H. M., Farber, M. D., and Levy, P. S. A case-control inquiry into the etiology of hairy cell leukemia. Am. J. Epidemiol., *121:* 675–683, 1985.
32. Aksoy, M. Chronic lymphoid leukaemia and hairy cell leukemia due to chronic exposure to benzene: report of three cases. Br. J. Haematol., *66:* 209–211, 1987.
33. Ng, J. P., Cumming, R. L. C., and Hogg, R. B. Hairy cell leukaemia due to chronic exposure to benzene. Br. J. Haematol., *67:* 116–121, 1987.
34. Stewart, D. J., and Keating, M. J. Radiation exposure as a possible etiologic factor in hairy cell leukemia (leukemic reticuloendotheliosis). Cancer (Phila.), *46:* 1577–1580, 1980.
35. Mackowiak, P. A., Demian, S. E., Sutker, W. L., *et al.* Infections in hairy cell leukemia: clinical evidence of a pronounced defect in cell-mediated immunity. Am. J. Med., *68:* 718–724, 1980.
36. Roco, L. P., Procopio, A., Maccallini, V., *et al.* Severe deficiency of natural killer activity in the peripheral blood of patients with hairy cell leukemia. Blood, *61:* 1132–1137, 1983.
37. Jacobs, R. H., Vokes, E. E., and Golomb, H. M. Second malignancies in hairy cell leukemia. Cancer (Phila.), *56:* 1462–1467, 1985.
38. Ross, R. K., Casagrande, J. T., Dworsky, R. L., Levine, A., and Mack, T. Kaposi's sarcoma in Los Angeles, California. J. Natl. Cancer Inst., *75:* 1011–1015, 1985.
39. Safai, B., Mike, V., Giraldo, G., *et al.* Association of Kaposi's sarcoma with second primary malignancies: possible etiopathogenic implications. Cancer (Phila.), *45:* 1472–1479, 1980.
40. Albright, T. M., and Santa Cruz, D. J. Kaposi's sarcoma: relationship with hematologic, lymphoid, and thymic neoplasia. Cancer (Phila.), *47:* 963–973, 1981.