A Midpoint Assessment of the American Cancer Society Challenge Goal to Decrease Cancer Incidence by 25% Between 1992 and 2015

Rebecca L. Sedjo, PhD; Tim Byers, MD, MPH; Ermilo Barrera, Jr., MD; Carmel Cohen, MD; Elizabeth T. H. Fontham, MPH, DrPH; Lisa A. Newman, MD, MPH; Carolyn D. Runowicz, MD; Alan G. Thorson, MD; Michael J. Thun, MD, MS; Elizabeth Ward, PhD; Richard C. Wender, MD; Harmon J. Eyre, MD; for the ACS Cancer Incidence & Mortality Ends Committee

ABSTRACT In 1998, the American Cancer Society (ACS) set a challenge goal for the nation to reduce cancer incidence by 25% over the period between 1992 and 2015. This report examines the trends in cancer incidence between 1992 and 2004. Trends were calculated using data on incident malignant cancer cases and population estimates from the Surveillance, Epidemiology, and End Results (SEER) Registry. Delay-adjusted incidence trends for all cancer sites; all cancer sites without prostate cancer included; all cancer sites stratified by gender, age, and race; and for 20 selected cancer sites are presented. Over the first half of the ACS challenge period, overall cancer incidence rates have declined by about 0.6% per year. The greatest overall declines were observed among men and among those aged 65 years and older. The pace of incidence reduction over the first half of the ACS challenge period was only half that necessary to put us on target to achieve the 25% cancer incidence reduction goal in 2015. New understandings of preventable factors are needed, and new efforts are also needed to better act on our current knowledge about how we can prevent cancer, especially by continuing to reduce tobacco use and beginning to reverse the epidemic of obesity. (CA Cancer J Clin 2007;57:326–340.) © American Cancer Society, Inc., 2007.

INTRODUCTION

In the United States, cancer is the second leading cause of death across all ages, but it is the leading cause of death for individuals under age 85 years.1 In 1996, the American Cancer Society (ACS) Board of Directors set an ambitious challenge goal to reduce cancer mortality by 50% over the 25-year period of 1990 to 2015.2 In 1998, they then set a further challenge goal to reduce cancer incidence by 25% between the high point of 1992 and 2015.3 This report examines the trends over the time period of 1992 to 2004, the approximate halfway point of the ACS challenge goal period.
and End Results (SEER) 9 Registry, using the Cancer Query System.4 SEER 9 is a set of cancer registries that includes approximately 10% of the US population, including Atlanta, Connecticut, Detroit, Hawaii, Iowa, New Mexico, San Francisco-Oakland, Seattle-Puget Sound, and Utah. Because cancers are sometimes reported to registries by physicians and medical facilities years after their initial diagnosis, delay-adjusted incidence rates were estimated to account for delays in reporting.5 Rates were examined for all cancer sites; all cancer sites without prostate cancer included (to examine trends without the effects of prostate-specific antigen [PSA] screening); all cancer sites stratified by gender, age (35 to 54, 55 to 64, and 65 to 84 years), and race/ethnicity; and for 20 selected specific sites (prostate, breast, lung, colorectal, corpus uteri, bladder, non-Hodgkin lymphoma, melanoma, ovary, leukemia, oral, pancreas, kidney, stomach, myeloma, liver, thyroid, brain, cervix uteri, and esophagus). All rates were age-adjusted to the 2000 US standard population (19 age groups).

### RESULTS

Over the time period of 1992 to 2004, overall cancer incidence rates declined by about 0.6% per year. Cancer incidence rates were about 8% lower in 2004 than they were in the year 1992. Increases in incidence in the early 1990s appeared to be due principally to a surge in prostate cancer incidence linked to the increasing use of PSA screening (Table 1). The declines in cancer incidence were most apparent among men and women.
A Midpoint Assessment of the American Cancer Society Challenge Goal to Decrease Cancer Incidence by 25% Between 1992 and 2015

Among those aged 65 years and older. Although decreasing trends were similar by race, incidence rates continue to be higher among African Americans than among Whites.

Over this 12-year period, reduction in cancer incidence has been observed for cancers of the prostate, lung (men), colorectum, ovary, oral cavity, stomach, and cervix uteri (Table 2, Figure 1). No overall downward trend was apparent for invasive breast cancer in women until after 1999, when a marked decline began. Incidence rates for lung cancer in women have stabilized in recent years. No appreciable downturn has been seen in multiple myeloma, non-Hodgkin lymphoma, leukemia, or in cancers of the corpus uteri, bladder, pancreas, or brain. Cancer incidence rates have increased for melanoma and cancers of the kidney, liver, thyroid, and esophagus.

There has been mixed success in reducing the prevalence of major cancer risk factors (Table 3). Tobacco use decreased substantially over the last 3 decades of the 20th century. Although the prevalence of smoking among US adults has remained fairly stable over the 14-year period of 1990 to 2004, the sale of cigarettes has continued to decline, as has the number of cigarettes smoked per day by continuing smokers.9,10 Obesity rates increased substantially between 1990 and 2004.11,12 The prevalence of obesity among women has remained constant between 1999 and 2004, while the rate of obesity among men has continued to increase.12 A substantial decline in the use of HT occurred after the 2002 publication of the Women’s Health Initiative trial reporting increased risk of breast cancer and cardiovascular disease due to HT using a particular combination of estrogen and progestin in postmenopausal women.8,13–15 The use of screening tests has been changing in recent years (Table 3). Screening for colorectal cancer has been increasing, with prevalence estimates of compliance with screening ranging from 48% in the National Health Interview Survey to 56% in the Behavioral Risk Factor Surveillance System.16,17 After continued

### TABLE 2  Trends in Delay-adjusted and Age-adjusted* Cancer Incidence Rates in the United States by Cancer Site, 1992 to 2004 (SEER 9 Registries)4

| Site               | 1992 | 1993 | 1994 | 1995 | 1996 | 1997 | 1998 | 1999 | 2000 | 2001 | 2002 | 2003 | 2004 | Change† |
|--------------------|------|------|------|------|------|------|------|------|------|------|------|------|------|---------|
| Prostate           | 237.5| 209.7| 180.5| 169.2| 169.4| 173.7| 171.0| 184.0| 183.2| 185.2| 182.5| 169.9| 165.3| –2.5    |
| Breast (females)   | 132.1| 129.3| 131.1| 132.7| 133.7| 137.9| 141.5| 141.4| 136.5| 138.2| 129.4| 125.9| 126.4| –0.4    |
| Lung (males)       | 97.2 | 94.0 | 91.0 | 89.8 | 88.1 | 86.4 | 88.2 | 84.7 | 82.1 | 81.1 | 80.2 | 78.4 | 75.8 | –1.8    |
| Lung (females)     | 49.9 | 49.2 | 50.6 | 50.5 | 51.2 | 52.7 | 53.0 | 52.6 | 51.3 | 51.6 | 52.3 | 53.0 | 52.0 | 0.4     |
| Colorectal         | 58.1 | 56.9 | 55.7 | 54.1 | 54.9 | 56.5 | 56.9 | 55.6 | 54.2 | 53.5 | 52.9 | 50.5 | 49.4 | –1.2    |
| Corpus uteri       | 24.8 | 24.3 | 25.2 | 25.4 | 25  | 25.9 | 25.8 | 25.4 | 24.9 | 25.3 | 24.9 | 24.2 | 24.8 | 0       |
| Bladder            | 21.3 | 21.3 | 20.8 | 20.7 | 20.8 | 21.1 | 21.7 | 21.9 | 21.9 | 21.7 | 21.3 | 21.4 | 21.2 | 0       |
| Non-Hodgkin lymphoma | 18.7 | 18.9 | 20.0 | 20.0 | 19.4 | 20.0 | 19.6 | 19.9 | 19.8 | 20.0 | 20.2 | 20.7 | 21.4 | 1.2     |
| Melanoma           | 14.8 | 14.6 | 15.7 | 16.5 | 17.3 | 17.8 | 18.0 | 18.3 | 19.0 | 19.7 | 19.3 | 19.3 | 20.4 | 3.2     |
| Ovary              | 14.9 | 15.2 | 14.5 | 14.6 | 14.2 | 14.7 | 14.3 | 14.7 | 14.3 | 14.5 | 13.7 | 13.6 | 13.2 | –1.0    |
| Leukemia           | 13.1 | 13.1 | 13.1 | 13.6 | 13.2 | 13.3 | 13.4 | 13.0 | 13.7 | 14.2 | 13.5 | 13.6 | 13.8 | 0.4     |
| Oral               | 12.0 | 12.5 | 11.7 | 11.6 | 11.8 | 11.5 | 11.3 | 10.6 | 10.8 | 10.8 | 11.1 | 10.4 | 10.7 | –0.9    |
| Pancreas           | 11.6 | 11.0 | 11.4 | 11.2 | 11.3 | 11.5 | 11.1 | 11.4 | 11.3 | 11.7 | 11.6 | 11.9 | 12.1 | 0.2     |
| Kidney             | 10.8 | 10.8 | 11.3 | 11.1 | 11.4 | 11.0 | 11.8 | 11.5 | 12.5 | 12.6 | 12.9 | 13.4 | 13.6 | 2.2     |
| Stomach            | 9.2  | 9.0  | 9.0  | 8.3  | 8.5  | 8.6  | 8.6  | 8.6  | 8.1  | 7.7  | 7.9  | 7.7  | 7.8  | –1.3    |
| Myeloma            | 5.9  | 5.6  | 5.7  | 5.8  | 6.1  | 5.9  | 5.6  | 6.1  | 6.0  | 6.0  | 6.1  | 5.8  | 5.8  | –0.1    |
| Liver              | 4.0  | 4.5  | 4.5  | 4.6  | 5.4  | 5.4  | 5.5  | 5.8  | 5.6  | 5.6  | 5.7  | 6.2  | 6.4  | 5.0     |
| Thyroid            | 5.9  | 5.6  | 6.1  | 6.2  | 6.5  | 6.8  | 7.0  | 7.4  | 7.6  | 8.3  | 9.2  | 9.6  | 10.0 | 5.8     |
| Brain              | 7.0  | 6.8  | 6.6  | 6.5  | 6.7  | 6.8  | 6.9  | 6.8  | 6.6  | 6.8  | 6.6  | 6.8  | 6.8  | –0.2    |
| Cervix uteri       | 10.0 | 9.7  | 9.5  | 8.9  | 9.7  | 9.3  | 9.2  | 8.3  | 7.8  | 8.0  | 7.4  | 7.3  | 7.2  | –2.3    |
| Esophagus          | 4.6  | 4.6  | 4.5  | 4.4  | 4.8  | 4.7  | 4.7  | 4.9  | 4.8  | 4.6  | 4.6  | 5.2  | 5.2  | 1.1     |

*Rates are per 100,000 and age-adjusted to the 2000 US standard population (19 age groups, Census P25–1130).
†The average percentage change per year is the mean percent change per year across the 12-year period, 1992 to 2004.
FIGURE 1  (continued on pages 330 and 331)
A Midpoint Assessment of the American Cancer Society Challenge Goal to Decrease Cancer Incidence by 25% Between 1992 and 2015

FIGURE 1  (continued on page 331)
increases in breast cancer screening by mammograms in the 1980s and 1990s, mammogram rates have declined somewhat in recent years, as indicated both by the Behavioral Risk Factor Surveillance System and other sources. Data are limited for PSA screening, but it seems that more than half of US men aged 50 years and older have been screened.

**DISCUSSION**

Cancer incidence during the time period from 1992 to 2004 has been decreasing, due mostly to a favorable trend among men and among those aged older than 65 years. However, there is considerable variation across cancer sites in both the magnitude and the direction of cancer incidence trends over this time period. Here we discuss the likely reasons for variations in incidence trends and speculate on the changes that can be expected between now and the year 2015. We first discuss trends by specific cancer sites and then by cross-cutting issues of tobacco, obesity, screening, and race/ethnicity.

**Cancer Sites**

**Prostate Cancer.** The prostate is the leading site for cancer incidence among US men. Prostate cancer incidence has been extremely variable in the past 20 years, largely due to the advent of PSA screening. A sharp increase in incidence began in the late 1980s, almost certainly due to the advent of PSA testing and the detection of a high number of previously undiagnosed, prevalent cases. Despite approximately 40% higher rates of prostate cancer incidence among African Americans as compared with Whites, declining trends are similar for both Whites and African Americans. Modifiable nutritional factors may also be important in prostate cancer, especially vitamin E and selenium. If the ongoing Selenium and Vitamin E Cancer Prevention Trial (SELECT) finds that either type of supplement can reduce prostate cancer risk, there could be benefits for lower incidence in the coming decade. Prostate cancer incidence can be reduced with finasteride chemoprevention, which inhibits the conversion of testosterone to dihydrotestosterone, but this medication has not been commonly used for this purpose because of concern about possible adverse effects leading to cancers with higher Gleason grades. Hopefully, findings from PSA screening trials will clarify the value of screening for reducing morbidity and mortality from prostate cancer, as well as the overall impact on duration and quality of life. The trends of declining prostate cancer incidence will be largely dependent on the rates of PSA testing in the years to come.
A Midpoint Assessment of the American Cancer Society Challenge Goal to Decrease Cancer Incidence by 25% Between 1992 and 2015

Breast Cancer. The breast is the leading site of cancer incidence in US women.1 Over the time period of 1992 to 1999, no substantial changes in overall incidence rates of invasive breast cancer were observed, but after 1999, breast cancer incidence began to decline. Initially the decline was likely due to the saturation of mammography screening, which had already identified many prevalent early breast cancers, ultimately impacting treatment and future mortality trends.18–19 This decline steepened substantially after 2002, primarily due to declines in estrogen receptor-positive tumors among women aged 50 to 69 years.26 This steeper decline since 2002 may be due to the combined effects of a decline in the rate of mammography screening and the sudden decline in HT following the 2002 publication of the Women’s Health Initiative for combined estrogen and progestin.13,26,27 Both of these factors will cause a continued decline in breast cancer incidence in the coming years. The obesity epidemic has had adverse effects on breast cancer incidence trends. Without past increases in obesity, incidence declines might have been steeper and seen much earlier. According to the National Health and Nutrition Examination Survey, there has been no significant increase in prevalence of obesity among women between 1999 to 2000 (33.4%) and 2003 to 2004 (33.2%).12 Despite this suggestion that obesity might be stabilizing among women, the current prevalence of overweight and obesity is still quite high (62%).12 Weight gain and excess adiposity are important modifiable risk factors for postmenopausal breast cancer.28 Therefore, if the obesity epidemic can be slowed and reversed in the coming decade, this could cause additional reductions in future breast cancer incidence. Future declines in breast cancer incidence may also be seen as the consequence of removal of atypical hyperplasia and ductal carcinomas in situ that were identified as suspicious lesions by mammography in past years. Tamoxifen and raloxifene have both been shown to substantially reduce the risk of incident breast cancer.29,30 The safety profile for tamoxifen discourages its widespread use, but raloxifene seems to have a better balance of risks and benefits.30 Raloxifene, currently used in the prevention and treatment of osteoporosis, was prescribed in only 12% of patient visits for osteoporosis in 2003.31 If the use of raloxifene increases substantially in the coming years, breast cancer incidence may be expected to fall. In the coming decade, the longer-term effects of decreased use of HT, increased chemoprevention, and slowing of the obesity trends should lead to continued decreases in breast cancer incidence rates.

| TABLE 3 | Trends in Cancer-related Risk Factors and Cancer Screening Practices, 1990 to 2004 |
|---------|------------------------------------------------------------------------------------------------|
|         | 1990 | 1991 | 1992 | 1993 | 1994 | 1995 | 1996 | 1997 | 1998 | 1999 | 2000 | 2001 | 2002 | 2003 | 2004 |
| Smoking (men)* | 24.9 | 25.1 | 24.2 | 24.0 | 23.9 | 24.8 | 25.5 | 25.4 | 25.3 | 24.2 | 24.4 | 25.4 | 25.7 | 24.8 | 23.0 |
| Smoking (women)* | 21.3 | 21.3 | 21.0 | 21.1 | 21.6 | 20.9 | 21.9 | 21.1 | 20.9 | 20.8 | 21.2 | 21.2 | 20.8 | 20.2 | 19.0 |
| Obesity (men)* | 11.5 | 12.2 | 12.3 | 13.2 | 14.7 | 16.3 | 16.3 | 17.1 | 18.4 | 19.9 | 20.6 | 21.2 | 23.1 | 23.1 | 23.6 |
| Obesity (women)* | 11.5 | 12.7 | 12.7 | 13.9 | 14.6 | 14.7 | 16.8 | 16.5 | 18.3 | 19.7 | 19.8 | 20.8 | 21.4 | 22.1 | 22.5 |
| Hormone therapy, millions† | 10 | 13 | 15 | 15 | 10 |
| Colorectal screening‡ | 29.4 | 32.4 | 43.7 | 48.1 | 53.0 |
| Mammograms§ | 58.3 | 62.2 | 63.1 | 66.5 | 66.6 | 69.2 | 70.3 | 72.3 | 72.8 | 76.1 | 75.9 | 74.7 |
| Prostate-specific antigen¶ | 53.9 | 52.1 |

*Median percent across all states using data from the Behavioral Risk Factor Surveillance System Survey Data.7
†Hormone therapy is the estimate of sales to women, in millions of women receiving prescriptions. Data from Hersh AL, Stefanick ML, Stafford RS.8
‡Based on questions from the Behavioral Risk Factor Surveillance System Survey for adults aged 50 years and older: Have you ever had a proctoscopic exam? (1995); Have you ever had a sigmoidoscopy or proctoscopic exam? (1997); Have you ever had a sigmoidoscopy or colonoscopy? (1999, 2002, and 2004).
§Female respondents aged 40 years and older who report that they had a mammogram in the past 2 years.
¶Men aged 40 years and older who have had a prostate-specific antigen test within the past 2 years (2002 and 2004).
Lung Cancer. The lung is the second leading site for cancer incidence and the leading site for cancer death among both US men and women.\(^1\) Lung cancer incidence rates are approximately 1.7 times higher in men than in women. The downward trend of lung cancer incidence in men is exceeding the 25% reduction goal, but the trends among women are not (see Figure 1). The primary cause of lung cancer is tobacco use, so incidence trends are largely a reflection of tobacco-use trends over the preceding 20-year period.\(^{32,33}\) The prevalence of smoking declined from 52% to 33% among men and from 34% to 28% among women during the time period from 1965 to 1985. Between 1985 and 1995, there was about a 5% decrease in the prevalence of tobacco use among both men and women.\(^34\) Despite a persistently higher rate of lung cancer among African Americans than among Whites, a steeper decline has been observed for African Americans compared with Whites over this time period, likely due to historical changes in smoking. Declines in lung cancer incidence have also been observed due to reductions in occupational carcinogen exposures; however, the relative contribution to overall lung cancer rates of these exposures is small compared with tobacco use.\(^{32}\) Screening is not recommended for lung cancer in the general population, but low-dose computed tomography (CT), chest x-ray, sputum cytology, molecular sputum testing, or a combination of these tests are still under investigation and, therefore, may hold promise.\(^{24,35-40}\) Promising findings for screening with spiral CT scans have led to the implementation of large randomized clinical trials (RCTs) now underway that will be completed by 2010.\(^37\) Apart from possible effects of screening, the incidence rates for lung cancer will likely decline in the coming decade as a consequence of past tobacco trends. If CT screening begins to be used widely, then incidence rates will substantially increase as an artifact of the initiation of screening and the detection of prevalent cases, as was observed for prostate cancer in the early 1990s. Apart from this potential artifact of screening, the major factor that will determine lung cancer incidence in the coming decade is the past history of tobacco use. Incidence will, therefore, likely continue to decline among men and soon begin to decline among women.

Colorectal Cancer. Modifiable risk factors associated with the development of colorectal cancer include physical inactivity; adiposity; cigarette smoking; and diets high in red meats, processed meat, or high energy intake, while preventive factors include the use of nonsteroidal anti-inflammatory drugs (NSAIDs); HT; and diets high in fruits, vegetables, calcium, and/or vitamin D.\(^41\) Colorectal cancer incidence rates increased until 1985, when they began to decline.\(^32\) The reasons for this decline are not clear, but could be tied to downward trends in cigarette smoking, increasing NSAID use, and increasing HT use. The recent decline in HT use may adversely affect colorectal cancer trends among women in the coming years, as HT reduces risk.\(^13\) Recent trials have demonstrated the potential for NSAIDs to reduce colorectal adenomas, but adverse effects from these agents will limit their widespread use for that purpose.\(^43,44\) Colorectal screening (especially colonoscopy and flexible sigmoidoscopy) leads to the identification and removal of adenomas, thus substantially reducing the risk of incident colorectal cancer.\(^55\) Colorectal screening rates (mostly colonoscopy) have been increasing in recent years (Table 3). Colorectal screening is higher among Whites than among African Americans,\(^46\) which may help explain the steeper decline observed among Whites. The current rate of decline in the incidence of colorectal cancer is on target to meet the 25% reduction challenge goal for 2015. This favorable trend in colorectal cancer incidence is occurring in spite of the obesity epidemic. If obesity trends can be improved and if we can continue progress in the use of colonoscopy for colorectal screening, the reduction in incidence of colorectal cancer may well exceed the 25% goal.

Uterine Cancer. The primary risk factor likely to affect the current trends in endometrial cancer is obesity.\(^47,48\) Obesity likely influences endometrial cancer risk in premenopausal women by causing androgen excess with altered ovarian physiology, whereas in postmenopausal women, adipose tissue increases estrogen production through the conversion of androgens to estrogens by aromatase.\(^49\) Another important risk factor for uterine cancer is the use of unopposed estrogens in HT.\(^50\) It is likely that the sharp
declines in the use of systemic unopposed estrogen after 2002 have led to only a small decrease in incidence, as probably only a small number of women who have not had hysterectomies would have been taking unopposed estrogen. However, a recent report from the Women’s Health Initiative suggests a lower prevalence of coronary artery calcification among postmenopausal women aged 50 to 59 years who had undergone hysterectomy and received conjugated equine estrogen as compared with those who received placebo. These findings, along with earlier findings suggesting that combined HT confers greater postmenopausal breast cancer risk than estrogen alone, may lead to an increase in the use of unopposed estrogen therapy in women with uteri, thus increasing their risk of endometrial cancer. Surveillance of specific prescribing factors for HT should be monitored. Overall, the high obesity prevalence may influence future trends. Therefore, the most reasonable prediction would be a continued stable rate of uterine cancer.

Bladder Cancer. Bladder cancer is the fourth leading cancer among US men. The primary modifiable risk factors for bladder cancer are cigarette smoking and occupational exposures to carcinogens. However, the anticipated decline in incidence of bladder cancer due to past reductions in tobacco use and occupational exposures has not been seen over the time period of 1992 to 2004. The reasons why bladder cancer rates have remained unchanged are not clear, hence it is not possible to confidently predict changes in the coming decade.

Non-Hodgkin Lymphoma. Very little is known about modifiable risk factors for non-Hodgkin lymphoma other than HIV/AIDS, Epstein-Barr virus, herpes virus 8, human T-cell lymphotropic virus, and immunosuppressive drugs. Potential roles of other infectious agents and occupational and environmental factors such as benzene or polychlorinated biphenyls, as well as others, are unclear. The most reasonable prediction for the coming decade is, therefore, continuation of the past trend of an increase in rates.

Melanoma. Melanoma rates have been increasing substantially in recent years. This is likely due to the combined effects of previous sun exposures and increased diagnosis of very small cancers due to improved awareness and surveillance of pigmented lesions. There has been a modest 8% increase in sunscreen use among adolescents from 1998 to 2004, but no significant decrease in reported sunburn in the previous summer (69% versus 72%). As most melanoma occurs in older people, it is likely rates will continue to increase into the coming decade resulting from past sun exposures.

Ovarian Cancer. The few modifiable risk factors that decrease risk include oral contraceptives, hysterectomy, tubal ligation, and high parity. In 1982, only 76% of US women had ever used the pill as compared with 82% in 1995 and 2002. Past trends in oral contraceptives may account for the observed declines in ovarian cancer incidence. There are currently no recommendations for screening in the general population, but 2 large screening trials are ongoing. Because the impact of oral contraceptive use on ovarian cancer risk persists for at least 15 years, recent trends in their use will likely lead to continued declines in ovarian cancer incidence in the coming decade.

Leukemia. Leukemia is diagnosed 10 times more frequently in adults, but it is the leading cause of cancer in children aged 0 to 14 years. Leukemia is comprised of several diverse types, with few having any identifiable modifiable risk factors. Nevertheless, occupational exposures, radiation exposure, chemotherapy, and smoking have all been implicated as risk factors for acute myeloid leukemia, the most frequently diagnosed leukemia. It is most reasonable, therefore, to predict that the unchanging rates will continue into the coming decade.

Cancers of the Oral Cavity and Pharynx. The primary modifiable risk factor for oral cancer is tobacco exposure, either by smoking or chewing. Alcohol is also a factor that works synergistically with tobacco. The decline in oral cancer between 1992 and 2004 is likely due to historical reductions in tobacco exposure. Continuing declines are likely in the coming decade.

Pancreatic Cancer. Cigarette smoking is the major risk factor for pancreatic cancers. The importance of other risk factors, including obesity and vegetable intake, is less certain. Despite declining cigarette exposures over the past several decades, rates of pancreatic cancer
have remained stable.\textsuperscript{70} This may be due in part to the more certain diagnosis of pancreatic cancer due to improved diagnostic imaging. Based on the past trends, no substantial change is likely in pancreatic cancer rates in the coming decade.

Kidney Cancer. Increased incidence of kidney cancer has been primarily attributed to small localized tumors identified in patients who undergo diagnostic evaluation for unrelated conditions.\textsuperscript{71} However, this may not completely explain the increased trend because other factors may also be contributing, including obesity trends,\textsuperscript{72} as obesity is an important risk factor.\textsuperscript{47,48} This rising trend in incidence has occurred in spite of decreasing prevalence of tobacco use, a major risk factor for kidney cancer.\textsuperscript{66} It is expected that the incidence of kidney cancer will continue to rise in the coming decade.

Stomach Cancer. The incidence of stomach cancer has been declining over the past several decades in the United States.\textsuperscript{73} Declining rates in the historical trends of stomach cancer have been attributed to the nutritional benefits coming from improved food storage and distribution systems and to the declining prevalence of smoking and chronic infection with \textit{Helicobacter pylori}.\textsuperscript{74,75} It is likely the long-term historical decline in stomach cancer will continue into the coming decade due in part to declines in smoking prevalence and to declines in the prevalence of persistent \textit{Helicobacter pylori} infection initiated in childhood among younger cohorts.

Myeloma. Because the etiologic factors for myeloma are not well understood,\textsuperscript{76} it is most reasonable to project the future rates will remain stable.

Liver Cancer. Liver cancer incidence has been substantially increasing in the past decades.\textsuperscript{77–79} The primary risk factors for liver cancer include chronic infection with hepatitis B virus and hepatitis C virus, along with excess alcohol and obesity.\textsuperscript{77–79} The risk of liver cancer is much higher among foreign-born persons, particularly Asian/Pacific Islanders, due to their high rates of chronic infection with hepatitis B virus.\textsuperscript{80} Despite the initiation of universal infant/childhood hepatitis B vaccination programs for children in the past 20 years,\textsuperscript{81} only limited effects on hepatocellular carcinoma will be observed by 2015. Based on immigration trends and the epidemic of hepatitis C virus infection, prediction models suggest that liver cancer will continue to rise in the United States over the coming 15 years.\textsuperscript{82,83}

Thyroid Cancer. The incidence of thyroid cancer has been increasing in the United States for the past several decades, primarily due to an increase in small papillary cancers.\textsuperscript{84} While the increase in small papillary cancers is most likely due to increased detection from improved medical imaging and diagnostic techniques, it is uncertain whether there are any other reasons contributing to this trend.\textsuperscript{84,85} With increasing numbers of people undergoing neck ultrasound examinations, this trend will likely continue into the future.

Cancers of the Brain and Nervous System. The incidence of brain cancer increased in the 1980s, perhaps due to the advent of better imaging methods for the brain, but rates have been steady in recent years. As little is known about the etiologic factors for brain cancer, it is not possible to confidently predict the trends in the coming decade.\textsuperscript{86}

Cervical Cancer. Invasive cervical cancer is uncommon in the United States because of widespread screening using Pap smears that identify and remove precursor lesions.\textsuperscript{87} In 2006, the Food and Drug Administration approved a human papillomavirus vaccine for use in girls and women aged 9 to 26 years.\textsuperscript{88} This vaccine has been shown to be highly effective in protecting against the human papillomavirus serotypes that together cause about 70% of cervical cancer cases.\textsuperscript{89} Little effect on cervical cancer incidence due to the use of this vaccine will be observed before 2015. Nonetheless, given the historical declining trend and continued high prevalence of Pap smear screening,\textsuperscript{7} the United States has already surpassed the 25% reduction goal for cervical cancer, and incidence will likely continue to decline, but at a slower rate in the future, as no substantial changes in the prevalence of Pap smear screening are likely.

Esophageal Cancer. The overall incidence of esophageal cancer has remained fairly constant over the past 12 years. However, trends in incidence have been decreasing for the more common squamous cell carcinoma of the esophagus and increasing for adenocarcinoma of the esophagus.\textsuperscript{90,91} Declines in the rates of squamous cell carcinoma of the esophagus are likely due to declining rates of smoking and alcohol consumption, while
increases in adenocarcinoma of the esophagus are likely due to factors causing acid-reflux disorders of the lower esophagus, especially abdominal obesity. Due to the trends in tobacco and obesity, it is expected that the past trends of reductions in the more common squamous cell cancer will continue, and increases in adenocarcinoma of the esophagus will also likely continue into the coming decade.

CROSS-CUTTING ISSUES

Tobacco. Tobacco usage has been demonstrated to be either the primary or contributing factor for numerous cancers, including lung, oral, bladder, pancreas, esophagus, stomach, and many others. Declines in incidence and mortality for tobacco-related cancers have been observed following declines in smoking prevalence, primarily due to smoking cessation, between 5 and 20 years previously. Recently, the trends in current smoking prevalence have been stable (Table 3), though smokers are smoking fewer cigarettes per day. Recent leveling of tobacco-use prevalence raises concern that the downward trends in tobacco-related cancers may eventually flatten as well. Clearly, tobacco remains the most modifiable factor for cancer prevention, thus US public policy to further discourage tobacco use is the most important continuing opportunity to decrease future cancer incidence.

Obesity. Since the early 1980s, an epidemic of overweight and obesity has been occurring in the US population. While recent data suggest that this upward trend may be moderating among women, increases continue in men. Obesity increases the risk of cancer at multiple sites, including the endometrium, kidney, colon, postmenopausal breast, and adenocarcinoma of the esophagus. The effect of obesity is clear on trends in cancers such as adenocarcinoma of the esophagus and kidney. Despite increasing prevalence estimates of obesity, incidence of colorectal cancer has continued to decline, as has postmenopausal breast cancer. These declines would likely have begun sooner and would have been steeper if it were not for the obesity epidemic. If the obesity epidemic can be stabilized and reversed in the coming years, substantial reductions in incidence of obesity-related sites can be expected. Efforts should be directed to discourage weight gain and encourage weight loss among the US population to reduce risk of cancer, as well as other chronic diseases.

Screening. Screening for early cancer detection in the general population is currently recommended for breast, colorectal, and cervical cancers. Additionally, providing information to support informed choices about screening for prostate cancer is recommended. The relationship between screening and cancer incidence is complex. In some instances, screening can reduce incidence through the detection and successful treatment of precursor lesions, such as for cervical intraepithelial neoplasia and colorectal adenomas, thereby lowering cancer incidence. For prostate cancer, however, screening leads to increased cancer incidence through the identification of small cancers that might not become clinically apparent until years into the future, if ever. Thus, screening can reduce cancer mortality while, at the same time, increasing cancer incidence. The balance of risks and benefits from the identification of cancers by screening can only be properly assessed in randomized, controlled screening trials. Continued public health effort should be focused on continuing to obtain better information about the benefits and risks of screening, especially for prostate and lung cancers, and on achieving high screening rates in the general population for those cancers showing evidence-based screening benefits.

Race/Ethnicity. Racial and ethnic differences exist in the rates of cancer incidence in the United States. Compared with non-Hispanic Whites, African Americans experience higher incidence rates for cancers of the colorectum, lung, prostate, liver, kidney, and cervix, and Hispanics, Asians, and Pacific Islanders experience higher incidence of cancers of the liver, stomach, and cervix. Racial differences in cancer incidence and mortality rates are complex and poorly understood. Disparities in cancer incidence, survival, and mortality by race and ethnicity seem to be due to a combination of factors, including biological, behavioral, cultural, and economic, as well as structural factors in health care. Changes in the racial and ethnic demographics of the United States are likely to contribute to transforming future patterns of cancer incidence.
2001, 7% of non-Hispanic White adults aged 18 to 64 years lived below the poverty level, compared with 19% of African Americans and 16% of other minorities. Members of some racial and ethnic minority groups also tend to have less education and are less likely to have health insurance, As lack of insurance leads to lower utilization of screening programs and to less adequate treatment for cancer, it follows that public policy strategies encouraging health care system reforms to reduce barriers to clinical preventive services such as cancer screenings, tobacco cessation support, and nutritional counseling could have an important effect on disparities in future cancer incidence.

CONCLUSION

Over the first half of the ACS 25-year challenge period, the overall cancer incidence rate has declined by 0.6% per year. The greatest declines have occurred among men and among those men and women aged 65 years and older. Declines were greater among men principally because of declining rates of incidence of the major cancer sites of lung and prostate. The reason that declines were steeper among those aged 65 years and older is unclear, but may be related to the fact that long-term benefits of smoking cessation are more apparent with advancing age and to the wider access to clinical preventive services in the Medicare system, including colorectal and cervical screenings. While declining trends have been observed for some cancer sites, others have remained constant or increased over this time period. Historical declines in the use of tobacco and recent declines in the use of certain hormone therapies have contributed to incidence reductions in several cancer sites and will likely result in steeper declines in the decade to come. However, these favorable changes are somewhat offset by the increasing prevalence of obesity, which contributes to higher risk for many types of cancers. Higher incidence rates continue among African Americans as compared with Whites, likely due to differences in risk factors and access to care. The lack of substantial change in recent tobacco-use trends leading to a leveling in future tobacco-related cancer incidence and mortality trends and recent declines in mammography screening leading to an increase in future mortality trends are of concern.

During the first half of the challenge period, the pace of decline was only about half that needed to put us on target to reduce cancer incidence by 25% by the year 2015. Assuming a continuation of the current cancer incidence reduction trend over the past 12 years, we estimate that the ACS challenge goal of reducing cancer incidence by 25% from 1992 to 2015 may be only half achieved. Similarly, it has been estimated that the ACS challenge goal of 50% for cancer mortality from 1990 to 2015 may also be only half achieved. New understandings of the preventable factors causing cancer are needed, and new efforts are also needed to better act on our current knowledge about how to prevent cancer, especially in the areas of continuing the decline in the epidemic of tobacco use, reversing the epidemic of obesity, and assuring wider access to clinical preventive services.
REFERENCES

1. Jemal A, Siegel R, Ward E, et al. Cancer statistics, 2007. CA Cancer J Clin 2007;57:43–66.
2. American Cancer Society Board of Directors. ACS Challenge Goals for U.S. Cancer Mortality for the Year 2015. Proceedings of the Board of Directors, Atlanta, GA: American Cancer Society; 1996.
3. American Cancer Society Board of Directors. ACS Challenge Goals for U.S. Cancer Incidence for the Year 2015. Proceedings of the Board of Directors, Atlanta, GA: American Cancer Society; 1998.
4. National Cancer Institute, U.S. National Institutes of Health. Surveillance, Epidemiology, and End Results (SEER) Program (www.seer.cancer.gov). Delayed-Adjusted Incidence Database: SEER Incidence Delay-Adjusted Rates, 9 Registries, 1975–2004. National Cancer Institute, DCCPS, Surveillance Research Program, Statistical Research and Applications Branch, released April 2007, based on the November 2006 SEER data submission.
5. Clegg LX, Feuer EJ, Midthune DN, et al. Impact of reporting delay and reporting error on cancer incidence rates and trends. J Natl Cancer Inst 2002;94:1537–1545.
6. National Cancer Institute, U.S. National Institutes of Health. Surveillance, Epidemiology, and End Results (SEER) Program (www.seer.cancer.gov). SEER*Stat Database: Incidence—SEER 9 Regs Limited-Use, Nov 2006 Sub (1973–2004), Linked to Country Attributes—Total US, 1969–2004 Counties, National Cancer Institute, DCCPS, Surveillance Research Program, Cancer Statistics Branch, released April 2007, based on the November 2006 submission.
7. Centers for Disease Control and Prevention. Behavioral Risk Factor Surveillance System Survey Data, 1990–2004. Atlanta, GA: National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention; 2007. Available at: http://www.cdc.gov/brfss/. Accessed August 1, 2007.
8. Hersh AL, Stanislaw ML, Stafford RS. National use of postmenopausal hormone therapy: annual trends and response to recent evidence. JAMA 2004;291:147–53.
9. Dobson R. US cigarette consumption falls to lowest point since 1981. BMJ 2006;332:687.
10. Al-Delaimy WK, Pierce JP, Messer K, et al. The California Tobacco Control Program’s effect on adult smokers: (2) Daily cigarette consumption levels. Tob Control 2007;16:91–95.
11. Flegal KM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among US adults, 1999–2000. JAMA 2002;288:1723–1727.
12. Ogden CL, Carroll MD, Curtin LR, et al. Prevalence of overweight and obesity in the United States, 1999–2004. JAMA 2006;295:1549–1555.
13. Writing Group for the Women’s Health Initiative Investigators. Risks and benefits of estrogen plus progestin in healthy postmenopausal women: principal results from the Women’s Health Initiative randomized controlled trial. JAMA 2002;288:321–333.
14. The Women’s Health Initiative Steering Committee. Effects of conjugated equine estrogen in postmenopausal women with hysterectomy: the Women’s Health Initiative randomized controlled trial. JAMA 2004;291:1701–1712.
15. Bost DS, Newton KM, Miglioretti DL, et al. Hormone therapy prescribing patterns in the United States. Obstet Gynecol 2004;104:1042–1050.
16. National Center for Health Statistics, Centers for Disease Control and Prevention. National Health Interview Survey Public Use Data Files, 2003. Hyattsville, MD: National Centers for Health Statistics, Centers for Disease Control and Prevention; 2004.
17. Centers for Disease Control and Prevention. Behavioral Risk Factor Surveillance System Survey Data, Atlanta, GA: US Department of Health and Human Services, Centers for Disease Control and Prevention; 2004.
18. Centers for Disease Control and Prevention. Use of mammograms among women aged ≥40 years—United States, 2000–2005. MMWR Mortal Wkly Rep 2007;56:49–51.
19. Feldstein AC, Vogt TM, Aickin M, Hu WR. Mammography screening rates decline: a persons’ time approach to evaluation. Prev Med 2006;43:178–182.
20. Potosky AL, Miller BA, Albertsen PC, Kramer BS. The role of increasing detection in the rising incidence of prostate cancer. JAMA 1995;273:548–552.
21. Hsing AW, Chokkalingam AP. Prostate cancer epidemiology. Front Biosci 2006;11:1388–1413.
22. Lippman SM, Goodman PJ, Klein EA, et al. Designing the Selenium and Vitamin E Cancer Prevention Trial (SELECT). J Natl Cancer Inst 2005;97:94–102.
23. Thompson IM, Goodman PJ, Tangen CM, et al. The influence of finasteride on the development of prostate cancer. N Engl J Med 2003;349:215–224.
24. Gohagan JK, Prorok PC, Hayes RB, Kramer BS. The Prostate, Lung, Colorectal and Ovarian (PLCO) Cancer Screening Trial of the National Cancer Institute: history, organization, and status. Control Clin Trials 2000;21(suppl):21S–27S.
25. de Koning HJ, Auvine A, Berenguer Sanchez A, et al. Large-scale randomized prostate cancer...
screening trials: program performances in the European randomized screening for prostate cancer trial and the prostate, lung, colorectal and ovary cancer trial. Int J Cancer 2002;97:237–244.
26. Jemal A, Ward E, Thun M. Recent trends in breast cancer incidence rates by age and tumor characteristics among U.S. women. Breast Cancer Res 2007;9:R28.
27. Ravdin PM, Cronin KA, Howlader N, et al. The decrease in breast-cancer incidence in 2003 in the United States. N Engl J Med 2007;356:1670–1674.
28. Feigelson HS, Patel AV, Teras LR, et al. Adult weight gain and histopathologic characteristics of breast cancer among postmenopausal women. Cancer 2006;107:12–21.
29. Fisher B, Costantino JP, Wickerham DL, et al. Tamoxifen for prevention of breast cancer: report of the National Surgical Adjuvant Breast and Bowel Project P-1 Study. J Natl Cancer Inst 1998;90:1371–1388.
30. Vogel VG, Costantino JP, Wickerham DL, et al. Effects of tamoxifen vs raloxifene on the risk of developing invasive breast cancer and other disease outcomes: the NSABP Study of Tamoxifen and Raloxifene (STAR) P-2 trial. JAMA 2006;295:2727–2741.
31. Stafford RS, Driehling RL, Hersh AL. National trends in osteopontin visits and osteoporosis treatment, 1988–2003. Arch Intern Med 2004;164:1525–1530.
32. Alberg AJ, Brock MV, Samet JM. Epidemiology of lung cancer: looking to the future. J Clin Oncol 2005;23:3175–3185.
33. Giovino GA. Epidemiology of tobacco use in the United States. Oncogene 2002;21:7326–7340.
34. Wingo PA, Ries LA, Giovino GA, et al. Annual report to the nation on the status of cancer, 1973–1996, with a special section on lung cancer and tobacco smoking. J Natl Cancer Inst 1999;91:675–690.
35. Humphrey LL, Teutsch SM, Johnson M. Lung cancer screening with sputum cytologic examination, chest radiography, and computed tomography: an update for the U.S. Preventive Services Task Force. Ann Intern Med 2004;140:740–753.
36. The International Early Lung Cancer Action Program Investigators. Survival of patients with stage I lung cancer detected on CT screening. N Engl J Med 2006;355:1763–1771.
37. Mulshine JL, Sullivan DC. Lung cancer screening trials: program performances in the European randomized screening for prostate cancer trial and the prostate, lung, colorectal and ovary cancer trial. Int J Cancer 2002;97:237–244.
38. Martinez ME. Primary prevention of colorectal cancer: lifestyle, nutrition, exercise. Recent Results Cancer Res 2005;166:177–211.
39. Ries LA, Wingo PA, Miller DS, et al. The annual report to the nation on the status of cancer, 1973–1997, with a special section on colorectal cancer. Cancer 2000;88:2396–2424.
40. Baron JA, Cole BF, Sandler RS, et al. A randomized trial of aspirin to prevent colorectal adenomas. N Engl J Med 2003;348:891–899.
41. Pasy BM, Potter JD. Risks and benefits of celecoxib to prevent recurrent adenomas. N Engl J Med 2006;355:950–952.
42. Pignone M, Rich M, Teutsch SM, et al. Screening for colorectal cancer in adults at average risk: a summary of the evidence for the U.S. Preventive Services Task Force. Ann Intern Med 2002;137:132–141.
43. American Cancer Society. Cancer Prevention & Early Detection, Facts & Figures 2007. Atlanta, GA: American Cancer Society; 2007.
44. Bergstrom A, Pisani P, Tenet V, et al. Overweight as an avoidable cause of cancer in Europe. Int J Cancer 2001;91:421–430.
45. Vaimio H, Bianchini F. Weight Control and Physical Activity, Vol. 6 Lyon, France: International Agency for Research on Cancer Press; 2002.
46. Kaaks R, Lukanova A, Kurzer MS, Obesity, endogenous hormones, and endometrial cancer risk: a synthetic review. Cancer Epidemiol Biomarkers Prev 2002;11:1531–1543.
47. Grady D, Gebretsadik T, Kerlikowske K, et al. Hormone replacement therapy and endometrial cancer risk: a meta-analysis. Obstet Gynecol 1995;85:304–313.
48. Manson JE, Allison MA, Rosowsky JE, et al. Estrogen therapy and coronary-artery calcification. N Engl J Med 2007;356:2591–2602.
49. Kirkali Z, Chan T, Manoharan M, et al. Bladder cancer: epidemiology, staging and grading, and diagnosis. Urology 2005;66(suppl):4–34.
50. Ekstrom-Smedby K. Epidemiology and etiology of non-Hodgkin lymphoma—a review. Acta Oncol 2006;45:258–271.
51. Smith MT, Jones RM, Smith AH. Benzene exposure and risk of non-Hodgkin lymphoma. Cancer Epidemiol Biomarkers Prev 2002;11:1531–1543.
52. Smith MT, Jones RM, Smith AH. Benzene exposure and risk of non-Hodgkin lymphoma. Cancer Epidemiol Biomarkers Prev 2002;11:1531–1543.
53. Ekstrom-Smedby K. Epidemiology and etiology of non-Hodgkin lymphoma—a review. Acta Oncol 2006;45:258–271.
54. Smith MT, Jones RM, Smith AH. Benzene exposure and risk of non-Hodgkin lymphoma. Cancer Epidemiol Biomarkers Prev 2002;11:1531–1543.
55. Engel LS, Lan Q, Rothman N. Polychlorinated biphenyls and non-Hodgkin lymphoma. Cancer Epidemiol Biomarkers Prev 2007;16:373–376.
56. Hartge P, Smith MT. Environmental and behavioral factors and the risk of non-Hodgkin lymphoma. Cancer Epidemiol Biomarkers Prev 2007;16:367–368.
57. Jemal A, DeVesa SS, Hartge P, Tucker MA. Recent trends in cutaneous melanoma incidence among whites in the United States. J Natl Cancer Inst 2001;93:678–683.
58. Kokkinnides V, Weinstock M, Glanz K, et al. Trends in sunburns, sun protection practices, and attitudes toward sun exposure protection and tanning among US adolescents, 1998–2004. Pediatrics 2006;118:853–864.
59. Lukanova A, Kaaks R. Endogenous hormones and ovarian cancer: epidemiology and current hypotheses. Cancer Epidemiol Biomarkers Prev 2005;14:98–107.
60. Mosher WD, Martinez GM, Chandra A, et al. Use of contraception and use of family planning services in the United States: 1982–2002. Adv Data 2004;350:1–36.
61. Colombo N, Van Gorp T, Parma G, et al. Ovarian cancer. Crit Rev Oncol Hematol 2006;60:159–179.
62. Szkand V, Green A, Bain C, Purdie D. Beyond ovulation: oral contraceptives and epithelial ovarian cancer. Epidemiology 2000;11:106–110.
63. American Cancer Society. Cancer Facts & Figures 2007. Atlanta, GA: American Cancer Society; 2007.
64. Deschler B, Lubbert M. Acute myeloid leukemia: epidemiology and etiology. Cancer 2006;107:2099–2107.
65. Rhodes NL. Oral cancer: leukoplakia and squamous cell carcinoma. Dent Clin North Am 2005;49:143–165.
66. International Agency for Research on Cancer. Tobacco Smoke and Involuntary Smoking. Vol. 83 Lyon, France: International Agency for Research on Cancer; 2002.
67. Castelvecchio S, Munoz N, De Stefano E, et al. Independent and joint effects of tobacco smoking and alcohol drinking on the risk of esophageal cancer in men and women. Int J Cancer 1999;82:657–664.
68. Lowenfeld AB, Maisonneuve P. Epidemiology and risk factors for pancreatic cancer. Best Pract Res Clin Gastroenterol 2006;20:197–209.
69. Li D, Xie K, Wolf R, Abbruzzese JL. Pancreatic cancer. Lancet 2004;363:1049–1057.
70. Shah YH, Davila JA, El-Serag HB. The epidemiology of pancreatic cancer in the United States: changes below the surface. Aliment Pharmacol Ther 2006;24:87–94.
71. Hollandsworth JM, Miller DC, Daignault S, Thun MJ. Rising incidence of small renal masses: a need to reassess treatment effect. J Natl Cancer Inst 2006;98:1331–1334.
72. Chow WH, Gridley G, Fraumeni JR, Jervholm B. Obesity, hypertension, and the risk of kidney cancer in men. N Engl J Med 2000;343:1305–1311.
73. Lambert R, Guilloux A, Oshima A, et al. Incidence and mortality from stomach cancer in Japan, Slovenia and the USA. Int J Cancer 2002;98:911–920.
74. Crew KD, Neugut AI. Epidemiology of gynecologic cancer. World J Gastroenterol 2006;12:384–362.
75. Henson DE, Dittus C, Yones M, et al. Cigarette smoking and the risk of gastric cancer in the United States, 1973–2000: increase in the signet ring cell type. Arch Pathol Lab Med 2004;128:765–770.
76. Durie BG. The epidemiology of multiple myeloma. Semin Hematol 2001;38(suppl):1–5.
77. Williams R. Global challenges in liver disease. Hepatology 2006;44:521–526.
A Midpoint Assessment of the American Cancer Society Challenge Goal to Decrease Cancer Incidence by 25% Between 1992 and 2015

78. Seeff LB, Hoofnagle JH. Epidemiology of hepatocellular carcinoma in areas of low hepatitis B and hepatitis C endemicity. Oncogene 2006;25:3771–3777.

79. El-Serag HB, Davila JA, Petersen NJ, McGlynn KA. The continuing increase in the incidence of hepatocellular carcinoma in the United States: an update. Ann Intern Med 2003;139:817–823.

80. Singh GK, Hiatt RA. Trends and disparities in socioeconomic and behavioural characteristics, life expectancy, and cause-specific mortality of native-born and foreign-born populations in the United States, 1979–2003. Int J Epidemiol 2006;35:903–919.

81. Shepard CW, Simard EP, Finelli L, et al. Hepatitis B virus infection: epidemiology and vaccination. Epidemiol Rev 2006;28:112–125.

82. Armstrong GL, Wasley A, Simard EP, et al. The prevalence of hepatitis C virus infection in the United States, 1999 through 2002. Ann Intern Med 2006;144:705–714.

83. Wong JB, McQuillan GM, McHutchison JG, Poynard T. Estimating future hepatitis C morbidity, mortality, and costs in the United States. Ann J Public Health 2000;90:1562–1569.

84. Davies L, Welch HG. Increasing incidence of thyroid cancer in the United States, 1973–2002. JAMA 2006;295:2164–2167.

85. Ross DS. Predicting thyroid malignancy. J Clin Endocrinol Metab 2006;91:4253–4255.

86. Wrensch M, Minn Y, Chew T, et al. Epidemiology of primary brain tumors: current concepts and review of the literature. Neuro Oncol 2002;4:278–299.

87. Moore DH. Cervical cancer. Obstet Gynecol 2006;107:1152–1161.

88. US Food and Drug Administration. FDA Licenses New Vaccine for Prevention of Cervical Cancer and Other Diseases in Females Caused by Human Papillomavirus: Rapid Approval Marks Major Advancement in Public Health. Available at: http://www.fda.gov/bbs/topics/NEWS/2006/ NEW01385.html. Accessed August 8, 2007.

89. Villa LL, Ault KA, Giuliano AR, et al. Immunologic responses following administration of a vaccine targeting human papillomavirus Types 6, 11, 16, and 18. Vaccine 2006;24:5571–5583.

90. Brown LM, Devesa SS. Epidemiologic trends in esophageal and gastric cancer in the United States. Surg Oncol Clin N Am 2002;11:235–256.

91. Vizcaino AP, Moreno V, Lambert R, Parkin DM. Time trends incidence of both major histologic types of esophageal carcinomas in selected countries, 1973–1995. Int J Cancer 2002;99:860–868.

92. Byers T, Barrera E, Fontham ET, et al. A midpoint assessment of the American Cancer Society challenge goal to halve the U.S. cancer mortality rates between the years 1990 and 2015. Cancer 2006;107:396–405.

93. Smith RA, Cokkinides V, Eyre HJ. American Cancer Society guidelines for the early detection of cancer. 2006. CA Cancer J Clin 2006;56:11–25.

94. US Census Bureau. Poverty Status of the Population in 2001 by Sex, Age, Race and Hispanic Origin: March 2002. Available at: http://www.census.gov/poverty/socdemo/race/black/ppl-164/tab16.xls. Accessed August 8, 2007.

95. Ni H, Cohen R. Trends in Health Insurance Coverage by Race/Ethnicity Among Persons Under 65 Years of Age: United States, 1997–2001. US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics. Available at: http://www.cdc.gov/nchs/products/pubs/pubd/hestats/healthinsur.htm. Accessed August 8, 2007.

96. Ward E, Jemal A, Cokkinides V, et al. Cancer disparities by race/ethnicity and socioeconomic status. CA Cancer J Clin 2004;54:78–93.

97. American Cancer Society. Cancer Prevention & Early Detection Facts & Figures 2004. Atlanta, GA: American Cancer Society; 2004.