



Original Contribution

Body Size, Recreational Physical Activity, and B-Cell Non-Hodgkin Lymphoma Risk Among Women in the California Teachers Study

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Nutritional status and physical activity are known to alter immune function, which may be relevant to lymphomagenesis. The authors examined body size measurements and recreational physical activity in relation to risk of B-cell non-Hodgkin lymphoma (NHL) in the prospective California Teachers Study. Between 1995 and 2007, 574 women were diagnosed with incident B-cell NHL among 121,216 eligible women aged 22–84 years at cohort entry. Multivariable-adjusted relative risks and 95% confidence intervals were estimated by fitting Cox proportional hazards models for all B-cell NHL combined and for the 3 most common subtypes: diffuse large B-cell lymphoma, follicular lymphoma, and B-cell chronic lymphocytic leukemia/small lymphocytic lymphoma. Height was positively associated with risk of all B-cell NHLs (for >1.70 vs. 1.61–1.65 m, relative risk = 1.50, 95% confidence interval: 1.16, 1.96) and chronic lymphocytic leukemia/small lymphocytic lymphoma (relative risk = 1.93, 95% confidence interval: 1.09, 3.41). Weight and body mass index at age 18 years were positive predictors of B-cell NHL risk overall. These findings indicate that greater height, which may reflect genetics, early life immune function, infectious exposures, nutrition, or growth hormone levels, may play a role in NHL etiology. Adiposity at age 18 years may be more relevant to NHL etiology than that in later life.

body mass index; body size; cohort studies; exercise; hip; lymphoma, non-Hodgkin; waist-hip ratio

Abbreviations: CLL, B-cell chronic lymphocytic leukemia; ICD-O-3, *International Classification of Diseases for Oncology*, Third Edition; NHL, non-Hodgkin lymphoma; SLL, small lymphocytic lymphoma.

Non-Hodgkin lymphoma (NHL), an etiologically and clinically heterogeneous group of lymphoid malignancies, is expected to account for 4.5% of new cancer diagnoses and 3.5% of cancer deaths in 2009 (1). Its incidence has increased steadily in the United States and other developed countries since the 1940s, and NHL is now the fifth most common cancer among men and women in the United States. Human immunodeficiency virus (HIV)-associated NHL and changes in detection and diagnosis of lymphoma explain only a small part of the increase in incidence (2). Since 1991, NHL incidence rates have reached a plateau among men, partly because of improved treatment for human immunodeficiency virus; incidence rates among women, however, have continued to increase at approximately 0.7% per year since 1995 (3).

Nutritional status and physical activity are known to alter immune function, thereby making them important candidate risk factors for NHL (4). Adult height partly reflects genetics, early life nutrition, health experience, and growth-related hormones. Height and the prevalence of overweight and obesity have been increasing markedly over the past several decades (5, 6), whereas recreational physical activity has been declining. If these factors are associated with NHL risk, then such secular changes may explain part of the observed increase of overall NHL incidence, and they may also offer modifiable ways to reduce risk. Nevertheless, results from studies of body size and physical activity in relation to NHL risk have been mixed (7–27).

In this study, we used data from the California Teachers Study, a large prospective cohort of women at elevated risk

of NHL (28), to investigate whether body size and physical activity are associated with risk of B-cell NHL, which accounts for more than 85% of all NHL in US women. We further explored associations with risk of the 3 most common histologic subtypes (diffuse large B-cell lymphoma, follicular lymphoma, and B-cell chronic lymphocytic leukemia (CLL)/small lymphocytic lymphoma (SLL)) as defined by the World Health Organization classification for hematopoietic malignancies (29).

MATERIALS AND METHODS

Study population

A detailed description of the California Teachers Study has been published elsewhere (28). Briefly, this prospective study comprises 133,479 female public school professionals recruited through the California State Teachers Retirement System in 1995. All participants completed a self-administered baseline questionnaire, which collected information on demographic factors, height, weight, menstrual and reproductive factors, personal and family cancer and health history, oral contraceptive and menopausal hormone therapy use, and lifestyle factors (recreational physical activity, diet, alcohol consumption, and smoking). Participants completed a second self-administered questionnaire in 1997–1998 providing information on passive smoking exposure, medical radiation history, and waist and hip circumferences.

Use of human subjects in this study has been approved by the institutional review boards at the City of Hope, University of Southern California, Northern California Cancer Center, University of California at Irvine, and the California Department of Health and Human Services.

For analyses of height, weight, and body mass index at cohort entry, weight and body mass index at age 18 years, and physical activity, we sequentially excluded women who were not California residents, were aged 85 years or older at cohort entry, who had limited their participation to breast cancer research, who had a prior history of a hematopoietic malignancy, or whose history of cancer was unknown. The resulting analytical cohort consisted of 121,216 women aged 22–84 years of age. Among these women, 90,640 participants returned the second questionnaire. During the time interval between submission of the baseline questionnaire and the second questionnaire, 92 women were diagnosed with a hematopoietic malignancy, and 1,224 women moved out of California. A total of 89,324 women were eligible for the analyses of waist circumference, hip circumference, waist/hip ratio, and waist/height ratio.

Case ascertainment and follow-up

Incident diagnoses of B-cell NHL (*International Classification of Diseases for Oncology*, Third Edition (ICD-O-3), morphology codes 9590, 9591, 9670–9675, 9678–9699, 9727, 9823, 9832, 9835, 9836) were identified through annual linkages with the population-based California Cancer Registry, which receives information on over 99% of all cancer diagnoses occurring in California residents as part of a state mandate.

The status of California residence was monitored through annual mailing of newsletters or questionnaires, annual linkage with the US Postal Service national change-of-address database, and change-of-address postcards submitted by participants. For censoring purposes, information on the date of death was obtained from linkage with the California state mortality file, the National Death Index, and the Social Security Administration death master files (28).

Measurements of body size and recreational physical activity

Each participant reported her height (in feet and inches), weight (in pounds) at age 18 years and at cohort entry, participation in strenuous and moderate recreational physical activities during different life periods (throughout high school; ages 18–24, 25–34, 35–44, and 45–54 years, as relevant; and the 3 years before cohort entry), and waist and hip circumferences (in inches). In the second questionnaire, women were asked to measure their waist circumference and hip circumference twice; detailed instructions and a measuring tape were sent with the second questionnaire. Waist circumference was to be measured 1 inch (2.54 cm) above the navel, and hip circumference was to be measured at the largest point between the waist and thighs. Measurements were to be made at least 2 hours after a meal while participants were wearing only minimal clothing (such as underwear) or no clothing. In an ancillary validation study conducted within the cohort (30), comparison of the self-reported measurements with measurements taken by trained interviewers suggested excellent validity, with Pearson correlations of 0.87, 0.93, 0.85, and 0.87 for height, weight at cohort entry, waist circumference, and hip circumference, respectively. For our analyses, we classified both waist circumference and hip circumference as missing when both measurements were at opposite extremes of the distribution, that is, when the waist measurement was less than the 10th percentile and the hip measurement was greater than the 90th percentile, or when the opposite was true, that is, when the hip measurement was less than the 10th percentile and the waist measurement was greater than the 90th percentile.

Height at cohort entry was categorized into 5 approximately equal groups based on the distribution within the analytical cohort. Similarly, weight at age 18 years and cohort entry, waist and hip circumferences, the waist/hip ratio, and the waist/height ratio were categorized according to quartile distributions. Self-reported height and weight were used to calculate body mass index (kg/m^2) at age 18 years and cohort entry. Cutpoints for body mass index at cohort entry were based on the World Health Organization categorization: lean ($<20 \text{ kg}/\text{m}^2$), normal weight ($20\text{--}24.9 \text{ kg}/\text{m}^2$), overweight ($25\text{--}29.9 \text{ kg}/\text{m}^2$), or obese ($\geq 30 \text{ kg}/\text{m}^2$) (31). Because of the limited number of women whose body mass index was above $25 \text{ kg}/\text{m}^2$ at age 18 years, the quartile categories were defined for body mass index at age 18 years. Strenuous and moderate recreational physical activities were defined as long term (from high school to age 54 years or age at cohort entry, whichever was younger) and for the past 3 years. Both measurements of physical activity were categorized into 3 groups (≤ 0.50 , $0.51\text{--}3.99$, and ≥ 4.00 hours/week/year).

Statistical analyses

Multivariate Cox proportional hazards regression models provided estimates of the hazard rate ratio, a measurement of relative risk, and 95% confidence intervals. Women were followed from cohort entry (or submission of the second questionnaire, as relevant) until the first of the following events: diagnosis of B-cell NHL; move outside of California; diagnosis of a T-cell NHL, Hodgkin lymphoma, multiple myeloma, or leukemia other than prolymphocytic leukemias and CLL; death; or December 31, 2007. Age was used as the time scale. Statistical models were stratified by age in years at cohort entry. We included weight, height, age at menarche, and long-term strenuous plus moderate recreational physical activity as appropriate in our models. We assessed other potential risk factors (race, area-level social economic status (32), family history of lymphoma, smoking status at cohort entry, and alcohol consumption 1 year before cohort entry), but none of these factors altered relative risk estimates by as much as 5%, and therefore none was included in the final models. We performed tests for trend for ordinal variables by fitting the median value for each category as a continuous variable. We performed likelihood ratio tests to examine whether the trend between height and B-cell NHL risk was modified by body mass index at cohort entry (<25 kg/m² vs. ≥ 25 kg/m²), smoking status at cohort entry (never vs. ever), alcohol consumption 1 year before cohort entry (drinker vs. nondrinker), and long-term recreational physical activity (below median, ≤ 3.3 hours/week/year, vs. above median, > 3.3 hours/week/year, of combined long-term strenuous and moderate activities). We also examined if the effect of body mass index at cohort entry was modified by age at cohort entry (< 50 vs. ≥ 50 years) or menopausal status (pre- vs. postmenopausal status).

To exclude the possibility that an existing serious disease or other unexpected situations influenced anthropometric measurements and recent recreational physical activity (i.e., in the past 3 years) at cohort entry, we performed secondary analyses that excluded participants whose follow-up periods were less than 2 years after the baseline (2,754 subjects including 102 cases) or second questionnaire (2,015 subjects including 70 cases), as relevant.

For each exposure, potential associations with all B-cell NHL ($n = 574$), diffuse large B-cell lymphoma (ICD-O-3 codes 9678–9680 and 9684, $n = 155$), follicular lymphoma (ICD-O-3 codes 9690–9698, $n = 121$), and CLL/SLL (ICD-O-3 codes 9670 and 9823, $n = 124$) were evaluated. Two-sided P values are reported for tests for trend with $P < 0.05$ considered statistically significant. All statistical analyses were performed using SAS, version 9.1, software (SAS Institute, Inc., Cary, North Carolina).

RESULTS

The mean age at cohort entry was 52.7 years for women in the analytical cohort; 574 women were diagnosed with B-cell NHL during an average of 11.0 years of follow-up. The mean age at diagnosis was 69.5 years (range, 33–92 years); 38% of the participants were at least 1.68 m tall, and 42.2% of the participants reported averaging at least

4 hours/week of some moderate or strenuous recreational physical activity from high school through the age of 54 years (Table 1). Women with greater height or higher levels of recreational physical activity were more likely to be non-Hispanic white and younger in age, to have a higher level of alcohol consumption in the year prior to joining the cohort, and to have a lower body mass index at cohort entry. The distribution of respondent characteristics (as shown in Table 1) for those women who responded to the second questionnaire did not differ from that of the cohort overall.

Increasing height was statistically significantly associated with higher risk of B-cell NHL and CLL/SLL (Table 2). The risk for B-cell NHL was 50% greater among tall women (> 1.70 m) than among women in the middle height category (1.61–1.65 m) (relative risk = 1.50, 95% confidence interval: 1.16, 1.96); the risk for CLL/SLL among tall women was nearly double that of women in the middle height category (relative risk = 1.93, 95% confidence interval: 1.09, 3.41). No association was observed for diffuse large B-cell lymphoma and follicular lymphoma risk.

Weight at cohort entry was not associated with NHL risk overall, but a marginally statistically significant positive trend was observed for follicular lymphoma risk ($P_{\text{trend}} = 0.09$) (Table 2). The relative risks were comparable after exclusion of women whose follow-up periods were less than 2 years. Although B-cell NHL risk overall was not associated with body mass index, an inverse, marginally statistically significant association was detected between body mass index and risk of CLL/SLL, with the highest risk for lean women and the lowest risk for obese women ($P_{\text{trend}} = 0.06$) (Table 2). The exclusion of women whose follow-up periods were less than 2 years strengthened this association ($P_{\text{trend}} = 0.03$, data not shown) but had no substantial influence on B-cell NHL risk overall or other subtypes. Weight and body mass index at age 18 years were positively associated with overall B-cell NHL risk. Compared with women in the second quartiles of weight and body mass index at age 18 years, women in the highest quartiles had 30% greater risk of NHL for both measurements ($P_{\text{trend}} = 0.02$ and 0.03, respectively) (Table 2). No association was observed between weight and body mass index at age 18 years and risk of any specific NHL subtype.

We observed no association between NHL risk and long-term recreational physical activity or activity in the past 3 years (Table 3). The exclusion of women whose follow-up periods were less than 2 years did not affect these findings (data not shown). To investigate whether early adult life activity by itself influenced risk, we also examined physical activity prior to age 25 years; activity during early adulthood was not associated with NHL risk (data not shown).

Waist circumference and hip circumference were highly correlated with weight at cohort entry ($r = 0.75$ and $r = 0.79$, respectively; both $P < 0.0001$). Waist circumference, hip circumference, the waist/hip ratio, and the waist/height ratio were not associated with the risk of B-cell NHL overall (377 NHL cases) or with any of the NHL subtypes evaluated separately (Table 4). The results did not change after exclusion of women whose follow-ups were less than 2 years after submitting the second questionnaire (data not shown).

Table 1. Baseline Characteristics in Relation to Long-Term Recreational Physical Activity, Height, and Weight at Baseline of Women in the California Teachers Study, 1995–2007

Characteristic	No. of Subjects	Long-Term Physical Activity, % ^a				Height, %			Weight, %		
		≤0.5 Hours/ Week/Year	0. 51–3.99 Hours/ Week/Year	≥4 Hours/ Week/Year	Unknown	<1.68 m	≥1.68 m	Unknown	<63.5 kg	≥63.5 kg	Unknown
Total	121,216	9.9	47.4	42.2	0.6	61.6	38.1	0.4	44.0	52.2	3.8
Age at cohort entry, years											
<35	12,741	2.9	36.2	60.7	0.2	54.0	45.9	0.1	54.9	43.7	1.4
35–44	21,448	3.9	43.3	52.5	0.3	57.3	42.6	0.2	48.8	49.4	1.8
45–54	36,468	7.8	51.6	40.1	0.5	60.3	39.5	0.2	43.3	54.6	2.2
55–64	23,475	12.1	50.6	36.8	0.6	61.5	38.2	0.3	38.1	58.4	3.6
65–74	17,797	16.4	47.8	34.8	1.0	67.7	31.7	0.7	39.4	54.0	6.7
75–84	9,287	23.3	46.4	28.6	1.7	75.5	23.0	1.6	44.5	41.9	13.6
Race ^b											
Non-Hispanic white	104,814	8.5	48.8	42.2	0.5	58.46	41.29	0.25	42.7	54.3	3.0
All other races	15,405	12.9	48.3	38.2	0.6	77.53	21.91	0.56	48.1	48.0	3.9
Unknown race	997	12.7	43.6	42.2	1.5	66.95	32.27	0.77	41.0	49.8	9.2
First-degree family history of lymphoma ^b											
No	114,276	8.9	48.7	41.8	0.5	60.9	38.8	0.3	43.5	53.4	3.0
Yes	3,177	9.6	51.1	38.8	0.5	59.8	39.8	0.3	40.1	56.9	3.1
Adopted/unknown	3,763	12.4	46.2	40.4	1.0	62.9	36.4	0.7	42.2	51.9	6.0
Area-level socioeconomic status ^b											
Below median	26,086	9.9	48.5	41.0	0.6	62.1	37.6	0.4	37.2	59.5	3.3
Above median	93,584	8.8	48.8	41.8	0.6	60.7	39.1	0.3	45.0	51.9	3.1
Unknown	1,546	7.8	44.7	47.1	0.4	58.8	40.9	0.4	41.1	55.7	3.2
Alcohol consumption, g/day ^b											
None	38,619	11.7	49.9	38.0	0.5	63.3	36.4	0.3	40.7	56.0	3.3
<15	56,970	7.6	48.9	43.1	0.4	60.6	39.2	0.3	44.3	53.0	2.7
≥15	19,301	7.4	46.7	45.4	0.5	56.8	43.0	0.3	45.5	51.4	3.1
Unknown	6,326	11.9	46.3	38.9	2.8	63.2	36.1	0.7	44.5	48.5	7.1
Smoking status ^b											
Never	79,627	9.2	48.6	41.7	0.5	61.6	38.1	0.3	44.7	52.3	3.0
Former	34,684	8.5	49.3	41.6	0.5	59.5	40.2	0.3	40.7	56.1	3.2
Current	6,181	9.8	46.8	42.9	0.5	60.3	39.3	0.5	42.2	54.0	3.8
Unknown	724	9.4	47.5	32.5	10.7	63.2	36.1	0.7	43.5	49.2	7.3
Body mass index at cohort entry, kg/m ^{2b}											
<20	12,651	8.1	45.2	46.1	0.6	55.1	44.9		99.2	0.8	
20–24.9	58,204	7.9	47.7	44.0	0.5	61.0	39.0		65.9	34.1	
25–29.9	29,100	9.7	50.4	39.4	0.5	60.8	39.2		6.1	93.9	
≥30	16,414	11.0	52.7	35.7	0.5	65.8	34.2			100.0	
Unknown	4,847	15.9	45.8	36.9	1.4	56.8	34.4	8.8	2.8	2.2	95.1

^a Long-term physical activity combines strenuous and moderate physical activity.^b Age-adjusted percentages.

Table 2. Relative Risk and 95% Confidence Intervals for the Association Between Body Size and B-Cell Non-Hodgkin Lymphoma Risk Among Women in the California Teachers Study, 1995–2007

Variable	Person-Years	All Cases			Diffuse Large B-Cell			Follicular			CLL/SLL		
		Cases (n = 574)	Relative Risk	95% Confidence Interval	Cases (n = 155)	Relative Risk	95% Confidence Interval	Cases (n = 121)	Relative Risk	95% Confidence Interval	Cases (n = 124)	Relative Risk	95% Confidence Interval
Height, m ^a													
≤1.57	133,542	56	0.83	0.61, 1.14	21	1.04	0.61, 1.78	12	0.94	0.48, 1.85	8	0.52	0.24, 1.14
1.58–1.60	295,917	121	0.91	0.72, 1.15	28	0.72	0.45, 1.16	18	0.66	0.37, 1.16	30	1.04	0.63, 1.71
1.61–1.65	388,405	160	1.00	Referent	45	1.00	Referent	36	1.00	Referent	33	1.00	Referent
1.66–1.70	319,663	143	1.18	0.94, 1.48	37	1.11	0.72, 1.72	36	1.23	0.77, 1.96	33	1.41	0.87, 2.30
>1.70	188,893	91	1.50	1.16, 1.96	23	1.40	0.84, 2.35	18	1.13	0.63, 2.02	20	1.93	1.09, 3.41
Unknown	4,500	3			1			1			0		
<i>P</i> _{trend}			<0.001			0.16			0.22			0.001	
Weight, kg ^b													
<56.7	269,229	101	1.14	0.87, 1.49	33	1.24	0.76, 2.03	16	0.86	0.45, 1.65	22	1.05	0.60, 1.85
56.7–<63.5	316,356	114	1.00	Referent	33	1.00	Referent	24	1.00	Referent	29	1.00	Referent
63.5–<73.0	372,853	163	1.05	0.83, 1.34	40	0.90	0.57, 1.43	34	1.07	0.63, 1.81	35	0.84	0.51, 1.38
≥73.0	323,294	161	1.18	0.93, 1.51	42	1.08	0.68, 1.72	40	1.41	0.84, 2.37	25	0.67	0.39, 1.15
Unknown	49,189	35			7			7			13		
<i>P</i> _{trend}			0.36			0.81			0.09			0.14	
Body mass index, kg/m ^{2b}													
<20	138,366	52	1.13	0.84, 1.53	17	1.42	0.83, 2.42	9	0.94	0.46, 1.92	15	1.55	0.88, 2.75
20–24.9	642,731	246	1.00	Referent	64	1.00	Referent	50	1.00	Referent	54	1.00	Referent
25–29.9	319,511	154	1.06	0.86, 1.29	41	1.07	0.72, 1.59	35	1.23	0.80, 1.90	32	0.97	0.63, 1.51
≥30	180,213	86	1.19	0.93, 1.52	26	1.37	0.86, 2.16	19	1.29	0.77, 2.19	10	0.63	0.32, 1.24
Unknown	50,099	36			7			8			13		
<i>P</i> _{trend}			0.34			0.50			0.26			0.06	
Weight at age 18 years, kg ^c													
<52.6	349,338	116	0.99	0.77, 1.27	33	0.88	0.54, 1.41	23	1.03	0.59, 1.81	22	0.75	0.43, 1.29
52.6–<57.2	394,356	152	1.00	Referent	45	1.00	Referent	31	1.00	Referent	40	1.00	Referent
57.2–<61.7	230,123	121	1.37	1.08, 1.75	30	1.16	0.72, 1.84	30	1.62	0.98, 2.70	23	0.97	0.58, 1.62
≥61.7	302,896	145	1.30	1.02, 1.64	40	1.23	0.79, 1.92	28	1.15	0.68, 1.95	27	0.88	0.53, 1.47
Unknown	54,207	40			7			9			12		
<i>P</i> _{trend}			0.02			0.19			0.55			0.93	
Body mass index at age 18 years, kg/m ^{2c}													
<19.5	324,719	134	1.04	0.81, 1.33	37	0.98	0.62, 1.56	25	0.95	0.54, 1.66	30	0.94	0.57, 1.57
19.5–20.7	307,661	123	1.00	Referent	36	1.02	Referent	25	1.00	Referent	30	1.00	Referent
20.8–22.4	310,622	124	1.03	0.80, 1.32	32	0.90	0.56, 1.45	31	1.28	0.75, 2.17	23	0.78	0.45, 1.35
>22.4	327,153	153	1.30	1.02, 1.64	43	1.23	0.79, 1.92	31	1.27	0.75, 2.15	29	1.03	0.62, 1.73
Unknown	60,765	40			7			9			12		
<i>P</i> _{trend}			0.03			0.30			0.22			0.87	

Abbreviations: CLL, chronic lymphocytic leukemia; SLL, small lymphocytic lymphoma.

^a Adjusted for weight at cohort entry, age at menarche (≤11, 12, ≥13 years, never had period, missing), and long-term strenuous plus moderate physical activity.^b Adjusted for height, age at menarche (≤11, 12, ≥13 years, never had period, missing), and long-term strenuous plus moderate physical activity.^c Adjusted for height, age at menarche (≤11, 12, ≥13 years, never had period, missing), and strenuous plus moderate physical activity in high school.

Table 3. Relative Risk and 95% Confidence Intervals for the Association Between Recreational Physical Activity and B-Cell Non-Hodgkin Lymphoma Risk Among Women in the California Teachers Study, 1995–2007

Variable	Person-Years	All Cases				Diffuse Large B-Cell				Follicular				CLL/SLL			
		Cases (n = 574)	Relative Risk	95% Confidence Interval	Cases (n = 155)	Relative Risk	95% Confidence Interval	Cases (n = 121)	Relative Risk	95% Confidence Interval	Cases (n = 124)	Relative Risk	95% Confidence Interval	Cases (n = 124)	Relative Risk	95% Confidence Interval	
Long-term strenuous plus moderate physical activity, hours/week/year ^a																	
0–0.50	126,700	86	1.00	Referent	25	1.00	Referent	13	1.00	Referent	20	1.00	Referent				
0.51–3.99	631,621	255	0.85	0.66, 1.09	66	0.76	0.48, 1.21	57	1.15	0.63, 2.12	59	0.91	0.55, 1.52				
≥4.00	565,023	226	1.00	0.78, 1.29	63	0.97	0.60, 1.55	49	1.29	0.69, 2.41	44	0.94	0.55, 1.60				
Unknown	7,576	7			1			2			1						
<i>P</i> _{trend}				0.27			0.46						0.43			0.92	
Recent strenuous plus moderate physical activity, hours/week/year ^{a,b}																	
0–0.50	302,306	131	1.00	Referent	38	1.00	Referent	25	1.00	Referent	24	1.00	Referent				
0.51–3.99	541,871	226	1.14	0.91, 1.44	59	1.02	0.66, 1.58	55	1.32	0.80, 2.19	51	1.48	0.89, 2.48				
≥4.00	479,168	210	1.11	0.86, 1.44	57	1.00	0.62, 1.62	39	1.01	0.57, 1.79	48	1.50	0.86, 2.63				
Unknown	7,576	7			1			2			1						
<i>P</i> _{trend}				0.63			0.98						0.58			0.31	

Abbreviations: CLL, chronic lymphocytic leukemia; SLL, small lymphocytic lymphoma.

^a Adjusted for height, weight at cohort entry, and age at menarche (≤11, 12, ≥13 years, never had period, missing).

^b Recent activity indicates activity in the 3 years before cohort entry; model was further adjusted for long-term strenuous plus moderate physical activity.

We did not detect any statistically significant modification of the association between height and risk of all B-cell NHL by body mass index at cohort entry, alcohol consumption, smoking status, or long-term physical activity (data not shown). Body mass index at cohort entry was not associated with NHL risk among younger or older women or among premenopausal or postmenopausal women (data not shown).

DISCUSSION

In this large cohort of female public school professionals, we detected statistically significant positive associations between height and risk of all B-cell NHL and the CLL/SLL subtype. Weight and body mass index at age 18 years were positively associated with B-cell NHL risk overall, but weight and body mass index at cohort entry were not, other than an inverse association with CLL/SLL risk.

The association between height and NHL risk has been examined in several studies with inconsistent results. Among the studies that reported the associations separately for women, the Nurses’ Health Study (33) and the Multiethnic Cohort Study (13) reported positive associations between height and overall NHL risk, whereas the Iowa Women’s Health Study cohort (8) reported no impact of height on risk of NHL overall or any subtype. Two cohort studies from Europe both reported a stronger association among women than men (26, 27); however, one study reported a statistically significant association with follicular lymphoma (27), whereas another study reported increased risk for diffuse large B-cell lymphoma (26). A pooled analysis from the InterLymph Consortium (14) reported that the tallest men were at increased risk of NHL, but no association was observed for women. Of 3 studies reporting results for men and women combined, 1 study found increased risk for diffuse large B-cell lymphoma and CLL/SLL with increasing height (12); 1 study reported a significantly increased risk for follicular lymphoma among tall individuals (9); and the third study found a positive association between height and risk of CLL, but not NHL overall (10). A study of children and adolescents (<18 years at diagnosis) found that girls with NHL or Hodgkin lymphoma are taller than population norms (34).

Adult height is influenced by both genetic factors and environmental exposures that may occur in utero or during infancy, childhood, or puberty. Frequent infections in early life may influence height, with persistent effects on cancer risk later in life (35, 36). One possibility is that individuals exposed to high-pathogen environments may invest energy in developing antipathogen defenses, which could result in impaired growth and shorter height (37, 38). Early exposures to pathogens may also prime the immune system to optimize protection against infectious pathogens that are likely to be encountered later in life. Data showing significant differences in the levels of immunoglobulins M, G, and A between urban and rural residents in a relatively genetically homogenous population in Nigeria support this hypothesis (39). On this basis, we might hypothesize that the

Table 4. Relative Risk and 95% Confidence Intervals for the Association Between Waist Circumference, Hip Circumference, Waist/Hip Ratio, and Waist/Height Ratio and B-Cell Non-Hodgkin Lymphoma Risk Among Women in the California Teachers Study, 1997–2007

Variable	Person-Years	All Cases			Diffuse Large B-Cell			Follicular			CLL/SLL		
		Cases (n = 377)	Relative Risk	95% Confidence Interval	Cases (n = 99)	Relative Risk	95% Confidence Interval	Cases (n = 85)	Relative Risk	95% Confidence Interval	Cases (n = 81)	Relative Risk	95% Confidence Interval
Waist circumference, m ^a													
<0.74	200,090	67	1.01	0.72, 1.41	17	0.78	0.41, 1.48	11	0.67	0.31, 1.42	17	1.14	0.58, 2.25
0.74–0.81	194,095	85	1.00	Referent	26	1.00	Referent	22	1.00	Referent	19	1.00	Referent
0.82–0.89	164,529	76	0.84	0.61, 1.15	20	0.78	0.43, 1.43	15	0.62	0.32, 1.23	18	0.89	0.46, 1.73
>0.89	177,277	111	1.03	0.74, 1.44	27	0.92	0.48, 1.74	30	1.02	0.53, 1.97	22	0.99	0.49, 2.02
Unknown	94,628	38			9			7			5		
<i>P</i> _{trend}				0.76			0.72			0.25			0.62
Hip circumference, m ^a													
<0.95	205,668	88	1.14	0.81, 1.59	24	0.88	0.47, 1.65	18	1.62	0.75, 3.52	25	1.38	0.70, 2.71
0.95–1.00	169,586	70	1.00	Referent	22	1.00	Referent	12	1.00	Referent	16	1.00	Referent
1.01–1.07	189,641	91	1.03	0.75, 1.43	22	0.85	0.46, 1.59	22	1.34	0.65, 2.78	15	0.77	0.37, 1.60
>1.07	169,403	89	1.15	0.79, 1.66	20	0.85	0.41, 1.76	27	1.77	0.80, 3.91	20	1.40	0.65, 3.05
Unknown	96,321	39			11			6			5		
<i>P</i> _{trend}				0.98			0.85			0.64			0.98
Waist/hip ratio ^a													
<0.76	198,768	61	0.92	0.65, 1.30	18	1.25	0.63, 2.48	13	0.66	0.33, 1.32	12	0.81	0.38, 1.73
0.76–0.79	172,577	70	1.00	Referent	15	1.00	Referent	21	1.00	Referent	16	1.00	Referent
0.80–0.85	182,829	92	1.00	0.73, 1.37	23	1.22	0.63, 2.34	15	0.57	0.29, 1.11	24	1.12	0.59, 2.11
>0.85	177,793	112	0.94	0.69, 1.29	32	1.35	0.71, 2.55	28	0.83	0.45, 1.51	23	0.82	0.42, 1.59
Unknown	98,652	42			11			8			6		
<i>P</i> _{trend}				0.97			0.56			0.55			0.77
Waist/height ratio ^b													
<0.45	204,921	71	1.10	0.79, 1.53	18	0.78	0.42, 1.44	12	0.78	0.37, 1.65	20	1.66	0.84, 3.28
0.45–0.48	175,034	76	1.00	Referent	26	1.00	Referent	18	1.00	Referent	15	1.00	Referent
0.49–0.55	171,804	84	0.86	0.63, 1.18	18	0.56	0.30, 1.04	22	0.93	0.49, 1.77	18	0.94	0.47, 1.89
>0.55	182,083	106	0.85	0.61, 1.18	27	0.69	0.37, 1.28	25	0.78	0.39, 1.54	23	0.98	0.48, 2.02
Unknown	96,777	40			10			8			5		
<i>P</i> _{trend}				0.16			0.57			0.91			0.18

Abbreviations: CLL, chronic lymphocytic leukemia; SLL, small lymphocytic lymphoma.

^a Adjusted for height, weight at cohort entry, age at menarche (≤ 11 , 12, ≥ 13 years, never had period, missing), and long-term strenuous plus moderate physical activity.^b Adjusted for weight at cohort entry, age at menarche (≤ 11 , 12, ≥ 13 years, never had period, missing), and long-term strenuous plus moderate physical activity.

immune function of taller individuals is less developed and, therefore, the proliferative response of T and B cells in adulthood to unfamiliar antigens may result in the expansion of malignant clones. Several studies have examined the "hygiene hypothesis" and markers of infectious burden as they relate to NHL risk but, altogether, these studies remain equivocal (40, 41).

Height is positively associated with circulating levels of insulin-like growth factor 1 in childhood (42–45) but not in adulthood (46). Insulin-like growth factor 1 is a major regulator of childhood growth and mediates most of the anabolic actions of growth hormone (47). In a rodent model, insulin-like growth factor 1 administration increased the number of lymphocytes and the size of lymphoid organs (e.g., spleen and thymus) (48, 49). Insulin-like growth factor 1 also potentiates pro-B to pre-B cell maturation (50), stimulates B-cell proliferation (51), and inhibits apoptosis (52), all of which may contribute to the promotion of NHL development. Thus, insulin-like growth factor 1 levels in childhood, as reflected by growth patterns and, ultimately, height, may have long-lasting impact on the development of NHL.

Evidence from recent studies suggests that early adult life body size may be associated with a modest increase in risk of NHL rather than body size later in life (13, 26). The Multiethnic Cohort Study reported that weight and body mass index at age 21 years were more strongly associated with NHL risk than was recent body mass index among women (13). The Netherlands Cohort Study observed a significant positive association between body mass index at age 20 years and risk of lymphatic malignancies overall, although no association was observed for NHL risk (26). Nevertheless, body size in later life was not associated with NHL risk as suggested by most recent cohort studies (13, 25–27). The InterLymph Consortium pooled analysis of 18 case-control studies also reported no association between body mass index around the age at diagnosis and NHL risk (14). Consistently, a British case-control study reported that the positive association between body mass index and NHL risk was more pronounced among individuals diagnosed at younger ages (11).

The average age of women in most cohort studies at the time of formation is greater than 50 years. For example, the mean age at cohort entry was 52.7 years in our study and 60 years in the Multiethnic Cohort Study (13). Thus, weight and body mass index in early adult life may better represent body size status during a relevant exposure period, and measurements at cohort entry may reflect later life events including changes in health status, menopause, or alterations in lifestyle. Besides the potential for higher levels of insulin-like growth factor 1 due to greater caloric intake during childhood and adolescence as noted above (53), other possible mechanisms that may mediate an association between early life obesity and NHL risk include insulin resistance, chronic hyperinsulinemia, or increased bioavailability of steroid hormones (6, 54, 55). Recently, studies of polymorphisms in the leptin and leptin receptor genes have shown that leptin, an adipocyte-derived hormone regulating food intake and immune function, may be a mediator in NHL pathogenesis (11, 19).

Compared with body mass index, the waist circumference, waist/hip ratio, and waist/height ratio may be better measurements of body fat distribution. Abdominal obesity, as defined by waist circumference or the waist/hip ratio, has been shown to be more strongly related to certain types of cancer than obesity as defined by body mass index (6). However, consistent with previous studies (8, 12, 27), this study did not find associations between these factors and risk of NHL.

We found no association between long-term or recent recreational physical activity and NHL risk, consistent with the results from the Iowa Women's Health Study (8) but in contrast with 2 case-control studies that reported inverse associations between recent recreational physical activity and NHL risk (9, 22). Studies of recent activity may provide spurious inverse associations, because early symptoms of NHL can result in reduced activity and, therefore, more reported activity among noncases than among cases. We did not collect occupational physical activity information in our study. However, results from 3 previous studies found that occupational physical activity was not associated with NHL risk (7, 9, 15).

The major strengths of this study include its prospective design, an evaluation of anthropometric variables reflecting various aspects of body size, comprehensive follow-up procedures, virtually complete ascertainment of cancer outcomes, and our ability to adjust for a broad range of potential confounders. Several limitations must be considered. First, the number of cases available by subtype was limited, resulting in lack of precision in risk estimates. Second, self-reported measurements of body size may be inaccurate, with heavier women being more likely to underreport weight and shorter women being more likely to overreport height (56). However, as we mentioned in Materials and Methods, the self-reported measurements are highly correlated with the measurements by trained interviewers. In addition, with our cohort design, these measurement errors would be expected to be nondifferential between affected women (cases) and unaffected women and, thus, would be expected to attenuate the true underlying associations with NHL risk. Finally, this cohort of mostly non-Hispanic white (86.4%), college-educated women is not representative of all women in the United States. Nevertheless, the height and obesity distributions within the cohort are comparable with those of the US non-Hispanic white population (57, 58).

In conclusion, we found that height was positively associated with risk of B-cell NHL, especially CLL/SLL. Height may well reflect the consequences of early life exposure to pathogens, suggesting that growth hormone levels or maturation of the immune system may play a role in NHL etiology. In addition, body size at age 18 years, which reflects early life nutritional status, may be modestly associated with increased B-cell NHL risk. Further studies are needed to provide insight into the mechanisms involved in these associations and to illuminate whether nutritional regulation or weight loss can decrease NHL risk.

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REFERENCES

- Jemal A, Siegel R, Ward E, et al. Cancer statistics, 2009. *CA Cancer J Clin*. 2009;59(4):225–249.
- Hartge P, Devesa SS. Quantification of the impact of known risk factors on time trends in non-Hodgkin's lymphoma incidence. *Cancer Res*. 1992;52(19 suppl):5566s–5569s.
- Ries LAG, Melbert D, Krapcho M, et al, eds. *SEER Cancer Statistics Review, 1975–2005*. Bethesda, MD: National Cancer Institute; 2008. (http://seer.cancer.gov/csr/1975_2005/).
- Hance KW, Rogers CJ, Hursting SD, et al. Combination of physical activity, nutrition, or other metabolic factors and vaccine response. *Front Biosci*. 2007;12:4997–5029.
- Crimmins EM, Finch CE. Infection, inflammation, height, and longevity. *Proc Natl Acad Sci U S A*. 2006;103(2):498–503.
- Pischoon T, Nöthlings U, Boeing H. Obesity and cancer. *Proc Nutr Soc*. 2008;67(2):128–145.
- Zahm SH, Hoffman-Goetz L, Dosemeci M, et al. Occupational physical activity and non-Hodgkin's lymphoma. *Med Sci Sports Exerc*. 1999;31(4):566–571.
- Cerhan JR, Janney CA, Vachon CM, et al. Anthropometric characteristics, physical activity, and risk of non-Hodgkin's lymphoma subtypes and B-cell chronic lymphocytic leukemia: a prospective study. *Am J Epidemiol*. 2002;156(6):527–535.
- Cerhan JR, Bernstein L, Severson RK, et al. Anthropometrics, physical activity, related medical conditions, and the risk of non-Hodgkin lymphoma. *Cancer Causes Control*. 2005;16(10):1203–1214.
- Chang ET, Hjalgrim H, Smedby KE, et al. Body mass index and risk of malignant lymphoma in Scandinavian men and women. *J Natl Cancer Inst*. 2005;97(3):210–218.
- Willett EV, Skibola CF, Adamson P, et al. Non-Hodgkin's lymphoma, obesity and energy homeostasis polymorphisms. *Br J Cancer*. 2005;93(7):811–816.
- Lim U, Morton LM, Subar AF, et al. Alcohol, smoking, and body size in relation to incident Hodgkin's and non-Hodgkin's lymphoma risk. *Am J Epidemiol*. 2007;166(6):697–708.
- Maskarinec G, Erber E, Gill J, et al. Overweight and obesity at different times in life as risk factors for non-Hodgkin's lymphoma: the Multiethnic Cohort. *Cancer Epidemiol Biomarkers Prev*. 2008;17(1):196–203.
- Willett EV, Morton LM, Hartge P, et al. Non-Hodgkin lymphoma and obesity: a pooled analysis from the InterLymph Consortium. *Int J Cancer*. 2008;122(9):2062–2070.
- Brownson RC, Chang JC, Davis JR, et al. Physical activity on the job and cancer in Missouri. *Am J Public Health*. 1991;81(5):639–642.
- Wolk A, Gridley G, Svensson M, et al. A prospective study of obesity and cancer risk (Sweden). *Cancer Causes Control*. 2001;12(1):13–21.
- Morton LM, Holford TR, Leaderer B, et al. Alcohol use and risk of non-Hodgkin's lymphoma among Connecticut women (United States). *Cancer Causes Control*. 2003;14(7):687–694.
- Samanic C, Gridley G, Chow WH, et al. Obesity and cancer risk among white and black United States veterans. *Cancer Causes Control*. 2004;15(1):35–43.
- Skibola CF, Holly EA, Forrest MS, et al. Body mass index, leptin and leptin receptor polymorphisms, and non-Hodgkin lymphoma. *Cancer Epidemiol Biomarkers Prev*. 2004;13(5):779–786.
- Bosetti C, Dal Maso L, Negri E, et al. Re: Body mass index and risk of malignant lymphoma in Scandinavian men and women. *J Natl Cancer Inst*. 2005;97(11):860–861.
- Oh SW, Yoon YS, Shin SA. Effects of excess weight on cancer incidences depending on cancer sites and histologic findings among men: Korea National Health Insurance Corporation Study. *J Clin Oncol*. 2005;23(21):4742–4754.
- Pan SY, Mao Y, Ugnat AM, et al. Physical activity, obesity, energy intake, and the risk of non-Hodgkin's lymphoma: a population-based case-control study. *Am J Epidemiol*. 2005;162(12):1162–1173.
- Fernberg P, Odenbro A, Belloc R, et al. Tobacco use, body mass index and the risk of malignant lymphomas—a nationwide cohort study in Sweden. *Int J Cancer*. 2006;118(9):2298–2302.
- Chiu BC, Soni L, Gapstur SM, et al. Obesity and risk of non-Hodgkin lymphoma (United States). *Cancer Causes Control*. 2007;18(6):677–685.
- Söderberg KC, Kaprio J, Verkasalo PK, et al. Overweight, obesity and risk of haematological malignancies: a cohort study of Swedish and Finnish twins. *Eur J Cancer*. 2009;45(7):1232–1238.
- Pylypchuk RD, Schouten LJ, Goldbohm RA, et al. Body mass index, height, and risk of lymphatic malignancies: a prospective cohort study. *Am J Epidemiol*. 2009;170(3):297–307.
- Britton JA, Khan AE, Rohrmann S, et al. Anthropometric characteristics and non-Hodgkin's lymphoma and multiple myeloma risk in the European Prospective Investigation into Cancer and Nutrition (EPIC). *Haematologica*. 2008;93(11):1666–1677.

28. Bernstein L, Allen M, Anton-Culver H, et al. High breast cancer incidence rates among California teachers: results from the California Teachers Study (United States). *Cancer Causes Control*. 2002;13(7):625–635.
29. Jaffe ES, Harris NL, Stein H, et al. *Pathology and Genetics of Tumours of Haematopoietic and Lymphoid Tissues*. Lyon, France: IARC Press; 2001.
30. Horn-Ross PL, Barnes S, Lee VS, et al. Reliability and validity of an assessment of usual phytoestrogen consumption (United States). *Cancer Causes Control*. 2006;17(1):85–93.
31. Physical status: the use and interpretation of anthropometry. Report of a WHO Expert Committee. *World Health Organ Tech Rep Ser*. 1995;854:1–452.
32. Reynolds P, Hurley S, Goldberg DE, et al. Regional variations in breast cancer among California teachers. *Epidemiology*. 2004;15(6):746–754.
33. Zhang S, Hunter DJ, Rosner BA, et al. Dietary fat and protein in relation to risk of non-Hodgkin's lymphoma among women. *J Natl Cancer Inst*. 1999;91(20):1751–1758.
34. Pui CH, Dodge RK, George SL, et al. Height at diagnosis of malignancies. *Arch Dis Child*. 1987;62(5):495–499.
35. McDade TW. Life history theory and the immune system: steps toward a human ecological immunology. *Am J Phys Anthropol*. 2003;122(suppl 37):100–125.
36. Blackwell DL, Hayward MD, Crimmins EM. Does childhood health affect chronic morbidity in later life? *Soc Sci Med*. 2001;52(8):1269–1284.
37. Butte NF, Wong WW, Garza C. Energy cost of growth during infancy. *Proc Nutr Soc*. 1989;48(2):303–312.
38. Lunn PG. The impact of infection and nutrition on gut function and growth in childhood. *Proc Nutr Soc*. 2000;59(1):147–154.
39. Mohammed I, Tomkins AM, Greenwood BM. Normal immunoglobulins in the tropics [letter]. *Lancet*. 1973;301(7801):481.
40. Smedby KE, Hjalgrim H, Chang ET, et al. Childhood social environment and risk of non-Hodgkin lymphoma in adults. *Cancer Res*. 2007;67(22):11074–11082.
41. Bracci PM, Dalvi TB, Holly EA. Residential history, family characteristics and non-Hodgkin lymphoma, a population-based case-control study in the San Francisco Bay Area. *Cancer Epidemiol Biomarkers Prev*. 2006;15(7):1287–1294.
42. Rogers I, Emmett P, Gunnell D, et al. Milk as a food for growth? The insulin-like growth factors link. *Public Health Nutr*. 2006;9(3):359–368.
43. Rogers I, Metcalfe C, Gunnell D, et al. Insulin-like growth factor-I and growth in height, leg length, and trunk length between ages 5 and 10 years. *J Clin Endocrinol Metab*. 2006;91(7):2514–2519.
44. Hoppe C, Udam TR, Lauritzen L, et al. Animal protein intake, serum insulin-like growth factor I, and growth in healthy 2.5-y-old Danish children. *Am J Clin Nutr*. 2004;80(2):447–452.
45. Rogers IS, Gunnell D, Emmett PM, et al. Cross-sectional associations of diet and insulin-like growth factor levels in 7- to 8-year-old children. *Cancer Epidemiol Biomarkers Prev*. 2005;14(1):204–212.
46. Bray I, Gunnell D, Holly JM, et al. Associations of childhood and adulthood height and the components of height with insulin-like growth factor levels in adulthood: a 65-year follow-up of the Boyd Orr cohort. *J Clin Endocrinol Metab*. 2006;91(4):1382–1389.
47. Butler AA, Le Roith D. Control of growth by the somatotropic axis: growth hormone and the insulin-like growth factors have related and independent roles. *Annu Rev Physiol*. 2001;63:141–164.
48. Clark R, Strasser J, McCabe S, et al. Insulin-like growth factor-1 stimulation of lymphopoiesis. *J Clin Invest*. 1993;92(2):540–548.
49. Clark R. The somatogenic hormones and insulin-like growth factor-1: stimulators of lymphopoiesis and immune function. *Endocr Rev*. 1997;18(2):157–179.
50. Landreth KS, Narayanan R, Dorshkind K. Insulin-like growth factor-I regulates pro-B cell differentiation. *Blood*. 1992;80(5):1207–1212.
51. Gibson LF, Piktel D, Landreth KS. Insulin-like growth factor-1 potentiates expansion of interleukin-7-dependent pro-B cells. *Blood*. 1993;82(10):3005–3011.
52. Buckbinder L, Talbott R, Velasco-Miguel S, et al. Induction of the growth inhibitor IGF-binding protein 3 by p53. *Nature*. 1995;377(6550):646–649.
53. Schernhammer ES, Tworoger SS, Eliassen AH, et al. Body shape throughout life and correlations with IGFs and GH. *Endocr Relat Cancer*. 2007;14(3):721–732.
54. Calle EE, Kaaks R. Overweight, obesity and cancer: epidemiological evidence and proposed mechanisms. *Nat Rev Cancer*. 2004;4(8):579–591.
55. Højgaard B, Gyrd-Hansen D, Olsen KR, et al. Waist circumference and body mass index as predictors of health care costs [electronic article]. *PLoS One*. 2008;3(7):e2619.
56. Gorber SC, Tremblay M, Moher D, et al. A comparison of direct vs. self-report measures for assessing height, weight and body mass index: a systematic review. *Obes Rev*. 2007;8(4):307–326.
57. Ogden CL, Carroll MD, Curtin LR, et al. Prevalence of overweight and obesity in the United States, 1999–2004. *JAMA*. 2006;295(13):1549–1555.
58. Ogden CL, Fryar CD, Carroll MD, et al. Mean body weight, height, and body mass index, United States 1960–2002. *Adv Data*. 2004;(347):1–17.