# Colon cancer incidence: recent trends in the United States

## Wong-Ho Chow, Susan S. Devesa, and William J. Blot

(Received 20 August 1991; accepted in revised form 17 September 1991)

Between 1976-78 and 1985-87, the age-adjusted incidence rates of invasive colon cancer in the United States rose by 15 percent, 3 percent, 21 percent, and 16 percent among White males, White females, Black males, and Black females, respectively. The increases in incidence occurred in all age groups over age 54 and affected each of the major subsites of the colon nearly equally. The larger rates of increase have resulted in higher incidence among Blacks than Whites by the mid-1980s and an increasingly greater excess of this cancer in males. Trends toward earlier diagnosis of invasive colon cancer were found, with increasing rates for localized and regional diseases coupled with stable or decreasing distant-stage disease-rates. The incidence of *in situ* colon cancer also rose substantially. The findings suggest that changes in diagnostic trends and risk-factor prevalence may be contributing to these patterns, and that the era when colon cancer predominated among White females is clearly over.

Key words: Colon cancer, incidence, race, stage, United States.

## Introduction

A rise in colon cancer incidence has been observed in the United States during recent decades.<sup>1,2</sup> This phenomenon may be due to an increase in the prevalence of potential risk factors,<sup>3-5</sup> although recent advances in screening and diagnostic techniques for colon cancer also may result in more complete case ascertainment and earlier diagnosis of the disease.<sup>6,7</sup> The present study updates and evaluates the stage- and age-specific incidence trends of colon cancer by sex and race based on data from a network of populationbased cancer registries in the US.

## Materials and methods

Data for this study were collected as part of the National Cancer Institute's Surveillance, Epidemiology and End Results (SEER) Program which has been described in detail elsewhere.<sup>2,8</sup> Briefly, this program is comprised of nine population-based cancer

registries, serving the states of Connecticut, Hawaii, Iowa, New Mexico, and Utah, and the greater San Francisco/Oakland (California), Detroit (Michigan), Seattle (Washington), and Atlanta (Georgia) metropolitan areas. These areas represent approximately 10 percent of the US population. Clinical and demographic data are obtained for all newly diagnosed cases among residents in these areas. The majority of cases are identified from local hospital records, with additional case ascertainment from free-standing pathology laboratories, outpatient clinics, physicians' offices, and death certificates.

Microscopically confirmed cases of *in situ* and invasive cancers of the colon (ICDO codes 153.0 to 153.9),<sup>9</sup> excluding melanomas, sarcomas, and lymphomas, diagnosed between 1976 and 1987 were included in this analysis. Malignant neoplasms arising from appendix (ICDO code 153.5) were excluded because of small numbers and potential confounding by the trends in

Authors are with the Epidemiology and Biostatistics Program, Division of Cancer Etiology, National Cancer Institute, National Institutes of Health. Address correspondence to Dr Chow, National Cancer Institute, 6130 Executive Blvd, EPN Room 407, Rockville, MD 20892, USA.

<b></b>	Time period				Overall	
	1976-78	1979-81	1982 - 84	1985 - 87	change %	AAPC
White males (No.)	(8,250)	(9,015)	(10,129)	(10,701)		
All ages	35.3	36.9	39.8	40.5	14.8	<i>1.6</i> <sup>d</sup>
Age < 55	5.0	4.8	4.8	4.7	- 6.5	- 0.7
55-64	80.2	82.7	88.7	92.0	14.7	1.6 <sup>d</sup>
65-74	191.5	198.9	215.6	216.0	12.8	1.5
>74	324.0	353.5	390.7	401.3	23.8	2.5 <sup>d</sup>
White females (No.)	(9,454)	(10,313)	(10,928)	(11,487)		
All ages	29.3	29.9	30.1	30.2	2.9	0.3
Age < 55	5.1	4.7	4.5	4.2	- 17.4	$-2.0^{d}$
55-64	70.0	69.4	69.3	69.0	- 1.5	— 0.2ª
65-74	154.7	153.0	153.7	154.8	0.1	0.0
>74	249.3	275.7	284.9	293.3	17.7	1.7 <sup>d</sup>
Black males (No.)	(621)	(736)	(816)	(937)		
All ages	34.7	38.3	39.5	41.9	20.7	2.0 <sup>d</sup>
Age < 55	6.3	5.9	6.4	7.2	14.0	1.7
55-64	90.6	100.2	100.0	99.8	10.2	0.9
65-74	173.2	180.7	192.6	234.5	35.4	3.5
>74	285.6	352.6	355.4	335.6	17.5	1.3
Black females (No.)	(704)	(875)	(966)	(1,061)		
All ages	29.7	33.4	33.9	34.5	16.4	1.5
Age < 55	6.6	7.1	5.7	6.3	- 5.7	- 1.3
55-64	77.8	80.7	88.1	91.7	18.0	2.0 <sup>d</sup>
65-74	122.9	162.0	188.1	178.7	45.4	3.8
>74	256.6	273.8	257.0	269.4	5.0	03

Table 1. Age-specific<sup>a</sup> and age-adjusted<sup>b</sup> incidence rates of invasive colon cancer by race and sex, nine SEER areas, 1976-78 to 1985-87

<sup>a</sup> Age-adjusted using five-year groups within each age stratum.

<sup>b</sup> Per 100,000 person-years, age-adjusted to the 1970 US population.

<sup>c</sup> Average annual percent change.

<sup>d</sup> Statistically significant at  $P \leq 0.05$ .

appendectomy. Cases diagnosed prior to 1976 were excluded because less than 94 percent were confirmed microscopically. The proportion of microscopically confirmed cases increased from 94.4 percent in 1976-78 to 96.8 percent in 1985-87, with little difference between Blacks and Whites. Also excluded were persons with racial origins other than Black or White because their numbers are too few for detailed analysis.

Cases were classified by age (<55, 55-64, 65-74, and >74), sex, and race. Trends for colon cancer were evaluated for the following time periods: 1976-78, 1979-81, 1982-84, and 1985-87. Incidence rates were computed using population estimates derived by the US Bureau of Census and the SEER Program, and age-adjusted to the 1970 US population using the direct method and five-year age intervals. Average, annual percent-changes were estimated by a linear regression of the logarithm of the respective rates on calendar year, weighted by the number of cases.<sup>10</sup>

#### Results

The overall, annual age-adjusted incidence rates of invasive colon cancer rose during the study period (Table 1, Figure 1). Within each gender, the increase was greater in Blacks than Whites, resulting in higher incidence rates of invasive colon cancer among Blacks since 1979-81. Within each race, the incidence rates climbed faster in males than females. Between 1976-78 and 1985-87, colon cancer rates rose by 15 percent, 3 percent, 21 percent, and 16 percent in White males, White females, Black males, and Black females, respectively (Table 1). The trends were generally consistent across the nine study areas, with evidence of convergence toward those in areas with initially high rates (data not shown).

The increases of invasive colon cancer were confined largely to localized and regional diseases, with the incidence of distant cancer generally declining (Figure 1).



Figure 1. Age-adjusted (1970) trends in colon cancer incidence by race, sex, and stage at diagnosis, nine SEER areas, 1976-78 to 1985-87.

Among Whites, comparable temporal trends and patterns toward earlier diagnosis were observed in all colon subsites, *i.e.*, cecum and ascending, transverse and the two flexures, and descending and sigmoid colon. The age-adjusted rates of invasive cancer rose by 9 percent, 10.3 percent, and 8.4 percent in each of the three subsites, respectively. Rates increased within each subsite more among Blacks than Whites, with the cecum and ascending colon rate rising 30 percent over the study period (12.4 percent and 15.2 percent increases were found for transverse/flexures and descending/sigmoid, respectively). Trends toward earlier diagnosis, with larger increases in localized disease compared with more distant diseases, also were apparent for each subsite among Blacks.

Furthermore, rates of invasive colon cancer rose in all age groups, except below age 55, where the rates generally declined (Table 1). The decrease in incidence at young ages occurred across all disease stages in females but mainly in distant cancers in males (data not shown). Increases were largest among Whites aged 75 or older and among Blacks aged 65-74 years. Throughout the study period, rates among Blacks exceeded those for Whites of the same sex for persons under age 65. For persons older than 74, rates were generally lower in Blacks than Whites. For persons aged 65-74, the age for which incidence increased most rapidly in Blacks of both genders, rates among Blacks were lower than among Whites in 1976-78, but surpassed those among Whites by 1979-81 in females and 1985-87 in males (Table 1). In general, a pattern of greater increase in localized disease and smaller decline in distant disease was observed in the older population compared with the younger groups (data not shown).

Substantial increases in rates of in situ colon cancer also were observed in all race and gender groups (Figure 1), and across all age strata. During the study period, the age-adjusted in situ disease rates rose by 163 percent, 134 percent, 181 percent, and 74 percent in White males, White females, Black males, and Black females, respectively. Similar to invasive colon cancer, the in situ cancer rates also were higher in Blacks than Whites throughout much of the study period. When in situ and invasive cancers are considered together, the percent in situ increased among Whites over the study period from 1.6 to 3.5 for cecum and ascending, from 2.3 to 4.0 for transverse, and from 4.4 to 10.0 for descending and sigmoid colon. The corresponding increases in percent in situ among Blacks were from 1.9 to 5.2, from 5.2 to 8.1, and from 5.7 to 9.9, respectively.

#### Discussion

The findings of increasing incidence of invasive colon cancer are consistent with results of earlier investigations.<sup>1,2</sup> Among Whites, colon cancer incidence in the

late 1940s was higher among females than males; however, an excess in incidence among males had emerged by 1970.<sup>1</sup> Our findings showed that the excess in males became more pronounced in the late 1980s, and occurred among both Blacks and Whites. The observations that the incidence rose more rapidly in Blacks than Whites, and that Blacks in recent years have a higher incidence of colon cancer than Whites, however, are not well appreciated generally. Also not well documented previously is the rapid rise in incidence of *in situ* colon cancer during the past decade.

It is of interest that, contrary to the colon cancer trends, the SEER data revealed fairly stable rectal cancer incidence-trends in this country, and higher rates in Whites than Blacks. Although cancers of colon and rectum often are studied together,<sup>35</sup> the present study did not include rectal cancer incidence-trends because their distinct epidemiologic patterns suggest that they may have different etiologic influences.

Although an alteration in reporting procedures or an improvement in case ascertainment may produce an apparent increase in incidence, the procedures for colon cancer case-finding and reporting for the SEER Program did not change during the study period. Fewer than five percent of the cases included for analysis were unstaged, and declines over time among Whites in unstaged cases were insufficient to account for the increases in early-staged diseases. Rates of unstaged cancer among Blacks remained fairly constant.

The current data suggest that invasive colon cancer is being diagnosed at earlier stages, as rates for localized disease rose and distant-staged cancers declined. In addition, *in situ* colon cancer increased proportionally more than invasive disease. Improved diagnostic techniques,<sup>11-14</sup> heightened awareness of colon cancer,<sup>15</sup> and increased availability of colon cancer screening<sup>16,17</sup> may contribute to the earlier diagnosis of this disease.

Despite the faster rise in overall rates among Blacks, the shifts to earlier stages at diagnosis are less evident among Blacks than Whites. This difference also is reflected in trends in five-year relative survival-rates for colon cancer. Between 1974-76 and 1981-86, the survival rates for Whites increased significantly from 49.6 percent to 56.2 percent; among Blacks, a smaller improvement in survival was observed, from 44.4 percent to 46 percent.<sup>2</sup>

While screening programs generally are credited with the earlier detection of colon cancer,<sup>6,17-19</sup> we were unable to determine what proportions of the increases in early-staged cancers were detected by routine screening in asymptomatic patients. Although Americans who ever had one of the screening procedures for colerectal cancers (*i.e.*, digital rectal examination, stool blood test, or proctoscopic examination) increased by 4 percent, 17 percent, and 11 percent respectively between 1983 and 1987, the majority of the population aged 50 or over still had not had one of these examinations by 1987.<sup>20</sup> Furthermore, the regional-staged cancer rates were consistently higher than localized disease rates in all race and sex groups, suggesting that much improvement still could be made toward earlier detection of colon cancer.

It is generally believed that a large proportion of colon cancer is preceded by adenomatous polyps.<sup>21-23</sup> An effective screening program that routinely detects and treats colon polyps before their progression to carcinoma eventually should reduce the incidence of colon cancer.<sup>19,24</sup> On the other hand, while colorectal cancers were detected at earlier stages in the study group than the control group in a randomized trial of annual multiphasic health checkups, the greater reduction in colorectal cancer mortality in the study than the control group was not attributed to a difference between these two groups in removal of colorectal polyps.<sup>18</sup> A subsequent case-control study conducted by the same investigators,<sup>25</sup> however, revealed that screening by rigid sigmoidoscope within 10 years prior to diagnosis reduced, by more than 50 percent, the risk of fatal cancers arising within the reach of the rigid sigmoidoscope, but not those beyond.

In addition to efforts in early detection of colon cancer, more research should be directed to identifying risk factors for this disease. The disparity in incidence trends of colon cancer between Blacks and Whites, between males and females, and between old and young may provide some clues to its etiology. Other than certain rare, predisposing genetic and medical conditions such as familial polyposis,26 Crohn's disease,27 and ulcerative colitis,<sup>28</sup> few specific risk factors for colon cancer have been established.29 Despite some inconsistencies in published results,<sup>30,31</sup> however, evidence is accumulating that high intake of animal fat, perhaps accompanied by low intake of dietary fiber, may increase the risk of colon cancer.<sup>4,29,32</sup> In addition, limited epidemiologic evidence has suggested that increased intake of fried or barbecued meat may be a risk factor for colon cancer,33-35 perhaps due to carcinogenic compounds such as heterocyclic amines<sup>33,36,37</sup> and polycyclic aromatic hydrocarbons<sup>38,39</sup> that are produced by high temperature cooking of meat. On the other hand, dietary intakes of calcium,40-42 vitamins A and C, and fruits and vegetables,43 have been suggested to reduce colon cancer risk. Other factors, such as high alcohol consumption,44-46 a sedentary lifestyle,47-49 increased body weight,<sup>50-52</sup> and low parity in women,<sup>53-56</sup> also have been linked to this disease.

It has been estimated from per capita food-use data

that in the US, the proportion of energy from fat increased from 32 percent to 43 percent, from carbohydrate decreased from 56 percent to 46 percent, and the proportion of energy from protein remained stable between 1909-13 and 1980.57 This increasing fat content in the American diet may explain, in part, the rising incidence of colon cancer over the past few decades. More recent survey data reported a small increase in grains, poultry, and fish, and a small decrease in red meat in the American diet during the past decade.<sup>58,59</sup> In addition, women consumed more dietary fiber per caloric intake<sup>60</sup> and had a higher ratio of polyunsaturated to saturated fats<sup>61</sup> than men. Dietary differences between Blacks and Whites have been less well characterized, but some studies report that Blacks have lower intakes of dietary fiber,60 vegetables,62 dairy products, and calcium63 than Whites. Although Blacks generally have comparable intakes of fat and calories as Whites, they have a consistently higher intake of dietary cholesterol.<sup>61</sup> These disparities in dietary patterns may have contributed to the lower colon cancer incidence in women than men, and the excess of colon cancer in Blacks than Whites of the same gender. It is not clear whether cooking patterns have changed over time (e.g., more barbecues) and, if so, how they might have influenced the colon cancer trends.

If level of physical activity is a risk factor for colon cancer,<sup>64</sup> it is conceivable that the increased level of leisure-time physical-activity levels over the recent decades<sup>65</sup> may have contributed to the leveling of colon cancer incidence in the youngest age groups. Decreased parity in women and the general reduction in the American family size probably did not have a substantial impact on recent colon cancer incidence-trends, since colon cancer incidence-rates in males have increased faster than those in females, and the incidence in young females has declined during the study period.

In conclusion, control and prevention of colon cancer can be achieved by screening and lifestyle changes. The heightened American consciousness regarding health promotion in recent years, including lowering animal fat intake and increasing exercise,<sup>66</sup> may reduce the incidence of colon cancer in the future. The current finding of leveling colon cancer incidence-trends among the youngest age groups also offers hope in the fight against this disease.

#### References

- 1. Devesa SS, Silverman DT, Young JL Jr. Cancer incidence and mortality trends among whites in the United States, 1947-84. JNCI 1987; **79:** 701-70.
- 2. Ries LAG, Hankey BF, Edwards BK, eds. Cancer Statistics Review 1973-87. Bethesda, Maryland: US Depart-

ment of Health and Human Services, Public Health Service, National Institutes of Health, 1991; NIH Pub. No. 90-2789.

- 3. Ziegler RG, Devesa SS, Fraumeni JF Jr. Epidemiologic patterns of colorectal cancer. In: Devita VT Jr, Hellman S, Rosenberg SA, eds. *Important Advances in Oncology* 1986. Philadelphia, PA: J.B. Lippincott, 1986: 209-32.
- Willett WC, Stampfer MJ, Colditz GA, et al. Relation of meat, fat, and fiber intake to the risk of colon cancer in a prospective study among women. N Engl J Med 1990; 323: 1664-72.
- 5. Lashner BA, Epstein SS. Industrial risk factors for colorectal cancer. *Int J Health Services* 1990; 20: 459-83.
- 6. Hardcastle JD, Armitage NC, Chamberlain J, et al. Fecal occult blood screening for colorectal cancer in the general population: results of a controlled trial. *Cancer* 1986; **58**: 397-403.
- 7. Hardcastle JD, Chir M, Pye G. Screening for colorectal cancer: a critical review. World J Surg 1989; 13: 38-44.
- Young JL Jr, Percy CL, Asire AJ, et al. Surveillance Epidemiology, End Results: incidence and mortality data, 1973-77. NCI Monogr 1981; 57: 1-1082.
- 9. World Health Organization. International Classification of Diseases for Oncology. Geneva: WHO, 1976.
- 10. Snedecor GW, Cochran WG. Statistical Methods, 6th edn. Ames, IA: Iowa State University Press, 1972.
- 11. Weisman GA, Winawer SJ, Baldwin MP, *et al.* Multicenter evaluation of training of non-endoscopists in 30-cm flexible sigmoidoscopy. CA 1987; **37:** 26-30.
- Winnan G, Berci G, Panish J, et al. Superiority of the flexible to the rigid sigmoidoscope in routine proctosigmoidoscopy. N Engl J Med 1980; 302: 1011-2.
- Longo WE, Ballantyne GH, Modlin IM. Colonoscopic detection of early colorectal cancers. Impact of a surgical endoscopy service. Ann Surg 1988; 207: 174-8.
- 14. Knutson CO, Max MH. Value of colonoscopy in patients with rectal blood loss unexplained by rigid proctosigmoidoscopy and barium contrast enema examinations. *Am J Surg* 1980; 139: 84-7.
- 15. Winchester DP, Fink DJ, Jolley PC. College and American society promote colorectal cancer awareness program. ACS Bull 1988; 73: 31-7.
- Lytle GH. Screening for colorectal carcinoma. Seminars Surg Oncol 1989; 5: 194-200.
- 17. Allison JE, Feldman R. Cost benefits of hemoccult screening for colorectal carcinoma. *Dig Dis Sci* 1985; 30: 860-5.
- Selby JV, Friedman GD, Collen MF. Sigmoidoscopy and mortality from colorectal cancer: the Kaiser Permanente Multiphasic Evaluation Study. J Clin Epidemiol 1988; 41: 427-34.
- 19. Winawer SJ, Schottenfeld D, Flehinger BJ. Colorectal Cancer Screening. JNCI 1991; 83: 243-53.
- 20. American Cancer Society. The 1987 Survey of Public Awareness and Use of Cancer Tests. Atlanta, GA: ACS, 1988; ACS Pub. No. 88-2M-No. 2520-LE.
- 21. Morson BC. Evaluation of cancer of the colon and rectum. *Cancer* 1974; 34: 845-9.
- Tierney RP, Ballantyne GH, Modlin IM. The adenoma to carcinoma sequence. *Gynecol Obstet* 1990; 171: 81-94.
- 23. Muto T, Bussey JHR, Morson BC. The evolution of cancer of the colon and rectum. *Cancer* 1975; **36**: 2251-70.
- Gilbertsen VA. Proctosigmoidoscopy and polypectomy in reducing the incidence of rectal cancer. Cancer 1974;

**34:** 936-9.

- 25. Selby JV, Friedman GD, Quesenberry CP Jr, *et al.* Efficacy of sigmoidoscopy in asymptomatic persons [Abstract]. Presented at the 24th Annual Meeting of the Society for Epidemiologic Research, Buffalo, New York, 11-14 June 1991.
- Leppert M, Burt R, Hughes JP, et al. Genetic analysis of an inherited predisposition to colon cancer in a family with a variable number of adenomatous polyps. N EnglJ Med 1990; 332: 904-8.
- 27. Ekbom A, Helmick C, Zack M, et al. Increased risk of large-bowel cancer in Crohn's disease with colonic involvement. *Lancet* 1990; **336**: 357-9.
- Ekbom A, Helmick C, Zack M, et al. Ulcerative colitis and colorectal cancer: a population-based study. N Engl J Med 1990; 323: 1228-33.
- 29. Willett W. The search for the causes of breast and colon cancer. *Nature* 1989; **338**: 389-94.
- Kolonel LN. Fat and colon cancer: how firm is the epidemiologic evidence? Am J Clin Nutr 1987; 45: 336-41.
- Nomura A. An international search for causative factors of colorectal cancer [Editorial]. JNCI 1990; 82: 894-5.
- 32. Prentice RL, Sheppard L. Dietary fat and cancer: consistency of the epidemiologic data, and disease prevention that may follow from a practical reduction in fat consumption. *Cancer Causes Control* 1990; 1: 81-97.
- Schiffman MH, Felton JS. Fried foods and the risk of colon cancer [Letter]. *Am J Epidemiol* 1990; 131: 376-8.
  Peters RK, Garabrant DH, Yu MC, *et al.* A case-control
- 34. Peters RK, Garabrant DH, Yu MC, et al. A case-control study of occupational and dietary factors in colorectal cancer in young men by subsite. *Cancer Res* 1989; 49: 5459-68.
- 35. Gerhardsson De Verdier M, Hagman U, Norell S, *et al.* Diet, body mass, physical activity and colorectal cancer [Abstract]. Presented at the 24th Annual Meeting of the Society for Epidemiologic Research, Buffalo, NY, 11-14 June 1991.
- Vanderlaan M, Watkins BE, Hwang M, et al. Monoclonal antibodies for the immunoassay of mutagenic compounds produced by cooking beef. Carcinogenesis 1988; 9: 153-60.
- Adamson RH, Thorgeirsson UP, Snyderwine EG, et al. Carcinogenicity of 2-amino-3-methylimidazo [4, 5-f] quinoline (IQ) in nonhuman primates: induction of tumors in three macaques. Jpn J Cancer Res 1990; 81: 10-4.
- International Agency for Research on Cancer. Certain Polycyclic Aromatic Hydrocarbons and Heterocyclic Compounds. Lyon, France: IARC, 1973; IARC Monogr Eval Carcinog Risk Chem Humans, Vol 3: 91-136.
- 39. Rothman N, Poirier MC, Baser ME, et al. Formation of polycyclic aromatic hydrocarbon—DNA adducts in peripheral white blood cells during consumption of charcoal-broiled beef. Carcinogenesis 1990; 11: 1241-3.
- Newmark HL, Wargovich MJ, Bruce WR. Colon cancer and dietary fat, phosphate, and calcium: a hypothesis. JNCI 1984; 72: 1323-5.
- Garland C, Shekelle RB, Barrett-Connor E, et al. Dietary vitamin D and calcium and risk of colorectal cancer: a 19-year prospective study in men. Lancet 1985; i(8424): 307-9.
- 42. Slattery ML, Sorenson AW, Ford MH. Dietary calcium intake as a mitigating factor in colon cancer. *Am J Epidemiol* 1988; **128**: 504-14.

- Vogel VG, McPherson RS. Dietary epidemiology of colon cancer. Hematol Oncol Clin North Am 1989; 3: 35-63.
- 44. Klatsky AL, Armstrong MA, Friedman GD, *et al*. The relations of alcohol beverage use to colon and rectal cancer. *Am J Epidemiol* 1988; 128: 1007-15.
- Longnecker MP. A case-control study of alcoholic beverage consumption in relation to risk of cancer of the right colon and rectum in men. *Cancer Causes Control* 1990; 1: 5-14.
- Longnecker MP, Orza MJ, Adams ME, et al. A metaanalysis of alcoholic beverage consumption in relation to risk of colorectal cancer. Cancer Causes Control 1990; 1: 59-68.
- Slattery ML, Schumacher MC, Smith KR, et al. Physical activity, diet, and risk of colon cancer in Utah. Am J Epidemiol 1988; 128: 989-99.
- Whittemore AS, Wu-Williams AH, Lee M, et al. Diet, physical activity, and colorectal cancer among Chinese in North America and China. JNCI 1990; 82: 915-26.
- Lee I-M, Paffenbarger RS Jr, Hsieh C-c. Physical activity and risk of developing colorectal cancer among college alumni. JNCI 1991; 83: 1324-9.
- Nomura A, Heilbrun LK, Stemmermann GN. Body mass index as a predictor of cancer in men. JNCI 1985; 74: 319-23.
- Graham S, Marshall J, Haughey B, et al. Dietary epidemiology of cancer of the colon in Western New York. Am J Epidemiol 1988; 128: 490-503.
- Chute CG, Willett WC, Colditz GA. A prospective study of body mass, height, and smoking on the risk of colorectal cancer in women. *Cancer Causes Control* 1991; 2: 117-24.
- 53. Weiss NS, Daling JR, Chow WH. Incidence of cancer of the large bowel in women in relation to reproductive and hormonal factors. *JNCI* 1981; **67:** 57-60.
- Potter JD, McMichael AJ. Large bowel cancer in women in relation to reproductive and hormonal factors: a casecontrol study. *JNCI* 1983; 71: 703-9.
- 55. Peters RK, Pike MC, Chang WWL, et al. Reproductive factors and colon cancers. Br J Cancer 1990; 61: 741-8.
- La Vecchia C, Franceschi S. Reproductive factors and colorectal cancer. Cancer Causes Control 1991; 2: 193-200.
- Welsh SO, Marston RM. Review of trends in food use in the United States, 1909-13 to 1980. J Am Diet Assoc 1982; 81: 120-8.
- Popkin BM, Haines PS, Reidy KC. Food consumption trends of US women: patterns and determinants between 1977 and 1985. Am J Clin Nutr 1989; 49: 1307-19.
- 59. US Institute of Medicine. *Improving America's Diet and Health: from Recommendations to Action*. Washington D.C.: National Academy Press, 1991: 42-6.
- 60. Lanza E, Jones DY, Block G, et al. Dietary fiber intake in the US population. Am J Clin Nutr 1987; 46: 790-7.
- 61. Block Ĝ, Rosenberger WF, Patterson BH. Calories, fat and cholesterol: intake patterns in the US population by race, sex and age. *Am J Public Health* 1988; **78:** 1150-5.
- 62. Patterson BH, Block G, Rosenberger WF, *et al.* Fruit and vegetables in the American diet: data from the NHANES II survey. *Am J Public Health* 1990; **80:** 1443-9.
- 63. Newell GR, Borrud LG, McPherson RS, *et al.* Nutrient intakes of whites, blacks and Mexican Americans in Southeast Texas. *Prev Med* 1988; 17: 622-33.

- 64. Bartram HP, Wynder EL. Physical activity and colon cancer risk? Physiological considerations [Editorial]. Am J Gastroenterol 1989; 84: 109-12.
- 65. Powell KE, Paffenbarger RS. Workshop on epidemiologic and public health aspects of physical activity and

exercise: a summary. Public Health Rep 1985; 100: 118-26.

 66. National Center for Health Statistics. Prevention Profile. Health, United States, 1989. Hyattsville, MD: Public Health Service, 1990; DHHS Pub. No. (PHS) 90-1232.