

Cancer incidence patterns among adolescents and young adults in the United States

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Received 24 June 2004; accepted in revised form 30 September 2004

Key words: adolescence, age groups, cancer incidence, cancer registry, epidemiology, epithelial cancer, gender, non-epithelial cancer, race, young adult.

Abstract

Objective: The purpose of this study was to examine age-specific cancer incidence patterns among adolescents and young adults (ages 15–49).

Method: Cancer incidence data for 1995–1999 from 22 population-based central cancer registries, covering about 47% of the US population, were used. Relative frequencies and average annual age-specific incidence rates per 100,000 person-year were computed for the five-year age groups from age 15–19 years through 45–49 years. Tests of significance for comparison were at a level of $p < 0.05$.

Results: The age at crossover from a predominance of non-epithelial cancers to a predominance of epithelial cancers during adolescence and young adulthood varied by gender and race. Epithelial cancer became the predominant type of tumor after age 40 years among males while it was the predominant type after age 25 years among females. There was also a shift in the top five cancer types with increasing age, which varied by race and gender. Epithelial cancers of the thyroid, breast, ovary, and cervix uteri started to increase sharply among young women in their 20s while among males epithelial cancers rarely occurred until the early 30s (ages 30–34). Cancers of the female breast, colon and rectum, and lung began to occur at an earlier age and increased more sharply among blacks than among whites. However, the incidence rates of epithelial thyroid and ovarian cancers rose more quickly among whites than blacks. Non-Hodgkin lymphoma and soft tissue sarcoma (excluded Kaposi's sarcoma) increased with age among both whites and blacks but the rates were significantly higher among blacks than among whites. Both Kaposi's sarcoma and testicular cancer incidence increased with age and peaked in the early 30s (ages 30–34). The former was significantly higher among blacks than whites while the latter was significantly higher among whites than blacks. Cervical cancer incidence leveled off when white women reached their 30s, but for black women the rate continued to rise with advancing age. Cutaneous melanoma rates were significantly higher among females than among males between the ages of 15 and 39.

Conclusion: Cancer incidence patterns among adolescents and young adults are distinctive. Specific cancer prevention and control strategies should be targeted accordingly and tailored to their specific needs.

Introduction

Because of their relatively low cancer incidence, the adolescent and young adult population has not been the focus of attention in cancer control and prevention in the United States. Although numerous cancer epidemi-

ology studies for specific types of cancer include adolescents and young adults, there have been few publications on overall cancer incidence patterns. Adolescents and young adults are a distinctive population in terms of cancer occurrence. Among children, the majority of incident cancers are of non-epithelial origin, while epithelial cancers predominate among adults [1, 2]. The shift from a predominance of non-epithelial cancers to a predominance of epithelial cancers proceeds through several decades of age in adolescence and young

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adulthood [3, 4]. Cancers occurring among adolescents and young adults are more likely to relate to genetic predisposition and exposure to risk factors early in life as compared with cancers among the elderly. Information on overall cancer incidence patterns will not only provide leads for etiologic research but also help us to identify cancers that have the greatest impact on these age groups. This study examined the age-specific incidence patterns for selected cancers by race and gender among 15 to 49-year-olds, using a large aggregated cancer incidence data file from the North American Association of Central Cancer Registries (NAACCR).

Materials and methods

Study data

Cancer incidence data file for 1995–1999 were obtained from NAACCR, including data for 22 population-based central cancer registries that met the NAACCR high quality criteria for incidence data and consented to the use of their data for this study. These registries were Colorado, Connecticut, Atlanta (Georgia), Hawaii, Iowa, Idaho, Illinois, Kentucky, Louisiana, Michigan, Minnesota, Nebraska, New Jersey, New Mexico, North Carolina, Pennsylvania, Rhode Island, Utah, Seattle/Puget Sound (Washington), West Virginia, Wisconsin, and Wyoming. These states and metropolitan areas cover approximately 47% of the US population, 47% of whites and 40% of blacks.

The NAACCR criteria for high-quality cancer incidence data include completeness of case ascertainment (90% or higher), non-duplication of reported cancer cases (less than 0.2% duplicate cases), internal consistency among data items defined by the NAACCR EDITS metafile (<http://www.naacr.org/Standards/Edits.html>), few death-certificate-only cases (less than 5%), and few cases with missing/unknown race (less than 5% unknown), gender, or age (less than 3% unknown) [5].

Only cancer incident cases among 15 to 49-year-olds were included in this study. All cancers were invasive except for bladder cancer for which *in situ* cases were included in order to be consistent with published population-based cancer incidence statistics.

This study focused on white and black populations. Case counts for other races were too small to produce reliable results. In addition, there was a uncertainty about quality of classification for some race groups such as American Indian and Alaska Natives. Because histologic misclassification is more likely in cases without microscopic confirmation, we excluded 6470 such cases from all cancers (2% of the total). Population estimates for 1995–

1999 were obtained from the SEER Program, based on US Bureau of Census population estimates for these years.

All incident cancer cases included in this study were coded according to the *International Classification of Diseases for Oncology, Second Edition* (ICD-O-2) topography (site) and morphology codes [6]. Based on the morphology codes, the cancer cases were divided into two major categories: epithelial and non-epithelial. The classification of non-epithelial and epithelial cancers in this study was consistent with conventional practice: the epithelial category included cancers with morphology codes from M-8010 through M-8580; the non-epithelial category included cancers with morphology codes from M-8590 through M-9941; cancers coded as “neoplasms not otherwise specified” (M-8000 through M8004), accounted for approximately 0.34% of cases, which were included in all cancers category but not in site-specific categories. Both epithelial and non-epithelial cases were further subdivided into site-specific categories according to the SEER site recodes [2], with two exceptions: (1) soft tissue sarcomas were categorized in accordance with the International Classification for Childhood Cancer (ICCC) scheme but Kaposi’s sarcoma was listed as a independent category from the soft tissue sarcoma group [7], and (2) ovarian carcinomas were categorized using the International Agency for Research on Cancer’s (IARC) histological groups [8]. Due to limitation of space, only those cancers that demonstrated large race or gender differences were focused in this report. According to the classification for epithelial and non-epithelial cancers in this study, cases for brain and other central nervous system (brain & CNS) tumors, Hodgkin lymphoma (HL), Kaposi’s sarcoma, leukemia, melanoma of the skin, non-Hodgkin lymphoma (NHL), soft tissue sarcoma (STS), and testicular cancer fell into the non-epithelial cancer category. The vast majority of cancers of the cervix uteri, colon & rectum, corpus uteri, female breast, lung, thyroid, oral cavity and pharynx (oral cavity), and prostate, and ovary (carcinoma) fell into the epithelial cancer category; less than 1% of cases (other than lymphoma and soft tissue sarcoma) from these sites were non-epithelial according to their morphology codes, and these cases were excluded from their respective site-specific analyses.

Data analyses

Data from all registries were combined for the analyses. Relative frequencies and average annual age-specific cancer incidence rates per 100,000 person-year for selected cancers were computed for the five-year age groups from ages 15 through 49 years by gender and

race. Standard errors were calculated to evaluate statistical significance of the rate differences by age group, race, and gender [9]. Tests of significance for comparison were at a level of $p \leq 0.05$. The rates in figures were presented using uniform semi-logarithmic scales so that rates can be easily compared by race, gender, and age group. Rates and percentages were suppressed when fewer than 16 cases were reported in a specific cell. All analyses were conducted using SEER*Stat 5.0.17 software [10].

Results

This study included a total of 304,668 cancer cases newly diagnosed during 1995–1999 among 15 to 49-year-olds. The white persons accounted for 84.1% of the cases, black persons 11.4%, API 2.4%, other races 0.4% and unknown race 1.4%. Sixty-two percent of these cases were females and 38% were males.

Non-epithelial versus epithelial cancers

Among 15 to 19-year-olds, non-epithelial cancers accounted for 82% of all cases while epithelial cancers

accounted for only 18%. With increasing age, the percentage of non-epithelial cancers steadily decreased, whereas the percentage of epithelial cancer increased. After age 30, the percentage of epithelial cancers exceeded the percentage of non-epithelial cancers (data not shown). Among 45 to 49-year-olds, only 19% of cancers were non-epithelial, while 81% were epithelial. For all races combined, the shift from a predominance of non-epithelial cancers to a predominance of epithelial cancers occurred at a younger age among females (ages 25–29) than among males (ages 40–44) (data not shown). This phenomenon was observed among both white and black persons (Figure 1). Within the same gender group, the crossover occurred slightly earlier among black persons than among white persons, especially among males.

For all races combined, age-specific incidence rates increased with age for both non-epithelial and epithelial cancers (Figure 2). However, the slopes of the age-incidence curve were steeper for epithelial cancer than for non-epithelial cancer. Overall, the rates of non-epithelial cancers were higher among males than among females from ages 25–29 through ages 45–49. In contrast, the rates of epithelial cancers were lower among males than among females. Non-epithelial can-

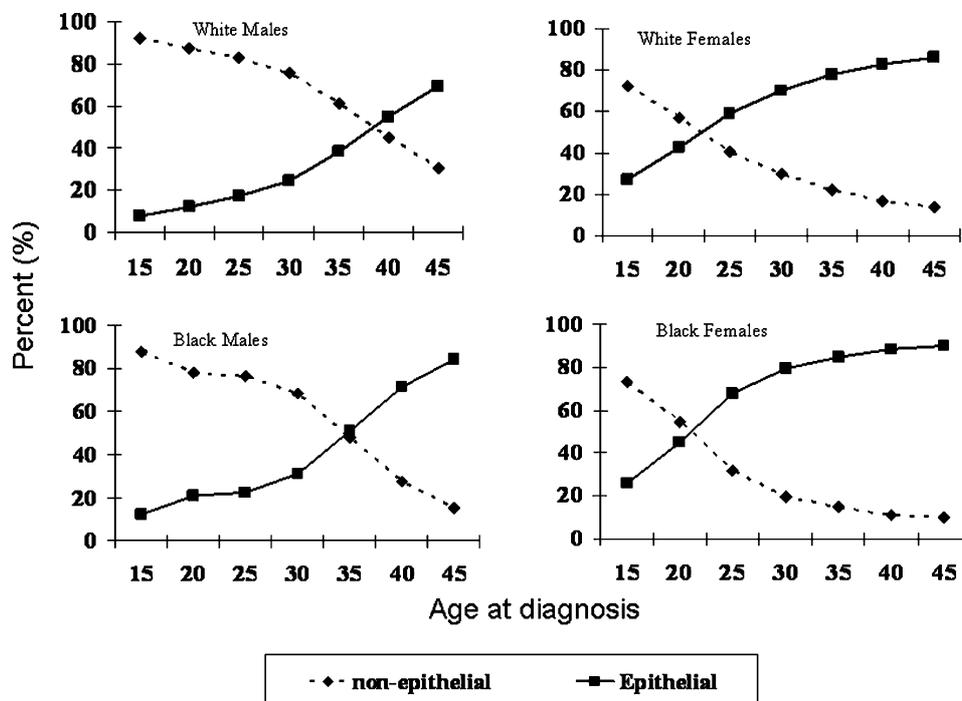


Fig. 1. Percentages of non-epithelial and epithelial cancers by race, gender, and age group in selected US areas, 1995–1999. Source: North American Association of Central Cancer Registries 1995–1999 analytic data file including Colorado, Connecticut, Atlanta (Georgia), Hawaii, Iowa, Idaho, Illinois, Kentucky, Louisiana, Michigan, Minnesota, Nebraska, New Jersey, New Mexico, North Carolina, Pennsylvania, Rhode Island, Utah, Seattle/Puget Sound (Washington), West Virginia, Wisconsin, and Wyoming.

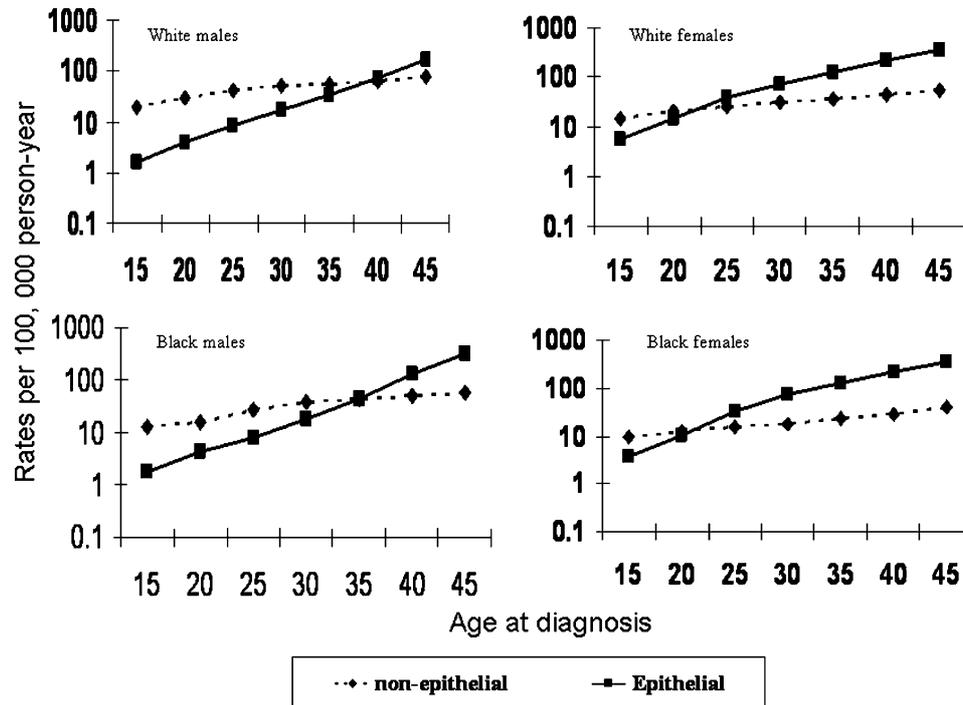


Fig. 2. Age-specific incidence rates for non-epithelial and epithelial cancers by race and gender in selected US areas, 1995–1999. Source: North American Association of Central Cancer Registries 1995–1999 analytic data file including Colorado, Connecticut, Atlanta (Georgia), Hawaii, Iowa, Idaho, Illinois, Kentucky, Louisiana, Michigan, Minnesota, Nebraska, New Jersey, New Mexico, North Carolina, Pennsylvania, Rhode Island, Utah, Seattle/Puget Sound (Washington), West Virginia, Wisconsin, and Wyoming.

cer rates were lower among black persons than among white persons, regardless of gender, across almost all age groups studied. The rates of epithelial cancers, by contrast, were higher among black males than among white males from ages 30–34 through ages 45–49. For females, the rates of epithelial cancers were about the same among white and black persons.

Changes in the five most common cancers from ages 15–19 through ages 45–49

Among white males in their late teens (ages 15–19), the five most common cancers were HL, testicular cancer, leukemia, brain & CNS tumors, and NHL (Table 1). After age 20, melanoma of the skin supplanted leukemia among the five most common cancers. From the early 20s (ages 20–24) through the early 30s (ages 30–34), the five most common cancers among white males remained the same although the relative frequency of each cancer varied with age. Colorectal cancer replaced HL among the top five cancers in the late 30s (ages 35–39); lung cancer supplanted brain & CNS tumors in the early forties (ages 40–44); and prostate cancer edged out testicular cancer in the late forties (ages 45–49), by which time the five most common sites were cancers of

the lung, colon & rectum, and prostate, followed by melanoma of the skin and NHL.

Among black males, the five most common cancers in each age group differed from those among white males (Table 1). Both Kaposi's sarcoma (data not shown) and all other STS occurred more commonly among black males than among white males. The opposite pattern was observed for testicular cancer. Epithelial malignancies such as colorectal, lung, and oral cavity cancers, began to occur at earlier ages among black males compared with white males.

The five most common cancers among females were different from those in males (Table 1). Epithelial malignancies such as cancers of the breast, cervix uteri, ovary (carcinoma), and thyroid began to occur in the early twenties and increased sharply thereafter. Although the five most common cancers were similar among white and black females across age groups, ovarian carcinoma was more common among white females and STS (excluding Kaposi's sarcoma) was more common among black females. Compared with white females, breast cancer was more common and occurred at an earlier age among black females. Among black females, colorectal cancer was one of the five most common cancers after age 30, and lung cancer joined the

Table 1. Average annual age-specific incidence rates per 100,000 person-year for all cancers combined and top five cancer types by age group, race and gender in the selected U.S. areas, 1995–1999 shaded = non-epithelial cancers; non-shaded = epithelial cancers

	Ages 15–19	Ages 20–24	Ages 25–29	Ages 30–34	Ages 35–39	Ages 40–44	Ages 45–49
White males							
All cancers	20.6	All cancers	32.5	All cancers	50.8	All cancers	67.7
HL	3.5	Testis	9.4	Testis	15.2	Testis	16.6
Testis	3.2	HL	5.3	HL	5.9	Melanoma	7.8
Leukemia	2.9	Melanoma	2.8	Melanoma	5.5	NHL	7.1
Brain & CNS	2.2	Brain & CNS	2.7	NHL	4.3	HL	5.0
NHL	2.1	NHL	2.5	Brain & CNS	3.5	Brain & CNS	4.7
Black males							
All cancers	14.1	All cancers	20.1	All cancers	34.3	All cancers	56.9
Leukemia	2.2	HL	3.6	NHL	5.3	NHL	10.9
HL	2.1	NHL	3.2	HL	5.1	Kaposi's	9.8
NHL	2.0	Soft Tissue ^a	2.6	Kaposi's	4.3	Colon & rectum	4.8
Soft Tissue ^a	1.8	Leukemia	2.1	Soft Tissue ^a	3.4	Testis	3.6
Brain & CNS	1.6	Testis	1.3	Leukemia	3.0	Soft Tissue ^a	3.5
White females							
All cancers	19.9	All cancers	35.3	All cancers	64.1	All cancers	102.6
HL	4.0	Thyroid	7.0	Thyroid	11.4	Breast	25.6
Thyroid	3.0	Melanoma	5.5	Melanoma	9.0	Thyroid	13.9
Leukemia	1.9	HL	5.4	Cervix uteri	8.4	Cervix uteri	13.4
Melanoma	1.9	Cervix uteri	2.2	Breast	7.7	Melanoma	11.7
Brain & CNS	1.6	Ovary (carcinoma)	2.1	HL	5.3	Ovary (carcinoma)	5.3
Black females							
All cancers	13.7	All cancers	23.6	All cancers	48.5	All cancers	92.3
HL	1.8	HL	3.5	Breast	11.8	Breast	35.3
Soft Tissue ^a	1.8	Cervix uteri	2.2	Cervix uteri	7.5	Cervix uteri	13.6
Leukemia	1.3	Breast	2.0	Thyroid	4.5	Thyroid	5.5
Brain & CNS	1.2	Soft Tissue ^a	2.0	HL	3.1	NHL	4.7
NHL	0.9	NHL	2.0	Soft Tissue ^a	3.0	Colon & rectum	4.5

^a Soft tissue sarcoma category does not include Kaposi's Sarcoma.

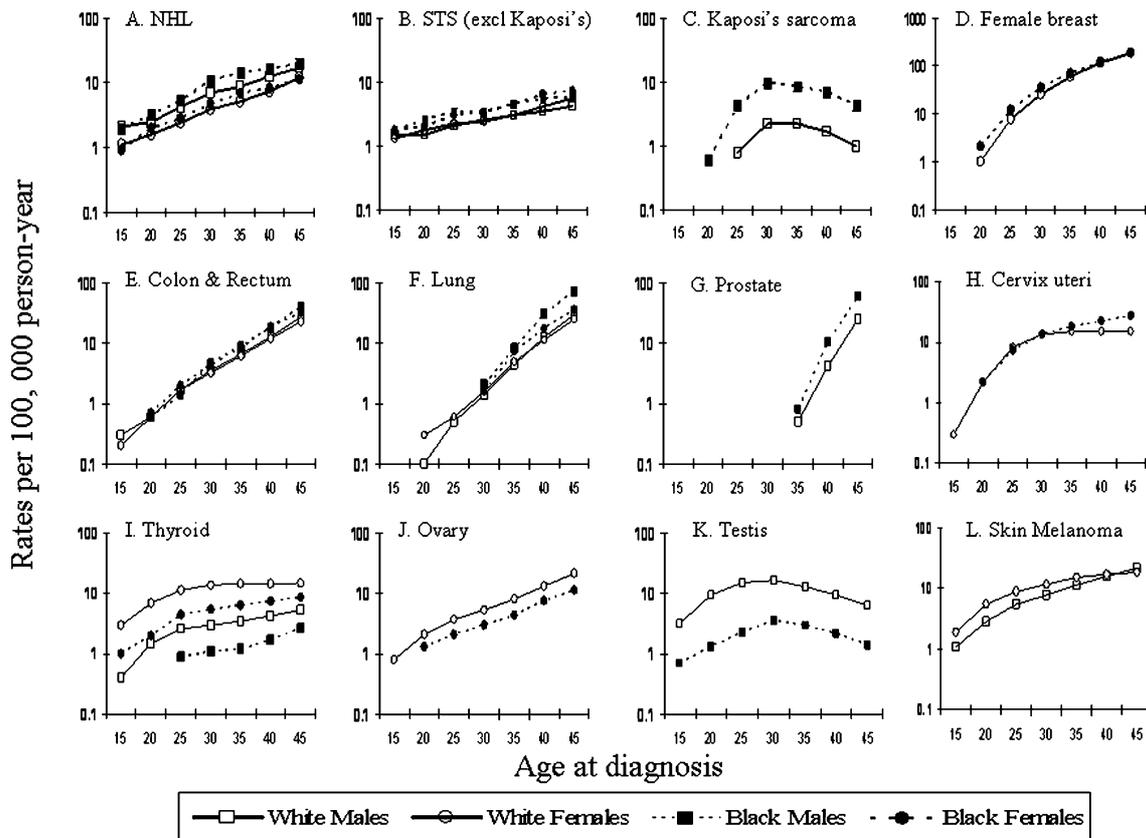


Fig. 3. Average annual age-specific incidence rates by race and gender for selected cancers in selected US areas, 1995–1999. Source: North American Association of Central Cancer Registries 1995–1999 analytic data file including Colorado, Connecticut, Atlanta (Georgia), Hawaii, Iowa, Idaho, Illinois, Kentucky, Louisiana, Michigan, Minnesota, Nebraska, New Jersey, New Mexico, North Carolina, Pennsylvania, Rhode Island, Utah, Seattle/Puget Sound (Washington), West Virginia, Wisconsin, and Wyoming.

top five sites after age 35; neither colorectal nor lung cancers were among the five most common sites for white females until after age 45.

Race and gender disparities in cancer incidence

Higher rates of NHL, STS, and Kaposi's sarcoma among blacks

Incidence of NHL increased progressively with age in all four race and gender groups (Figure 3). The rates were significantly higher among males than females, regardless of race, across all age groups studied. Although NHL rates among black males exceeded those among white males beginning in the twenties, the racial differences became statistically significant only after age 30. Black females likewise had higher NHL incidence rates than white females, except among those in their late teens and late forties.

Incidence of STS (excluding Kaposi's sarcoma) increased progressively with age. Between the ages of 20 and 49, STS rates were significantly higher among black

males than among whites of either sex. Kaposi's sarcoma rarely occurred among females. Among males, Kaposi's sarcoma incidence rates increased in the late twenties, peaking in the early thirties and decreasing thereafter. Incidence of Kaposi's sarcoma was statistically significantly higher among black males than among white males.

Earlier occurrence of common adult epithelial cancers among blacks

Cancers of the female breast, colon & rectum, and lung began to occur at an earlier age and increased more sharply among blacks than among whites (Figure 3).

Breast cancer rates were significantly higher among black females than among white females from the early twenties (2.1 *versus* 1.0 cases per 100,000 person-year) through the late thirties (69.0 *versus* 59.4 cases per 100,000 person-year); the racial disparity narrowed thereafter.

Colorectal cancer rates were significantly higher among black persons than among white persons in their 30s and 40s, regardless of gender. The rates among black

females were significantly higher than the rates among white males and females between the ages of 35 and 49. Although males had higher rates than females, regardless of race, this gender difference was statistically significant only after age 45.

Lung cancer rarely occurred before age 35, but increased rapidly thereafter. For persons in their late thirties, the lung cancer incidence rate among black males was about 87% higher than among white males, 68% higher than among white females, and 12% higher than among black females. Black males had the steepest age-specific lung cancer incidence curves. By ages 45–49, the lung cancer rate among black males was 138% higher than among white males, 183% higher than among white females, and 102% higher than among black females. All differences in lung cancer incidence between black males and the other groups were statistically significant in the age groups after 30 years of age.

Prostate cancer rarely occurred before age 40, but increased sharply thereafter, rising much faster among black males than among white males (Figure 3). The incidence rate for prostate cancer per 100,000 person-year among black males in their early forties was 157% higher than among white males and in the late forties, the rate among black males remained 136% higher than among white males. These racial differences in incidence of prostate cancer were statistically significant.

Higher invasive cervical cancer incidence rate among blacks over 35 years of age

Between the ages of 15 and 34, the rates of cervical cancer increased in parallel among white and black females. Cervical cancer incidence leveled off in the late thirties and forties among white females, but continued to increase among black females, such that the black–white disparity increased from 18% in the late 30s to 84% in the late forties.

Higher rates of thyroid cancer, ovarian carcinoma, and testicular cancer among whites

White females had the highest rate of thyroid cancer, followed by black females, white males, and black males in all age groups studied. Thyroid cancer incidence nearly quintupled among white females between the late teens (3.0) and early 30s (14.0), leveling off at approximately 15 per 100,000 person-year thereafter. Thyroid cancer rates among the other three race and gender groups increased gradually with age.

Incidence of ovarian carcinoma increased with age for both whites and blacks. However, the rates increased much faster among white persons than among black persons between the late teens and the late 40s. The white *versus* black rate ratio of ovarian carcinoma

increased from 1.6 in the early 20s to 1.92 in the late 40s. The differences in the rate ratio became statistically significant after age 25.

Testicular cancer incidence was approximately five times higher among whites than among blacks in all age groups studied. Using the late teen years as a baseline, testicular cancer incidence rates in males of both races first quintupled, peaking in the early 30s, then decreased to only twice the baseline level by the late 40s.

Higher cutaneous melanoma rates among white females

Cutaneous melanoma rarely occurred among black persons. Among white persons, melanoma rates increased with age. Cutaneous melanoma incidence rates were higher among white females than among white males across every age group studied; the difference was statistically significant for every age group except the early 40s.

Discussion

Our data reveal a shift from a predominance of non-epithelial cancers to a predominance of epithelial cancers in adolescence and early adulthood. This result is consistent with the findings reported by Miller *et al.* using 1973–1982 SEER data [3]. Differences in age patterns for epithelial and non-epithelial cancers may provide clues to the different etiologies of these classes of cancer. Previous studies have suggested that non-epithelial cancer is more likely to be related to viral infection, radiation, genetic conditions, and environmental chemical carcinogens [11–12]. In contrast, epithelial cancer is more likely to be related to lifestyle factors such as tobacco use, alcohol consumption, and dietary patterns. Obviously, the influence of lifestyle and diet on cancer risk among children is less evident than that among adults. Even though exposure to epithelial cancer risk factors may begin in adolescence, clinical manifestation may not emerge until adulthood because of latency [4]. However, family history of cancers and genetic predisposition may shorten the latency and increase likelihood of early onset of epithelial cancers [13–16].

Gender differences in the age at the shift from a preponderance of non-epithelial cancers to a preponderance of epithelial cancers were observed in our study. This shift occurred at a younger age among females than among males. This phenomenon primarily reflected sharper increases in the rates of epithelial cancers among females than among males in early 20s such as carcinomas of the thyroid, breast, ovary, and cervix uteri and high testicular cancer rate among young men.

After gender-specific cancers and thyroid and breast cancers were excluded, the proportions of epithelial and non-epithelial cancers were about the same among males and females across age groups. Early occurrence of these cancers may be related to physiological changes and exposure to risk factors. For example, previous studies have suggested that ovulation and gonadotrophic hormonal levels may contribute to the age-related progression from the predominance of germ cell ovarian cancer to the predominance of ovarian carcinoma among adolescents and young adults [17–22]. The sharp increase in thyroid cancer incidence among females beginning at ages 15–19 may be associated with puberty, pregnancy, and menstrual cycles because these stimulants of thyroid function might subject thyroid gland to increases in cell replication [23–24]. Primitive germ cells persisting into the pubertal period are thought to play a role in testicular cancer development. Because elevated testosterone is considered to provide a stimulus for progression of primitive germ cells to mature germ cells, low testosterone levels may increase the risk of testicular cancer [25].

The age-specific incidence patterns for NHL, STS, and Kaposi's sarcoma among adolescents and young adults reflect age-related exposure to certain risk factors. Although the etiologies of most NHL are not well known [26], patients with acquired immunodeficiency secondary to human immunodeficiency virus (HIV), congenital or therapeutical immunodeficiency are at an elevated risk of both NHL and Kaposi's sarcoma [27–30]. Black persons had the highest incidence of AIDS among racial groups as of 1997 [31]. Particularly in the young adult age group, the higher incidence rates for NHL and Kaposi's sarcoma among black persons, especially among black males, than among white persons may reflect the corresponding racial disparity in the prevalence of AIDS [32]. However, racial discrepancies in age-specific incidence patterns may also suggest differences in risk factors and pathological mechanisms for NHL and Kaposi's sarcoma. Reasons for higher incidence rate of STS (other than Kaposi's sarcoma) among black persons than among white persons are not well known. Unfortunately, although numerous risk factors such as radiation, herbicide exposure, genetic predisposition, and immunologic defects are suspected to increase the risk of non-Kaposi's STS, none of these risk factors are well-established due to paucity of data based on this age group [33].

Unlike NHL, STS, and Kaposi's sarcoma, occurrence of testicular cancer, one of the non-epithelial cancers seems less related to external risk factors. The most established risk factor for testicular cancer is the presence of an undescended testis [34]. Family history,

prior testicular tumor, immunosuppression, and pesticide exposure are also associated with increased risk of testicular cancer [35–39]. The rarity of testicular cancer among black males has been well documented [2, 40]. However, the underlying reason why black males have a much lower testicular cancer incidence rate than white males is not well known. Henderson *et al.* postulated that increased testosterone levels among black women during early pregnancy compared with white women may protect against testicular cancer among black male offspring [41].

Most breast and colorectal cancers occur among the elderly. Early onset of these cancers is more likely to be related to family history and genetic predisposition. [13–14, 16, 42–46]. However, because race-specific data on family history and predisposition are scarce, the racial disparity in early onset of these cancers is not well understood. Given the fact that breast cancer can be detected at an earlier stage and most colorectal cancer can be prevented by removal of polyps, the high risk individuals among adolescents and young adults should be targeted for early cancer screening. Since black persons are more likely to be underserved in terms of access to health care, a focused intervention strategy may be required to ensure all high risk individuals are to be served equally [47–49].

Although overall, most lung cancer cases, and about 78% and 90% of subsequent deaths, are attributed to cigarette smoking [50], the attributable risk of lung cancer to tobacco smoking may vary by age due to duration of smoking, histologic types, genetic predisposition, and metabolic gene polymorphisms [51–53]. Compared with older people, lung cancer among young people are more often adenocarcinoma, which may be less closely related to smoking than squamous cell or small cell lung carcinoma and may be more likely to be associated with genetic predisposition to cancers [52, 54–56]. Nevertheless, the role of tobacco smoking in elevated lung cancer risk among young adults is evident [57–58]. US lung cancer death rates for young adults by state have been associated with a statewide summary tobacco control index [57–58]. Since the latency of lung cancer spans several decades, cases diagnosed among persons in their forties may generally be related to cigarette smoking prevalence among teens in the same birth cohort [58]. However, previous studies have revealed that black persons are less likely than white persons to start smoking in adolescence [59]. This finding appears to be inconsistent with the racial disparity in lung cancer incidence among young adults observed in this and previous studies. Lung cancer risk reflects the combined effects of cancer-predisposing genes, carcinogen exposures,

and protective factors. Genetic polymorphisms in metabolic enzymes, which activate most carcinogens and exert deleterious effects, may be associated with inter-individual variability in susceptibility to tobacco smoke [60–61]. Prevalence of deficient CYP2D6, levels of serum cotinine, and patterns of smoking behavior and nicotine pharmacokinetics and brand mentholation may be related to differences in lung cancer risk between white and black persons [62–64]. Previous studies have found that blacks are more likely to smoke mentholated cigarettes than whites. However, whether mentholated cigarettes can lead to a higher likelihood of lung cancer than non-mentholated brands is still controversial [65–67]. More research is needed to discover the reasons for the racial differences in association of smoking with lung cancer occurrence and the factors that increase the likelihood of early onset of lung cancer. Since tobacco smoking is known and preventable risk factor for lung cancer, effectively promote smoking cessation program among adolescents and young adults is very much needed.

Higher prostate cancer rates among black males compared to white males in their late forties have been reported in previous studies [2]. This difference may be related to true differences in prostate cancer risk as well as prostate cancer screening [68]. Since black males are known to have a higher risk of developing prostate cancer at younger ages, it is recommended that black males initiate prostate cancer screening at age 45 [69]. The differences in testosterone level between black and white men may contribute, at least in part, to the disparate rates of prostate cancer between black and white men [70–72].

Our analyses revealed a plateau in the cervical cancer incidence rate after age 35 among white females, while among black females the rate continued to increase with advancing age. This pattern has also been observed in the SEER data [2]. The reasons for the racial differences in the cervical cancer age-specific incidence curves after age 35 are not well known. Genital human papillomavirus (HPV) infection is a well-established risk factor for most cervical cancers [73–74]. The observed plateau may reflect continuing high exposure to the HPV among black women with increasing age and a better detection of *in situ* cervical cancer and subsequent treatment for the *in situ* lesions among white women than among black women [75].

We observed steeper increases in the incidence of thyroid and ovarian carcinomas among white persons than among black persons. These findings are consistent with previous published studies [2, 24]. The risk of thyroid cancer is associated with therapeutic irradiation

of the head or neck; benign thyroid disease such as goiter or nodules, and family history of thyroid cancer [76]. No published study has investigated the reasons for the black–white disparity in thyroid cancer risk. Reasons for the higher rate of thyroid cancer among white persons than among black persons remain obscure. Papillary microcarcinoma of the thyroid, the most common form, usually remains clinically silent until it is identified at autopsy [77]. Autopsy rates vary from 3% to 36% throughout the world. Although the incidence of clinical thyroid cancer is higher among females than among males, the prevalence of thyroid cancer at autopsy does not vary by age or gender. Because thyroid nodules are more likely to be found among people with greater utilization of medical care, the racial disparity in thyroid cancer incidence may be related to overdiagnosis among white persons.

Higher ovarian carcinoma incidence rates among white women have been reported by numerous previous studies [78–80]. Two previously published studies have investigated the known ovarian cancer risk factors in an effort to explain the lower risk of ovarian cancer among black females than among white females [80–82]. Those studies reported that although use of oral contraceptives, hysterectomy, and increasing parity may reduce the risk of ovarian carcinoma and family history of the disease has been associated with increased incidence, the distributions of these factors did not differ by race [83–84]. Family history, which has been associated with an increased risk of ovarian carcinoma among white females but not among black females, accounts for only a very small proportion of ovarian carcinoma, and therefore may not play a major role in the racial disparity. To date, reasons for the black–white difference in incidence rates of ovarian carcinoma are not well understood.

Higher cutaneous melanoma incidence among young women than among young men has been reported previously [2]. The opposite pattern has been observed among the elderly. The reasons for higher likelihood of early onset of the melanoma of the skin among young women than among young men are not well known. The evidence on a possible etiologic role of female hormones in the development of malignant melanoma is still contradictory [85–86]. Since a link between cutaneous melanoma and sun exposure has been clearly established, avoidance of direct sun exposure should be advocated.

In summary, the 15–49 year-olds represent a unique population in cancer occurrence. Common cancers vary with gender, race, and age among adolescents and young adults and are different from those in childhood and adulthood. Cancers occurring among young people

are more likely to be related to genetic predisposition and specific health behaviors among young people. Therefore, cancer prevention and control strategies should be targeted accordingly and tailored to their specific needs.

Acknowledgements

This is the work of the Comparative Analysis of Incidence Rates Subcommittee (CAIR) of the Data Evaluation and Publication Committee (DEPC) of the North American Association of Central Cancer Registries (NAACCR). The authors are also grateful to Dr. John Young and Dr. Bernardo Ruiz for their advice on classification of non-epithelial and epithelial cancers. The authors also thank Elizabeth Hamilton-Byrd and Lisa M. Roche for their contributions to this manuscript.

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