Preface/Introduction

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Preface

This monograph on colorectal cancer in Ontario was developed as a means to provide comprehensive background information for the Colorectal Cancer Screening Workshop in November 1998. This workshop will review the effectiveness of screening for colorectal cancer in Ontario and it will formulate public policy recommendations. The basis for the information in this monograph is the Ontario Cancer Registry (OCR) which is operated by Cancer Care Ontario (CCO), through funding provided by the Ontario Ministry of Health. The OCR is situated within the Surveillance Unit of the Division of Preventive Oncology at the Provincial Office of Cancer Care Ontario in Toronto.

The authors would like to thank the New South Wales Central Cancer Registry for the use of their recent monographs on Cancer in NSW, which served as models for the design and layout of our monograph. We are also grateful to Dr. Richard Schabas, Head of the Division of Preventive Oncology, for reviewing a draft of this document, as well as Dr. Neill Iscoe for advice about treatment information.

This monograph could not have been produced without the valuable assistance of Mrs. Virginia Hunter (desktop publishing, and project manager) and Ms. Sandrene Chin Cheong (technical support and graphics). Finally, the authors would like to acknowledge the contribution of all the operations staff within the Surveillance Unit, and the Registry Support Group (within the Information Systems Department) for their ongoing efforts in ensuring the generation of timely, high-quality, and useful cancer incidence data for Ontario.
About this monograph

This monograph comprises information on the incidence of, and mortality and survival from, colorectal cancer in Ontario, 1971 to 1996. The focus is on presentation of data relevant to assessing the burden of this cancer and the progress made towards reducing this burden. The section following these remarks provides some general background information about colorectal cancer, including the anatomy of the colorectum, risk factors, prevention and early detection, treatment and prognosis. In the second part of the monograph, the data on colorectal cancer in Ontario are presented, highlighting time trends, geographic comparisons, subsite and morphology distributions, and survival rates, with a small amount of descriptive text. These data come from the Ontario Cancer Registry, which is described in the “Guide to Readers” which follows. Aspects of the data and their presentation, such as data quality, classification of site and statistical methods, are also described in “Guide to Readers. The appendices include details of the classification schemes, the populations used to calculate rates, and the geographic regions of Ontario.

Background

Cancer of the colorectum will be newly diagnosed in 6,500 Ontarians in 1998, based on projected incidence. Over a lifetime, it will affect about one in 17 Ontarians, or 6%, based on current incidence rates. Over half of the risk of colorectal cancer manifests after 70 years of age, as shown in Table 1.

It is estimated that, by the end of 1998, there will be 47,000 Ontarians alive who have ever been diagnosed with colorectal cancer. Colorectal cancer is second only to lung cancer as the leading cause of cancer deaths in Ontario. It is estimated that 2,200 Ontarians will die from colorectal cancer in 1998 (NCIC, 1998). Current evidence suggests that a low-fibre, high-fat diet plays a major role in the development of this cancer (Potter, 1993).

Table 1. Probability* of developing colorectal cancer in Canada, by age and sex (NCIC, 1998)

<table>
<thead>
<tr>
<th>Sex</th>
<th>Probability (%) of Developing CRC by Age</th>
<th>Lifetime Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>30</td>
<td>40</td>
</tr>
<tr>
<td>Male</td>
<td>-</td>
<td>0.1</td>
</tr>
<tr>
<td>Female</td>
<td>-</td>
<td>0.1</td>
</tr>
</tbody>
</table>

* The probability of developing colorectal cancer is based on age- and sex-specific cancer incidence rates for Canada in 1993 and on life tables based on 1992-1994 all causes mortality rates

The study of colorectal cancers at various stages of development has improved our understanding of the genetic alterations involved in tumourigenesis. Abundant clinical and pathologic data suggest the hypothesis that most, if not all, colorectal cancers arise from pre-existing benign tumours, called adenomas or adenomatous polyps (Fearon, 1990). Emerging evidence suggests that most colorectal cancers probably arise from a minimum of five or more somatic genetic alterations consistent with the multi-step theory of tumourigenesis (Fearon, 1990). The discovery of somatic genetic changes that are restricted to neoplastic cells suggests that the future detection of early colorectal tumours may be possible through identification of mutant gene products secreted into blood or feces. Additionally, the identification of the specific genetic alterations present in tumours may provide a molecular tool for improved estimation of prognosis and response to treatment.
Anatomy and physiology

The colon and rectum make up the last 180-210 cm, (6-7 feet) of the intestinal tract. The colon consists of the appendix, caecum, ascending colon, hepatic flexure, transverse colon, splenic flexure, descending colon and sigmoid colon. As it leaves the abdominal cavity, the colon becomes the rectum which makes up the last 12-15 cm, (5-6 inches) of the intestinal tract. The last 2 cm. (1 inch) of the rectum is called the anal canal. The colon and rectum are part of the body’s digestive system, which removes nutrients from food and stores waste until it passes out of the body as fecal material.

Figure 1. Anatomy of the colorectum

The anatomic classification used in this monograph is based on the International Classification of Diseases - Ninth Revision (ICD-9) coding system (WHO, 1977). ICD-9 codes for subsites of the colorectum are given in Appendix A. Because of the anatomic and physiologic similarity of tissue in the colon and rectum, and the frequent problem of determining which anatomic subsite the cancer actually arose in, malignancies in the colon and rectum are often classed together as “colorectal cancer.”

Malignancies of the colorectum

Most colorectal cancers develop from the inside lining of the intestinal tract, called the mucosa. This cell layer contains cells that produce mucus, which protects the intestinal lining from bacteria and toxic substances in the stool, and lubricates the rectal canal. Most colorectal cancers arise from these glandular cells and are called adenocarcinomas. Adenocarcinomas represent approximately 95% of microscopically verified cases of colorectal cancer. Most colorectal cancers arise from benign adenomatous polyps which may be found on the inner wall of the colorectum. Adenomatous polyps most likely to become cancers include those which grow to a large size, have a villous appearance (finger-like projections) and/or contain dysplastic cells. Other colorectal tumours include carcinoids, lymphomas, melanomas and sarcomas, but these are all quite uncommon.
**Risk factors**

Risk factors include high dietary fat and meat intake, and low intake of fibre, fruits and vegetables (Potter, 1993). Chronic inflammatory bowel disorders, inherited disorders (e.g. familial adenomatous polyposis, hereditary non-polyposis colorectal cancer), and possibly obesity and low physical activity also increase risk (Lee, 1991). Higher parity and use of non-steroidal anti-inflammatory drugs may be protective, although the latter has not been consistently reported (Garewell, 1994). Alcohol consumption may increase the risk of rectal cancer. In addition, a slight increase in risk of colorectal cancer has been found for smokers after a latency period of around 30 years. The risk of colorectal cancer is also higher after breast, endometrial and gastric cancers, and also following earlier colorectal cancers (Schottenfeld, 1996).

The inherited forms of colorectal cancer, most notably familial adenomatous polyposis coli (FAP) and hereditary non-polyposis colorectal cancer (HNPCC), together make up approximately 6-10% of all colorectal cancer cases diagnosed (Lindor, 1998). The patients and families exhibiting these disorders have proven to be rich sources of material for studying the genetic changes associated with cancer, and the interaction of genetic and environmental factors. A tumour suppressor gene associated with FAP has been mapped to chromosome 5q and has been called the adenomatous polyposis coli (APC) gene. HNPCC appears to be associated with alterations of normally found mismatch repair genes responsible for detecting and correcting DNA base pair mismatches (Lindor, 1998).

**Prevention and early detection**

Dietary change may provide the best approach for reducing colorectal cancer incidence. There is increasing evidence that dietary change, as well as non-steroidal anti-inflammatory drugs, may play a part in the prevention of polyp development, or may reduce the transition of smaller to larger polyps (Miller, 1992).

There is increasing evidence that screening is effective in reducing the risk of fatal colorectal cancer (Solomon, 1994). Several screening modalities have been proposed, either singly or in combination, including fecal occult blood testing, sigmoidoscopy, colonoscopy and digital-rectal examination. The feasibility of screening as public health policy in Ontario is currently under study at Cancer Care Ontario (CCO), taking into consideration the effectiveness, cost and compliance associated with various strategies.

**Signs and symptoms**

Colorectal cancer most frequently causes a change in bowel habits, such as constipation, diarrhea or even the feeling that the bowel does not empty completely. Blood in the stool may also occur, either bright red or very dark in colour, although bleeding can occur from other sources, such as hemorrhoids. Tiny amounts of blood, called occult blood, which are not visible to the naked eye, may be detectable in the stool and may serve as an earlier indication of cancer or large polyps. General abdominal discomfort, pain, vomiting and/or weight loss may also occur, but these are often late symptoms and, of course, may be caused by other conditions.
Diagnosis and stage

To help find the cause of symptoms or positive laboratory findings, a thorough medical history, including family history, and physical examination are conducted. A number of diagnostic tests may be useful, including x-rays of the intestinal tract (barium enema), sigmoidoscopy (endoscopy of the rectum and lower part of colon), colonoscopy (endoscopy of the rectum and the entire colon) and biopsy.

If the diagnosis is cancer, then knowledge of the extent of spread (stage) will be necessary to plan treatment and estimate prognosis. Through a combination of physical examination, imaging, endoscopy and/or surgical exploration, the extent of local growth, lymphatic involvement and distant metastasis can be determined. Where stage is assigned by clinicians and recorded in the patient chart, most commonly the TNM classification or the Dukes classification is used (Sobin, 1997). Stage I (Dukes A) describes those tumours that are confined to the innermost layers of the bowel, notably the submucoosa and muscularis propria. Stage II (Dukes B) describes tumours that have grown into the muscle layer of the colon or rectum or through the bowel wall, but without regional lymph node involvement or distant metastasis. Stage III (Dukes C) describes tumours that have spread to one or more regional lymph nodes, but not distant metastasis. Stage IV (Dukes D) describes tumours that have metastasized to distant organs, such as the liver or lung. Patients with locally advanced large bowel cancer (Stages II and III) have a significantly increased risk of relapse after surgical resection alone. In these cases, adjuvant therapy may be added to surgery to prevent clinical metastatic disease.

Treatment

Treatment depends on a number of factors, including general health of the patient, and the size, location and extent of the tumour. Patients are often treated by a team of specialists, which may include a surgeon, medical oncologist, radiation oncologist and gastroenterologist. While there are many different treatments and combinations that are used to treat colorectal cancer, surgery is the primary method of treatment for most cases.

The type of surgery depends on where the cancer is found and the extent of its spread. If the cancer is only found in a polyp, the polyp may be removed (polypectomy) during colonoscopy. If a larger area is affected, a bowel resection is most commonly performed, with or without a temporary or permanent colostomy. If healthy portions of the colon or rectum cannot be reconnected, a temporary or permanent colostomy is performed. For resection of the colon, the common procedures include partial colectomy (less than hemicolectomy), hemicolectomy (all of right or left colon and a portion of the transverse colon), and total colectomy (beginning with caecum and ending with rectosigmoid part of rectum). It is not uncommon for colectomies to include partial or total removal of other organs, such as adjacent bowel, stomach or abdominal wall. Without distant metastases, cure is still possible in spite of multiple organ resection. Occasionally, a small tumour may be destroyed without pathologic specimen (cryosurgery, fulguration, electrocautery).

Cancer of the rectum has traditionally been treated with an abdominoperineal resection, which is performed from both the abdomen above and the rectal area below. The entire rectum, the tumour and surrounding tissue, including lymph nodes, are removed and the end of the colon is brought out through the abdominal wall as a permanent colostomy. If the tumour is situated high enough in the
rectum, a low anterior resection can often be accomplished without the need for a colostomy. At times a temporary colostomy may be performed to optimize healing of the anastomosis. Newer surgical techniques such as mesorectal excisions have been developed. Early reports suggest these may lead to enhanced local control without the loss of continence. Other procedures include pull-through operations and pelvic exenteration, as well as local excising and ablation of smaller tumours (Iscoe, 1997). The classification of surgical procedures for colorectal cancer is found in Appendix B.

The major complications of surgery include sepsis, anastomotic leak, bowel obstruction, urinary tract infection, ureteral injury, thromboembolism and wound dehiscence.

Adjuvant radiation therapy, given in addition to surgery, is commonly administered to reduce the rate of local recurrence of rectal cancer. It can be administered before or after surgery. Complications with modern radiotherapy are unusual, but may include rectal inflammation and acute bleeding; later complications include intestinal obstruction, and inflammation of the intestines and bladder.

The most common chemotherapy drug for colorectal cancer is 5-fluorouracil (5-FU). 5-FU may be given in a number of ways, including daily injections for several days, as weekly injections, as a 48 hour infusion or continuously through a pump. Patients with resected Stage III colon cancer are commonly offered adjuvant therapy with 5-FU and folinic acid (a biomodulator). Side effects are commonly seen during therapy; indeed, therapeutic response is unlikely to occur without some evidence of toxicity. Alopecia, dermatitis, stomatitis, esophagitis, diarrhea, vomiting, gastrointestinal bleeding, leukopenia and thrombocytopenia are common side effects (Gillis, 1997).

**Prognosis**

In general, tumours of the colon have a somewhat better prognosis than rectal cancer. The five-year cause-specific survival rate for right-sided colon cancer in Ontario is 53.1%; for left-sided colon cancer, it is 56.0%; and for rectal cancer it is 49.8%.

The incidence of subsequent polyps or second cancers of the colorectum is high, perhaps approaching 15 to 20% after 15 to 20 years of survival. Periodic colonoscopy is often recommended during follow-up. While recurrence of the original tumour is generally a pessimistic sign, some recurrences have a significant chance of being cured, including anastomotic recurrences, pelvic recurrences that are not fixed to the pelvic walls or even apparently single metastases to other organs. Surgery, radiotherapy and chemotherapy may all play a role in the management of advanced colorectal cancer.
Colorectal Cancer in Ontario
1971-1996

Incidence and Mortality in Ontario

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Most common cancers

The Ontario Cancer Registry (OCR) recorded 221,249 cancers newly diagnosed in Ontario residents in 1992-1996. Of these, 29,045 (13.1%) were in the colorectum, making this the fourth most common cancer site in the two sexes combined, after lung (14.5%), breast (13.7%) and prostate (13.2%). These four sites together accounted for over 50% of cancer diagnoses.

In females, colorectal cancer ranked second with 13,686 new diagnoses (12.9%), compared to 30,334 breast cancers (28.6%) (Table 2 and Figure 2). Lung cancer, with 12,149 new diagnoses (11.5%), ranked third.

In males, the colorectum was the third most common site of cancer with 15,359 new diagnoses (13.3%), following the prostate (29,264 cancers, 25.4%) and the lung (19,863 cancers, 17.2%).

Table 2. Most common cancers diagnosed in Ontario, 1992-1996, by sex

<table>
<thead>
<tr>
<th>Females</th>
<th></th>
<th>%</th>
<th>Males</th>
<th></th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breast</td>
<td>30,334</td>
<td>28.6</td>
<td>Prostate</td>
<td>29,264</td>
<td>25.4</td>
</tr>
<tr>
<td>Colorectum</td>
<td>13,686</td>
<td>12.9</td>
<td>Lung</td>
<td>19,863</td>
<td>17.2</td>
</tr>
<tr>
<td>Lung</td>
<td>12,149</td>
<td>11.5</td>
<td>Colorectum</td>
<td>15,359</td>
<td>13.3</td>
</tr>
<tr>
<td>Corpus uteri</td>
<td>5,751</td>
<td>5.4</td>
<td>Bladder</td>
<td>5,816</td>
<td>5.0</td>
</tr>
<tr>
<td>Non-Hodgkin’s lymphoma</td>
<td>4,078</td>
<td>3.9</td>
<td>Non-Hodgkin’s lymphoma</td>
<td>4,881</td>
<td>4.2</td>
</tr>
<tr>
<td>Melanoma</td>
<td>2,985</td>
<td>2.8</td>
<td>Kidney</td>
<td>3,700</td>
<td>3.2</td>
</tr>
<tr>
<td>Cervix uteri</td>
<td>2,892</td>
<td>2.7</td>
<td>Leukemia</td>
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<td>Stomach</td>
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<tr>
<td>Other</td>
<td>28,606</td>
<td>27.0</td>
<td>Other</td>
<td>26,440</td>
<td>22.9</td>
</tr>
</tbody>
</table>

Figure 2. Most common cancers diagnosed in Ontario, 1992-1996, by sex
Most common cancer causes of death

Cancer was the cause of death for 104,814 Ontario residents in the 5-year period 1992-1996. Colorectal cancer was the second most common cancer cause of death in the two sexes combined (11,095 deaths, 10.6%), after lung cancer (26,068 deaths, 24.9%). Breast cancer ranked third (9,490 deaths, 9.1%) and prostate cancer fourth (6,614 deaths, 6.3%).

Colorectal cancer ranked third as a cancer cause of death for each sex (Table 3 and Figure 3), with 5,162 deaths in females (10.5% of cancer deaths in women) and 5,933 deaths in males (10.6%). Only breast and lung cancers in females and lung and prostate cancers in males were more common cancer causes of death.

Table 3. Most common cancer causes of death in Ontario, 1992-1996, by sex

<table>
<thead>
<tr>
<th>Females</th>
<th>#</th>
<th>%</th>
<th>Males</th>
<th>#</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breast</td>
<td>9,400</td>
<td>19.4</td>
<td>Lung</td>
<td>16,645</td>
<td>29.8</td>
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<td>Lung</td>
<td>9,423</td>
<td>19.3</td>
<td>Prostate</td>
<td>6,614</td>
<td>11.8</td>
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<tr>
<td>Colorectum</td>
<td>5,162</td>
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<td>5,933</td>
<td>10.6</td>
</tr>
<tr>
<td>Ovary</td>
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<td>Pancreas</td>
<td>2,548</td>
<td>4.6</td>
</tr>
<tr>
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<td>2,556</td>
<td>5.2</td>
<td>Leukemia</td>
<td>2,119</td>
<td>3.8</td>
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<td>Non-Hodgkin’ s lymphoma</td>
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<td>2,089</td>
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<tr>
<td>Leukemia</td>
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<td>Stomach</td>
<td>2,081</td>
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<td>Esophagus</td>
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<td>Brain</td>
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<td>Bladder</td>
<td>1,608</td>
<td>2.9</td>
</tr>
<tr>
<td>Other</td>
<td>13,851</td>
<td>28.3</td>
<td>Other</td>
<td>14,563</td>
<td>26.1</td>
</tr>
</tbody>
</table>

Figure 3. Most common cancer causes of death in Ontario, 1992-1996, by sex
Numbers of new cases and deaths

The numbers of new cases of colorectal cancer diagnosed annually in Ontario residents increased from 1,528 to 2,731 (79% increase) between 1971 and 1996 in females and from 1,398 to 3,167 (127% increase) in males (Figure 4). In the earlier years more females than males were diagnosed with colorectal cancer, but since 1984 the reverse has been true. By the year 2011, 4,955 males and 3,636 females are expected to be diagnosed with colorectal cancer per year. This increase in the number of new diagnoses is due to projected population growth and population aging. The projected numbers should be interpreted with caution, as they are based on incidence rates during a period when the trend was changing (see Trends in Incidence and Mortality) and on population estimates projected from 1991 census data.

The numbers of deaths are smaller than the numbers of new cases but also rose over time (Figure 5). Proportional increases in the numbers of deaths are, however, considerably smaller than those for the numbers of new cases. Colorectal cancer deaths per year in females rose from 828 in 1971 to 1,050 in 1996 (a 22% increase) and from 804 to 1,195 in males (a 49% increase) over the same period.

Figure 4. Number of new cases of colorectal cancer in Ontario, 1971-1996, and projected to 2011, by sex and year

Figure 5. Number of deaths from colorectal cancer in Ontario, 1971-1996, by sex and year
Trends in incidence and mortality

Age-standardized incidence rates for cancer of the colorectum show quite different patterns over time in females and in males (Figure 6). For females, incidence rates increased slightly until about 1979, were stable for a few years, and then declined by an average of 1.6% per year (a statistically significant change) between 1982 and 1996. Males showed a similar pattern but were about five years behind females; incidence rates increased between 1971 and 1984, stabilized for a few years, then decreased by 1.0% per year (statistically significant) between 1987 and 1996.

In contrast, mortality rates have been declining continuously since 1971 for both men and women. Although not evaluated statistically, mortality may have declined more quickly than previously after about 1982 in women and 1987 in men. The early declines in mortality suggest improvements in survival, while the later acceleration in downward trend may reflect the observed reductions in incidence.

As a result of the different trends in males and females the ratio of male to female incidence rates rose from 1.1 in 1971, denoting a 10% male excess, to nearly 1.5 in 1990. It has remained constant at about 1.4-1.5, representing a 40-50% higher incidence in males, since that time. The male to female mortality rate ratio has exceeded the incidence rate ratio for every year, but showed a similar pattern, increasing from 1.2 in 1971 to 1.5-1.6 by the 1990s.

Figure 6. Age-standardized incidence and mortality rates (3-year moving averages) for colorectal cancer in Ontario, 1971-1996, by sex
Numbers of new cases and deaths, by age

Colorectal cancer occurred very infrequently prior to the age of 35. After age 35, the number of newly diagnosed cases increased with age to a peak in the 70-74 age group, where 536 males and nearly 450 females were diagnosed with colorectal cancer each year, on average, between 1992 and 1996 (Figure 7). The numbers of cases in each age group were similar in males and females at young ages, greater in males than females from age 50 to age 79, then greater in females at the oldest ages.

The number of colorectal cancer deaths was considerably less than the number of new cases in every age group, but the patterns by age and sex were roughly similar (Figure 8). Of note is the relatively large number of female deaths at age 85+.

Figure 7. Number of new cases of colorectal cancer in Ontario, by age and sex, 1992-1996

Figure 8. Number of deaths from colorectal cancer in Ontario, by age and sex, 1992-1996
Age-specific incidence and mortality rates

Age-specific incidence rates increased with age in both sexes (Figure 9), with a slight drop or levelling off at the oldest ages. The increase was steeper for men than for women. Incidence rates were similar in males and females at young ages but gradually diverged as age increased.

The pattern was similar for age-specific mortality, except there was no levelling off at older ages.

Figure 9. Age-specific incidence and mortality rates in Ontario, by sex, 1992-1996
Trends in incidence rates, by age group

In females, incidence rates showed an almost identical trend over time in each age group (Figure 10). Incidence rates were increasing at the beginning of the time period, then stabilized and finally began to decline, in a manner similar to that seen for the age-standardized rates (Figure 6). Table 4 gives estimates of the annual percentage change for the period 1982-1996 in females. Declines were statistically significant and of similar magnitude in each age group, ranging from 1.1% to 1.9% per year.

Incidence rates were higher in males than in females for every age group and each year. As in females, rates in males at each age rose, then stabilized, and then began to decline with time. Since the decline did not begin until later in males, the annual percent change was estimated only from 1987 (Table 4). Declines were observed for every age group, but differed significantly from zero only for those aged 65-79 (-1.0% per year) and 80+ (-1.9% per year).

Figure 10. Age-standardized incidence rates (3-year moving averages) for colorectal cancer in Ontario, 1971-1996, by age group and sex

Table 4. Annual percentage change (APC) and 95% confidence intervals (CI) for colorectal cancer in Ontario, by age group and sex

<table>
<thead>
<tr>
<th>Age Group</th>
<th>APC (%)</th>
<th>95% CI</th>
<th>Age Group</th>
<th>APC (%)</th>
<th>95% CI</th>
</tr>
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<tbody>
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<td>35-49</td>
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<td>(-2.8, -1.0)</td>
<td>35-49</td>
<td>-1.0</td>
<td>(-2.5, +0.6)</td>
</tr>
<tr>
<td>50-64</td>
<td>-1.1</td>
<td>(-1.6, -0.6)</td>
<td>50-64</td>
<td>-0.7</td>
<td>(-1.4, +0.0)</td>
</tr>
<tr>
<td>65-79</td>
<td>-1.3</td>
<td>(-1.7, -1.0)</td>
<td>65-79</td>
<td>-1.0</td>
<td>(-1.6, -0.4)</td>
</tr>
<tr>
<td>80+</td>
<td>-1.9</td>
<td>(-2.1, -1.2)</td>
<td>80+</td>
<td>-1.9</td>
<td>(-3.0, -0.9)</td>
</tr>
<tr>
<td>All Ages</td>
<td>-1.6</td>
<td>(-1.6, -1.2)</td>
<td>All Ages</td>
<td>-1.0</td>
<td>(-1.4, -0.6)</td>
</tr>
</tbody>
</table>
Trends in mortality rates, by age group

Trends in mortality rates across age groups (Figure 11) were similar to those observed for incidence, except that the declines began earlier (as noted in Trends in incidence and mortality). In both males and females in each age group except the oldest, rates were decreasing from the beginning of the time period (i.e. 1971). In females aged 80+, mortality was stable prior to 1981 and then declined, while in the oldest males, mortality rose before stabilizing and finally turning downward in the mid-1980s.

The downward trends in mortality may have accelerated in recent years in all age groups, corresponding to the observed downturns in incidence, as noted for age-standardized mortality in Trends in incidence and mortality.

Figure 11. Age-standardized mortality rates (3-year moving averages) for colorectal cancer in Ontario, 1971-1996, by age group and sex
Colorectal Cancer in Ontario
1971-1996

Geographic Patterns

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LD Marrett
R Parkes
G Fehringer

Surveillance Unit
Division of Preventive Oncology
Cancer Care Ontario

October, 1998
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Table 6.  Age-standardized mortality rates (per 100,000), rate ratios (RR) and 95% confidence intervals (CI) for colorectal cancer according to CCOR, 1992-1996

Figure 12.  Age-standardized (world) incidence rates for colorectal cancer by province of Canada, 1988-1992

Figure 13.  Age-standardized (world) incidence rates for colorectal cancer by country/region, 1988-1992
Regional patterns of incidence and mortality

Age-standardized incidence rates for males by Cancer Care Ontario Region (CCOR) ranged from 57.4 per 100,000 in the Central East Region to 73.7 in the Northeast in 1992-1996, a 28% difference (Table 5). The variation in females was much less. Incidence rate ratios significantly exceeded 1.0 (i.e. the rate in the CCOR was significantly greater than that for all Ontario) in the Northeast and Southwest CCORs and were significantly less than 1.0 in Central East, for both sexes.

Mortality rates do not always conform to the same pattern as incidence rates. For example, although both incidence and mortality were significantly high for males in the Northeast CCOR, males in the Southwest, where incidence was also high, had a significantly lower rate of mortality in spite of a significantly high incidence rate (Table 6). The discrepancies between incidence and mortality patterns may reflect underlying differences in diagnostic practices or in survival.

Table 5. Age-standardized incidence rates (per 100,000), rate ratios* (RR) and 95% confidence intervals (CI) for colorectal cancer according to CCOR, 1992-1996

<table>
<thead>
<tr>
<th>CCORs</th>
<th>Males</th>
<th></th>
<th></th>
<th>Females</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rate RR*</td>
<td>95% CI</td>
<td></td>
<td>Rate RR*</td>
<td>95% CI</td>
<td></td>
</tr>
<tr>
<td>South</td>
<td>60.8 1.00</td>
<td>(0.91, 1.09)</td>
<td>39.3</td>
<td>0.93</td>
<td>(0.85, 1.02)</td>
<td></td>
</tr>
<tr>
<td>Eastern</td>
<td>59.4 0.97</td>
<td>(0.92, 1.03)</td>
<td>42.5</td>
<td>1.01</td>
<td>(0.95, 1.06)</td>
<td></td>
</tr>
<tr>
<td>Southeast</td>
<td>62.3 1.02</td>
<td>(0.96, 1.08)</td>
<td>42.8</td>
<td>1.01</td>
<td>(0.95, 1.08)</td>
<td></td>
</tr>
<tr>
<td>Central East</td>
<td>57.4 0.94</td>
<td>(0.92, 0.96)</td>
<td>40.2</td>
<td>0.95</td>
<td>(0.93, 0.98)</td>
<td></td>
</tr>
<tr>
<td>Central West</td>
<td>63.1 1.03</td>
<td>(0.99, 1.08)</td>
<td>42.2</td>
<td>1.00</td>
<td>(0.95, 1.05)</td>
<td></td>
</tr>
<tr>
<td>Southwest</td>
<td>64.8 1.06</td>
<td>(1.02, 1.10)</td>
<td>46.9</td>
<td>1.11</td>
<td>(1.07, 1.16)</td>
<td></td>
</tr>
<tr>
<td>Northeast</td>
<td>73.7 1.23</td>
<td>(1.16, 1.31)</td>
<td>45.8</td>
<td>1.10</td>
<td>(1.02, 1.18)</td>
<td></td>
</tr>
<tr>
<td>Northwest</td>
<td>65.9 1.08</td>
<td>(0.97, 1.19)</td>
<td>41.6</td>
<td>0.99</td>
<td>(0.88, 1.11)</td>
<td></td>
</tr>
<tr>
<td>All Ontario</td>
<td>61.5 1.00</td>
<td>-</td>
<td>42.5</td>
<td>1.00</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>

Table 6. Age-standardized mortality rates (per 100,000), rate ratios* (RR) and 95% confidence intervals (CI) for colorectal cancer according to CCOR, 1992-1996

<table>
<thead>
<tr>
<th>CCORs</th>
<th>Males</th>
<th></th>
<th></th>
<th>Females</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rate RR*</td>
<td>95% CI</td>
<td></td>
<td>Rate RR*</td>
<td>95% CI</td>
<td></td>
</tr>
<tr>
<td>South</td>
<td>26.2 1.07</td>
<td>(0.93, 1.23)</td>
<td>15.4</td>
<td>1.00</td>
<td>(0.86, 1.16)</td>
<td></td>
</tr>
<tr>
<td>Eastern</td>
<td>23.6 0.97</td>
<td>(0.89, 1.05)</td>
<td>17.0</td>
<td>1.10</td>
<td>(1.01, 1.20)</td>
<td></td>
</tr>
<tr>
<td>Southeast</td>
<td>25.0 1.02</td>
<td>(0.93, 1.13)</td>
<td>15.0</td>
<td>0.97</td>
<td>(0.87, 1.08)</td>
<td></td>
</tr>
<tr>
<td>Central East</td>
<td>24.3 1.00</td>
<td>(0.96, 1.04)</td>
<td>15.1</td>
<td>0.98</td>
<td>(0.94, 1.02)</td>
<td></td>
</tr>
<tr>
<td>Central West</td>
<td>24.9 1.02</td>
<td>(0.95, 1.09)</td>
<td>15.4</td>
<td>1.00</td>
<td>(0.92, 1.07)</td>
<td></td>
</tr>
<tr>
<td>Southwest</td>
<td>22.4 0.92</td>
<td>(0.86, 0.99)</td>
<td>15.3</td>
<td>0.99</td>
<td>(0.92, 1.07)</td>
<td></td>
</tr>
<tr>
<td>Northeast</td>
<td>28.3 1.19</td>
<td>(1.07, 1.32)</td>
<td>15.4</td>
<td>1.04</td>
<td>(0.92, 1.17)</td>
<td></td>
</tr>
<tr>
<td>Northwest</td>
<td>24.7 1.01</td>
<td>(0.85, 1.19)</td>
<td>16.4</td>
<td>1.07</td>
<td>(0.88, 1.28)</td>
<td></td>
</tr>
<tr>
<td>All Ontario</td>
<td>24.5 1.00</td>
<td>-</td>
<td>15.5</td>
<td>1.00</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>

* Ratio of the incidence or mortality rate in a CCOR to that for all Ontario (known residence only)
Interprovincial comparisons

Figure 12 provides a comparison of the incidence of colorectal cancer in Ontario with that in other provinces of Canada. Note that the time period, 1988-1992, and the standard population used, the World Standard Population, are different than those used elsewhere in this publication. Data for the Atlantic provinces have been combined, since each province is small and the incidence across the four provinces is similar. The Northwest Territories and Yukon have been omitted as the numbers of cases are small and hence rates are unstable.

An east to west gradient is evident, with incidence in the east about 25% higher than that in the west for both men and women. Ontario’s incidence was intermediate, as is its geographic position.

Figure 12. Age-standardized (world*) incidence rates for colorectal cancer by province of Canada, 1988-1992

Females

Males

* standardized to the World Standard Population
International comparisons

There is considerably more variation in colorectal cancer incidence internationally (Figure 13) than there is either regionally within Ontario or inter-provincially (Figure 12). Ontario, along with other parts of North America, had among the highest incidences of colorectal cancer in the world. There was a five-fold difference between the incidence in Ontario and that in the lowest-incidence area shown (Bombay, India), suggesting that, theoretically, 80% of colorectal cancer in Ontario maybe preventable. Europe generally had somewhat lower rates than North America, followed by Latin America and Asia. There is, however, also considerable variation in incidence within each of these continents. It is worth noting that, although patterns are the same in male and females, incidence was higher in males than females in every country/region shown.

Figure 13. Age-standardized (world*) incidence rates for colorectal cancer by country/region, 1988-1992

**Females**

* US, SEER: Black
  * Canada, Ontario
  * US, SEER: White

* Denmark
  * UK, Scotland
  * Czech Republic
  * Finland

* US, Puerto Rico
  * Colombia, Cali

* Hong Kong
  * Japan, Osaka
  * China, Shanghai
  * India, Bombay

**Males**

* US, SEER: Black
  * Canada, Ontario
  * US, SEER: White

* Czech Republic
  * UK, Scotland
  * Denmark
  * Finland

* US, Puerto Rico
  * Colombia, Cali

* Hong Kong
  * Japan, Osaka
  * China, Shanghai
  * India, Bombay

* * standardized to the World Standard Population*
Colorectal Cancer in Ontario 1971-1996

Subsites and Morphology

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Division of Preventive Oncology
Cancer Care Ontario

October, 1998
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Figure 14. Age-standardized incidence rates (3-year moving averages) for .................... 2
  colorectal cancer by subsites in Ontario, 1979-1996, by sex
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  cancer by subsite, males and females combined, 1992-1996
**Incidence according to ICD-9 subsite**

The colorectum can be divided into a number of subsites, based on anatomic location (see Figure 1 and Appendix A). In 1992-1996, about 14% of tumours for each sex were not specified as to subsite (Table 7, “Other colon, colon not otherwise specified (NOS).”) The distribution of tumours across the remaining subsites differed for males and females. The most common subsite in males was rectum/rectosigmoid junction, with splenic flexure/descending colon/sigmoid colon second and caecum/ascending colon third; in contrast, the caecum/ascending colon was the most frequently occurring subsite in females, followed by splenic flexure/descending colon/sigmoid colon and rectum/rectosigmoid junction. As a result of these differences, the male to female incidence rate ratios varied from 1.10 for caecum/ascending colon to 1.94 for rectum/rectosigmoid junction, gradually increasing from the upper colon to the lower colon to the rectum. In both sexes, anus and appendix accounted for very small numbers of cases.

Table 8 shows the numbers of cancers at each subsite (excluding anus and appendix) according to age at diagnosis in 1992-1996. Over 90% of the cancers at each subsite were diagnosed after age 50. Note that a few cases had to be excluded due to unknown age at diagnosis.

**Table 7. Distribution, age-standardized incidence rates (per 100,000) and male to female incidence rate ratios (RR) for colorectal cancer subsites, Ontario, 1992-1996**

<table>
<thead>
<tr>
<th>Subsite</th>
<th>#</th>
<th>%</th>
<th>Rate</th>
<th>#</th>
<th>%</th>
<th>Rate</th>
<th>RR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Caecum, ascending colon</td>
<td>3,746</td>
<td>27.4</td>
<td>11.4</td>
<td>3,060</td>
<td>19.9</td>
<td>12.5</td>
<td>1.10</td>
</tr>
<tr>
<td>Hepatic flexure, transverse colon</td>
<td>1,027</td>
<td>7.5</td>
<td>3.2</td>
<td>894</td>
<td>5.8</td>
<td>3.6</td>
<td>1.14</td>
</tr>
<tr>
<td>Splenic flexure, descending colon, sigmoid colon</td>
<td>3,339</td>
<td>24.4</td>
<td>10.6</td>
<td>4,102</td>
<td>26.7</td>
<td>16.3</td>
<td>1.55</td>
</tr>
<tr>
<td>Rectosigmoid junction, rectum</td>
<td>3,080</td>
<td>22.5</td>
<td>9.7</td>
<td>4,764</td>
<td>31.0</td>
<td>18.8</td>
<td>1.94</td>
</tr>
<tr>
<td>Other colon, colon NOS</td>
<td>1,950</td>
<td>14.2</td>
<td>5.9</td>
<td>2,081</td>
<td>13.5</td>
<td>8.5</td>
<td>1.45</td>
</tr>
<tr>
<td>Anus</td>
<td>450</td>
<td>3.3</td>
<td>1.4</td>
<td>367</td>
<td>2.4</td>
<td>1.4</td>
<td>1.01</td>
</tr>
<tr>
<td>Appendix</td>
<td>94</td>
<td>0.6</td>
<td>0.3</td>
<td>91</td>
<td>0.6</td>
<td>0.3</td>
<td>1.06</td>
</tr>
<tr>
<td><strong>All colorectum</strong></td>
<td><strong>13,686</strong></td>
<td>100.0</td>
<td><strong>42.5</strong></td>
<td><strong>15,359</strong></td>
<td>100.0</td>
<td><strong>61.5</strong></td>
<td><strong>1.45</strong></td>
</tr>
</tbody>
</table>

**Table 8. Number of new cases of colorectal cancer in Ontario, 1992-1996, according to subsite and age at diagnosis**

<table>
<thead>
<tr>
<th>Subsite</th>
<th>&lt;50 years</th>
<th>Age at diagnosis</th>
<th>50+ years</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Females</td>
<td>Males</td>
<td>Total</td>
<td>Females</td>
<td>Males</td>
</tr>
<tr>
<td>Caecum, ascending colon</td>
<td>180</td>
<td>199</td>
<td>379</td>
<td>3,564</td>
</tr>
<tr>
<td>Hepatic flexure, transverse colon</td>
<td>65</td>
<td>69</td>
<td>134</td>
<td>962</td>
</tr>
<tr>
<td>Splenic flexure, descending colon, sigmoid colon</td>
<td>275</td>
<td>293</td>
<td>568</td>
<td>3,064</td>
</tr>
<tr>
<td>Rectosigmoid junction, rectum</td>
<td>288</td>
<td>375</td>
<td>663</td>
<td>2,791</td>
</tr>
<tr>
<td>Other colon, colon NOS</td>
<td>111</td>
<td>117</td>
<td>228</td>
<td>1,837</td>
</tr>
</tbody>
</table>
Trends in incidence rates for ICD-9 subsites

Trends over time for colorectal cancer subsites, excluding anus and appendix (which have too few cases), are shown in Figure 14 for the period 1979-1996. Years prior to 1979 were not included because a significant percentage of colorectal cancers reported to the OCR in this period were recorded only as “colon NOS” (see Figure 19). Thus, trends in specific subsites before 1979 would not be meaningful.

Figure 14 illustrates, as did Table 7, the different ranking of subsites within sex. Trends differed by subsite within sex also: in females, incidence decreased for all subsites except caecum/ascending colon and hepatic flexure/transverse colon, where it did not change over time. In males, incidence increased and then levelled off or declined slightly for all specific subsites except hepatic flexure/transverse colon, where it remained stable throughout the period. Only for other colon/colon NOS did incidence decrease noticeably in males, probably as a result of gradual improvements in data quality resulting in more frequent specification of subsite. The strong downward trend in the incidence of this non-specific site in both sexes may partially conceal the true trends in the more specific sites. Thus, these must be interpreted with some caution.

Differences in subsite trends between the sexes resulted in differential changes in the male to female rate ratio by subsite. Although male to female rate ratios (not shown) increased over time for all subsites, those subsites with the greatest male excess (splenic flexure/descending colon/sigmoid colon; rectosigmoid junction/rectum) also showed the greatest increase in the male to female rate ratio.

**Figure 14.** Age-standardized incidence rates (3-year moving averages) for colorectal cancer subsites in Ontario, 1979-1996, by sex

<table>
<thead>
<tr>
<th>Females</th>
<th>Males</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Caecum, ascending colon</td>
</tr>
<tr>
<td></td>
<td>Hepatic flexure, transverse colon</td>
</tr>
<tr>
<td></td>
<td>Splenic flexure, descending colon, sigmoid colon</td>
</tr>
</tbody>
</table>

**Legend:**
- 0 2 4 6 8 10 12 14 16 18 20
- Age-standardized rate per 100,000
- Year of diagnosis
Morphologic patterns by subsite

Colon excluding appendix

The large majority of colon cancer cases were adenocarcinomas - 97.9% of all microscopically confirmed cases (Table 9). Approximately 12.3% were cystic or mucinous or mucin-producing adenocarcinomas and 11.0% were adenocarcinomas in adenomatous polyps or villous adenocarcinomas.

The incidence rate for adenocarcinomas was 30.7 per 100,000 in the most recent time period, 1992-96. The incidence rates for specific subtypes, most notably mucinous adenocarcinomas and adenocarcinomas in adenomatous polyps, including villous adenocarcinomas, were 4.3 and 3.8 per 100,000, respectively. Concerning time trends, the age-standardized incidence rate of adenocarcinomas of the colon has been falling since the mid-1980s in females and the late-1980s in males. Primarily, the left colon has contributed to this decrease. There was an increase in the rates of mucinous adenocarcinomas and adenocarcinomas in adenomatous polyps over the 1980s, followed by a levelling of rates in the 1990s.

The age-standardized incidence rates for adenocarcinomas (all types) were higher in the left colon than in the right colon, with rates of 17.1 and 13.7 per 100,000, respectively, during the period 1992-96. The incidence rate of mucinous adenocarcinomas was also higher in the left colon than in the right, with rates of 2.5 and 1.8 per 100,000 respectively. Malignant carcinoids and signet ring adenocarcinomas, both of which are relatively uncommon, were the only morphologies that occurred at rates that were somewhat higher in the right colon than the left.

Table 9. Percentage distribution of morphologic type for colorectal cancer by subsite, males and females combined, Ontario, 1992-1996

<table>
<thead>
<tr>
<th></th>
<th>Colon</th>
<th>Rectum</th>
<th>Anus</th>
<th>Appendix</th>
</tr>
</thead>
<tbody>
<tr>
<td>#</td>
<td>%</td>
<td>#</td>
<td>%</td>
<td>#</td>
</tr>
<tr>
<td>Adenocarcinoma in</td>
<td>1,499</td>
<td>637</td>
<td>29</td>
<td>2</td>
</tr>
<tr>
<td>adenomatous polyps</td>
<td>7.4</td>
<td>8.1</td>
<td>3.6</td>
<td>1.1</td>
</tr>
<tr>
<td>Villous adenocarcinomas</td>
<td>716</td>
<td>437</td>
<td>37</td>
<td>5</td>
</tr>
<tr>
<td>3.6</td>
<td>5.6</td>
<td>4.6</td>
<td>2.7</td>
<td></td>
</tr>
<tr>
<td>Cystic and mucinous</td>
<td>2,478</td>
<td>582</td>
<td>54</td>
<td>43</td>
</tr>
<tr>
<td>adenocarcinomas</td>
<td>12.3</td>
<td>7.4</td>
<td>6.6</td>
<td>23.4</td>
</tr>
<tr>
<td>All other specified</td>
<td>300</td>
<td>124</td>
<td>15</td>
<td>12</td>
</tr>
<tr>
<td>adenocarcinomas</td>
<td>1.5</td>
<td>1.6</td>
<td>1.8</td>
<td>6.5</td>
</tr>
<tr>
<td>Adenocarcinoma, NOS</td>
<td>12,672</td>
<td>5,274</td>
<td>223</td>
<td>39</td>
</tr>
<tr>
<td>62.8</td>
<td>67.3</td>
<td>27.4</td>
<td>21.2</td>
<td></td>
</tr>
<tr>
<td>Epidermoid carcinoma</td>
<td>15</td>
<td>45</td>
<td>402</td>
<td>0</td>
</tr>
<tr>
<td>0.1</td>
<td>0.6</td>
<td>49.4</td>
<td>0.0</td>
<td></td>
</tr>
<tr>
<td>Malignant carcinoid</td>
<td>107</td>
<td>77</td>
<td>1</td>
<td>46</td>
</tr>
<tr>
<td>0.5</td>
<td>1.0</td>
<td>0.1</td>
<td>25.0</td>
<td></td>
</tr>
<tr>
<td>Other and ill-defined</td>
<td>304</td>
<td>121</td>
<td>13</td>
<td>3</td>
</tr>
<tr>
<td>adenocarcinomas</td>
<td>1.5</td>
<td>1.5</td>
<td>1.6</td>
<td>1.6</td>
</tr>
<tr>
<td>Subtotal (histologically</td>
<td>18,091</td>
<td>7,297</td>
<td>774</td>
<td>150</td>
</tr>
<tr>
<td>confirmed)</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td></td>
</tr>
<tr>
<td>Non-histologically</td>
<td>2,101</td>
<td>544</td>
<td>40</td>
<td>34</td>
</tr>
<tr>
<td>confirmed</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>20,192</td>
<td>7,841</td>
<td>814</td>
<td>184</td>
</tr>
</tbody>
</table>
Rectum including rectosigmoid junction

96.9% of rectal cancers were adenocarcinomas. Approximately 7.4% were classified as cystic or mucinous adenocarcinomas and 13.7% were classified as adenocarcinomas in adenomatous polyps or villous adenocarcinomas.

The age-standardized incidence rate for adenocarcinomas (all types) in 1992-96 was 12.3 per 100,000. For mucinous adenocarcinomas, it was 1.0 per 100,000, and for adenocarcinomas in adenomatous polyps, including villous adenocarcinomas, it was 1.9 per 100,000; there was some evidence of an increase in incidence rates, for these latter entities over the period 1979 to 1996 in the face of declining rates of adenocarcinoma NOS in females and males.

Anal canal and anus

Approximately 49.4% of anal cancers were classified as epidermoid, including squamous cell, basaloid, transitional cell and cloacogenic morphologies (Table 9). Adenocarcinomas comprised 44.0% of all anal tumours. The majority (60%) of melanomas of the large bowel occurred in the anus, although these accounted for only 1% of all anal cancers.

The age-standardized incidence of invasive tumours in the anus, anal canal and anorectum was 1.3 per 100,000 in the period 1992-96. There is some evidence of an increase in the age-standardized incidence rate of epidermoid carcinomas of the anus over the period 1979 to 1996. This finding may be of interest in view of a higher incidence of squamous cell carcinoma of the anus among homosexual men and the relationship between human papilloma virus and anogenital neoplasms.

Appendix

Malignant carcinoids were the commonest morphology, comprising 25.0% of all malignancies in the appendix (Table 9). Cystic and mucinous adenocarcinomas comprise 23.4% and adenocarcinoma NOS accounted for 21.2% of these malignancies. The age-standardized incidence rate for each of the three dominant morphologies - carcinoid adenocarcinoma, adenocarcinoma NOS, and mucinous adenocarcinoma - was approximately 0.1/100,000. This rate stayed constant for these three entities over the period 1979 to 1996.
Colorectal Cancer in Ontario
1971-1996

Survival

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October, 1998
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Survival trends over time

Cause-specific survival increased steadily with year of diagnosis from 1979 to 1991, as shown in Figure 15. This improvement, as well as declining incidence rates, is responsible for the declining mortality in both males and females. Since 1991, survival has plateaued with no further improvements seen.

The steady increase in survival for men and women likely reflects advances in surgical techniques and better control of post-operative infections, although, more recently, earlier detection may have contributed to better survival (Chu, 1994). It should be noted that earlier detection does not only result from widespread population screening, but may also result from earlier recognition of symptoms and improved diagnostic workups of symptomatic patients (Chu, 1994).

Survival by age and sex

As shown in Figure 16 there is little change in cause-specific survival at one year and five years following diagnosis among adult males and females up to approximately 70 years of age, after which survival deteriorates. Females have a small survival advantage compared with males, for each of colon and rectal cancer, with five-year cause-specific survival rates of 55.7% and 53.8% for colon cancers respectively, and 51.9% and 48.3% for rectal cancers, respectively. There is not a statistically significant difference between the sexes at one year, but by five years following diagnosis, females clearly display a cause-specific survival advantage. Multivariate modelling, adjusting for age and subsite, does not change this sex-specific difference. It is possible that some of the apparent benefit for females is an artefact of duplicate registrations in females, as described in the Guide to Readers section.
Survival by subsite

For all Ontarians diagnosed with colorectal cancer from 1979 to 1996, the five-year cause-specific survival rates for cancers of the right colon, left colon and rectum were 53.1%, 56.0% and 49.8% respectively, demonstrating that colon cancers have a somewhat better prognosis than rectal cancers.

The survival curves shown in Figure 17 describe cause-specific survival following cancer originating in various subsites in the colorectum. The survival advantage for malignancies of the appendix and anus reflects better prognosis for carcinoid tumours and epidermoid cancers, respectively. Prognosis following rectal cancer is somewhat poorer than that for colon cancer, but this begins to manifest only three to four years after diagnosis.

There is a small survival advantage for left-sided (lower) colon tumours as compared with right-sided (upper colon) tumours but this difference diminishes over time. This finding may reflect somewhat earlier diagnosis of left-sided tumours. This is supported by the higher proportion of adenocarcinomas in adenomatous polyps and villous adenocarcinomas in the left colon versus the right colon, as well as the markedly better survival for those two morphologies in the left colon versus the right colon.

Figure 17. Cause-specific survival for histologically confirmed cancers, Ontario, 1979-1996, by anatomic site
Survival by morphologic type

The survival curves shown in Figure 18 display a broad gradient in cause-specific survival by major morphologic type. Adenocarcinomas in adenomatous polyps show the best prognosis; undoubtedly, this reflects early stage at diagnosis. The prognosis for villous adenocarcinomas and adenocarcinomas in villous adenomas is somewhat poorer, likely reflecting the prognostic importance of the villous architecture. Both epidermoid carcinomas and carcinoids display a reasonably good prognosis, although these are more commonly seen in the rarer sites, namely, anus and appendix.

The prognosis for the common adenocarcinomas is somewhat poorer, but slightly above that for cystic and mucinous adenocarcinomas. Finally, the prognosis for other (e.g. sarcomas) and ill-defined (e.g. carcinoma NOS) cancers is very poor.

Figure 18. Cause-specific survival for histologically confirmed cancers, Ontario, 1979-1996, by morphology
Colorectal Cancer in Ontario
1971-1996

Guide to Readers

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Data sources

New cases of cancer

The Ontario Cancer Registry (OCR), operated by Cancer Care Ontario (CCO), registers all newly diagnosed cases of cancer (invasive neoplasms), except non-melanoma skin cancers. While cancer is not a legally reportable disease in Ontario, the Cancer Act provides a legal mandate for this undertaking and offers legal protection for physicians, dentists and health care agencies who report information on cancer cases.

The process of cancer registration in Ontario is passive, relying almost completely on records collected for other purposes. Since 1977, the OCR has relied on the same four major data sources: hospital discharge summaries which include a diagnosis of cancer; pathology reports with any mention of cancer; records of patients referred to CCO’s eight Regional Cancer Centres (RCCs) or the Princess Margaret Hospital (PMH), the specialized institutions treating cancer patients in Ontario; and death certificates with cancer as the underlying cause of death.

All records except pathology reports are coded at the source and provided to the OCR in machine-readable form. Paper copies of pathology reports are sent to the OCR by all hospital and private pathology laboratories and are coded and key-entered by OCR staff. Since 1991, the OCR has also received day surgery summaries which include a diagnosis of cancer. The OCR receives about 400,000 records from these multiple sources each year. The OCR is highly automated, relying heavily on automated edit-checking, computerized probabilistic record linkage and automated rule-based systems for summarizing patient and tumour information. Further details about the operation are available in recent monographs (Holowaty, 1995; Marrett, 1995; McLaughlin, 1995).

Deaths from cancer

Mortality data in the OCR are largely derived from the Office of the Registrar General of Ontario, where death certificate information is collected and coded. Mortality rates are estimated from the reported underlying cause of death, coded according to the International Classification of Diseases - Ninth Revison (ICD-9) (WHO, 1997), among persons residing in Ontario at the time of death. The period 1971 to 1996 was chosen for analysis to correspond to that chosen for incidence data.

Population data

Rates were calculated using annual mid-year estimated resident populations by sex and five-year age group. These estimates are based on the National Population Census, conducted every five years, and are corrected for census undercounts. Populations for both Ontario and its census divisions were provided by Statistics Canada (Statistics Canada, 1997). Populations for CCO Regions (CCORs) were determined by adding the populations for all census divisions that comprised each region.

The age and sex distribution of the 1991 Canadian population, adjusted for census undercount, was used to calculate most of the age-standardized rates appearing in this monograph (see Appendix C). For interprovincial and international comparisons, the World Standard Population was used to calculate the age-standardized rates (see Appendix C).
National and international data

Data on the incidence of colorectal cancer across Canada and around the world were recently published by the International Agency for Research on Cancer (IARC) in the monograph Cancer Incidence in Five Continents, Volume VII (Parkin, 1997). This monograph covers the period 1988 to 1992 for most reporting registries. For the purposes of comparisons with Ontario incidence data, regions/countries were selected to represent different continents, but also had to meet all of the data quality requirements of the IARC publication. Unfortunately, no areas of Africa qualified for these comparisons.

As well, data from the SEER (Surveillance, Epidemiology and End Results Program) Cancer Incidence Public-Use Dataset and Cancer Statistics Review, 1973-1995, were used, primarily as a benchmark for data quality, focusing on the most recent period 1991-1995 (Ries, 1998). For this purpose, blacks and those with second primaries of the same organ (i.e. colon or rectum) were excluded from the SEER data.

Incidence and mortality data

Data quality

Microscopic examination of tissue or cells is the definitive diagnostic test for cancer. During the period 1971 to 1996, 84% of colorectal cancers were microscopically verified in the OCR. This proportion increased to over 90% for the more recent interval 1979 to 1996, largely because of more complete reporting from pathology laboratories. It should be noted that this rate of microscopic confirmation is still somewhat below that reported by established active registries (e.g. SEER: 97%).

Another parameter of data quality is the percentage of cases for which a death certificate is the only source of information supporting a diagnosis of colorectal cancer. Only 2.0% of colorectal cancers registered from 1971 to 1996 were registered from death certificates only. Over the more recent period 1979 to 1996, the percentage was even smaller, at 1.2%.

Because it may be difficult to decide whether colorectal cancer is in the lower sigmoid colon or in the rectosigmoid area of the rectum, and because about one-third of all colorectal cancers are found in these areas, the sites of colon and rectum are often combined. Because of this problem, and uncertainty about the precise anatomic origin, particularly in late stage disease, site is not infrequently reported as colon, not otherwise specified (NOS) (ICD-9 153.9). The percentage of colorectal cancer so classified is another indication of the quality of the diagnosis. Over the period 1971 to 1996, 17.9% of Ontario cases were registered as colon NOS. Over the more recent period 1979 to 1996, the percentage fell somewhat to 15.1%. This is still above the percent reported by established active registries (e.g. SEER: 3.1%). The temporal trends in these parameters of data quality for colorectal cancer are shown in Figure 19.

Because the management of colorectal cancer almost always requires contact with institutions that comprise two or more of the OCR’s major reporting sources, it is likely that reporting is quite complete. Using an index derived from the ratio of the age-standardized incidence to mortality rates over the interval 1991 to 1995 inclusive, the relative completeness of colorectal cancer registration in the OCR, relative to the combined SEER registries, was 100% and 109% for men and women respectively, which suggests some over-reporting in females. There is some additional evidence of
over-reporting from a recent OCR field study that found duplicate registrations among 3% (3/87) of female colorectal cancer cases, largely because of incomplete identifiers on pathology reports.

This field study was undertaken in 1994/95, in order to estimate the validity of core data elements in the OCR, for a stratified random sample of approximately 1,200 cases, diagnosed from 1988 to 1991. Medical charts were reviewed in public hospitals and RCCs. Of this sample, there were 159 cases of colorectal cancer. All registered cases were confirmed to be colorectal cancer (confirmation rate or positive predictive value: 100%). However, two additional cases were found that had been originally registered to less specific sites (i.e. detection rate or sensitivity: 98.8%).

Using these two parameters of accuracy, colon cancer alone had an accuracy approaching 95%, whereas rectal cancer had a lower accuracy approaching 90%. Analysis of individual subsites showed that accuracy was quite good (greater than 80%) for cancers of the caecum, ascending colon, transverse colon, sigmoid colon and rectum, but accuracy was substantially lower for splenic flexure, descending colon and rectosigmoid junction. The subsite, colon NOS, was particularly problematic - most of these cases (12/16) were found on review to have occurred either in the sigmoid colon or rectum (9/12) or in the ascending colon or caecum (3/12). The anatomic origin of the remainder (4/16) could not be determined and remained as colon NOS.

Information about the accuracy of the colorectal subsites aided in subsequent groupings. For example, several cancers initially registered as originating in the descending colon or rectosigmoid...
junction were found to have actually originated in the splenic flexure. Thus, the splenic flexure was grouped with the descending colon and the rest of the lower bowel.

**Cancer site**

The primary site of cancer/cancer cause of death has been coded according to ICD-9 (see Appendix A). Sites in the colon are coded 153.0 - 153.9, and sites in the rectum 154.0 - 154.8. The fourth digit signifies the subsite within the colon or rectum. The rectosigmoid junction is coded as part of the rectum. For the purpose of this monograph, the right (upper) colon consists of the caecum, ascending colon, hepatic flexure and transverse colon. The appendix is excluded from the right colon, primarily because of a difference in morphologies. Additionally, it is a rare site.

The left (lower) colon consists of the splenic flexure, descending colon and sigmoid colon. The rectum consists of the rectosigmoid junction and rectum, excluding the anus, anal canal and anorectum. Based on the recent OCR validation study, these groupings are reasonably accurate, with detection and confirmation rates of 90-95% for the right colon, and rates of 80-90% for the lower colon and rectum. These groupings also minimize over- and under-reporting. The categories colon NOS (153.9) and colon with overlapping subsites (153.8) were also combined with the left colon.

For each person, only the first invasive cancer at each site (153 or 154) is registered in the OCR. Cancers occurring at the same time (synchronous), or subsequent (metachronous) cancers defined by the same three digit ICD-9 code, are not included in the frequencies or rates. It should be noted that many registries in Canada and the U.S.A. follow the SEER rules for coding multiple primaries. These rules permit the registration of second metachronous cancers in the colon or rectum if they occur at different subsites. The occurrence of second cancers of the colon or rectum is not rare. In the combined SEER database, over the recent period 1991 to 1995, second cancers of the same site accounted for 4.6% of registered colon cancers, and 0.7% of registered rectal cancers.

**Morphology**

The microscopic morphology or histopathology of tumours concerns the appearance of cancerous cells, tissues and organs under the light microscope. The histologic type of a cancer may be a prognostic factor; additionally, it may be important for treatment decisions. It may also be important in epidemiologic studies of cancer etiology. Cancers of different histologic types that occur in the same anatomic site often have different etiologies, incidences and prognoses.

The morphologic data presented in this monograph have been coded according to the First Edition of the International Classification of Diseases for Oncology (ICD-O) (WHO, 1976). The morphology (M) code in ICD-O consists of five digits - the first four digits describe the morphologic type, and the fifth digit describes the behaviour. The OCR registers only malignant neoplasms; i.e. with a behaviour code of 3.

The total number of morphologic types recognized in ICD-O approaches 500. Therefore, grouping of the various types is necessary. In the Subsites and Morphology section of this monograph, the grouping scheme employed is a variant of Berg’s traditional scheme for use in epidemiologic studies (Berg, 1982; Thomas 1995). However, some of the adenocarcinomas (e.g. adenocarcinoma in adenomatous polyp; mucinous adenocarcinoma) have been classified separately, primarily because of
prognostic differences compared with the dominant type, adenocarcinoma NOS. It should be noted that malignant lymphomas and Kaposi's sarcoma of the colorectum are not included in this monograph.

Spread of cancer at diagnosis

The OCR does not routinely collect information about the stage of cancer at diagnosis, primarily because this information is not captured at all by three of the reporting sources, and is incompletely captured by the fourth source (RCCs/PMH). However, in the recent OCR validation study, which included 161 validated cases of colorectal cancer diagnosed from 1988 to 1991, the distribution of summary stage was similar to that reported over the same period for the SEER registries. This is shown in Table 10.

Table 10. Comparison of stage distribution in OCR versus SEER

<table>
<thead>
<tr>
<th>AJCC Summary Stage</th>
<th>OCR Cases (1988-91) n=161</th>
<th>SEER Cases (1988-91) n=42,777</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage I (Dukes A)</td>
<td>16%</td>
<td>17.1%</td>
</tr>
<tr>
<td>Stage II (Dukes B)</td>
<td>20%</td>
<td>28.1%</td>
</tr>
<tr>
<td>Stage III (Dukes C)</td>
<td>18%</td>
<td>21.1%</td>
</tr>
<tr>
<td>Stage IV (Dukes D)</td>
<td>25%</td>
<td>17.1%</td>
</tr>
<tr>
<td>Unknown</td>
<td>21%</td>
<td>16.6%</td>
</tr>
</tbody>
</table>

This relatively simple classification, and others like SEER historic summary stage, have been readily implemented by many cancer registries on a population-wide basis. More complex staging classifications, such as the TNM System and the SEER EOD System, require the availability of much more precise information, which is not always found in a single source.

Time periods

In this monograph, incidence and mortality trends are presented for the period 1971 to 1996 inclusive. While the OCR actually begins in 1964, population estimates that have been adjusted for census undercount are only available since 1971. 1996 has been chosen as the final year for this monograph because it represents the most recent year for which there is complete reporting.

Concerning estimates of the burden of cancer or descriptions of selected characteristics of tumours or cases, the most recent five-year period was used, 1992 to 1996. This five-year aggregate was used in order to stabilize our estimates of infrequent events (e.g. diagnoses at younger ages) while providing the most current information.

More detailed analysis by subsite, morphologic type and survival focused on the period 1979 to 1996, excluding the earlier years because of an appreciable improvement in the rates of microscopic confirmation and in the precision of reported subsite since 1979 (Figure 2).
Residence at diagnosis and death

The geographic variation in incidence and mortality of colorectal cancer in Ontario over the period 1992 to 1996 is described in terms of CCORs, which are aggregations of census divisions. Census division of residence at the time of diagnosis comes primarily from hospital records in the OCR. All but 2.3% of cases of colorectal cancer from 1992 to 1996 have a census division of residence recorded. Due to the small percentage of cases with missing residence, all incidence rates calculated for the purpose of comparing the CCORs to the province exclude missing residence. Census division of residence at time of death comes from death certificates in the OCR. Mortality rates for CCOR comparisons were also calculated with missing residence excluded, since 0.45% of records of colorectal cancer deaths for the 1992 to 1996 time period did not report a valid census division of residence.

The eight CCORs cover the entire province, with populations (1996 estimate) ranging from 5.3 million in the largest (Central East CCOR), to 367,000 in the smallest (South CCOR). Within each CCOR is situated one of CCO’s RCCs. In addition, PMH is also located within the Central East CCOR. Maps describing CCOR boundaries and census division components are shown in Appendices D and E.

Statistical methods

Age-specific rates

Age-specific rates for each sex were calculated in five-year age groups by dividing the number of cases or deaths by the Ontario population in the same age group for the same calendar period. Age-specific rates are expressed per 100,000 person-years.

Age-standardized rates

To compare incidence and mortality rates between populations which have different age structures, age-standardized rates were calculated. The age-standardized rate is a weighted average of the age-specific rates, using a standard population age distribution. The standardized rates reflect the incidence and mortality that would have been expected if the population of interest had an age structure identical to the standard population. The 1991 Canadian population, adjusted for census undercount (Appendix C), was used as the standard throughout most of this monograph. However, in comparisons between Ontario’s rates and those of other provinces or countries, the World Standard Population was used (see Appendix C), largely because of data availability. Age-standardized rates are expressed per 100,000 person-years.

Time trends

Trends in the total burden of colorectal cancer for the period of 1971 to 1996 were described by plotting the annual number of incident cases and deaths, by sex. Three-year moving averages of age-standardized incidence and mortality rates by sex, and by sex and age group were plotted to describe the relative trends in colorectal cancer. Trends by anatomic subsite were described from 1979 to 1996 by sex, again using three year moving averages.

Annual rate of change for colorectal cancer incidence was estimated for the period 1982-1996 for females, and 1987-1996 for males. Each of these periods were selected as the longest period in which
the nonlinear components (quadratic and higher) of the trend were not statistically significant. Poisson regression using SAS/STAT PROC GENMOD (SAS Institute, 1997) was used to model the incidence data for each sex separately, estimating the common trend parameter in separate age strata (single year age groups 0-89, and 90+).

**Rate ratios**

The comparison of incidence or mortality rates in two populations (or subgroups), as a ratio of these rates, is termed a rate ratio (RR) or relative rate. In this monograph, this technique is employed for comparison of the rates of colorectal cancer for each CCOR to that of the province as a whole and for comparison of incidence in males and females. The rates for each population or subgroup are age-standardized, using the 1991 Canadian population, adjusted for census undercount (Appendix C) as the standard. A rate-ratio in excess of 1.00 for regional comparisons, for example, means that the region has a higher rate than Ontario as a whole.

**Confidence intervals**

A confidence interval (CI) indicates the level of precision of an estimate relative to the unobserved “true” value. For example, an incidence estimate of 94 cases with a 95% CI of (75-113) indicates that 94 is the best estimate of the expected number of cases, and that the interval of 75 to 113 has a 95% chance of encompassing the “true” value. Ideally, with perfect data quality, the estimate of 94 would be exactly correct for that population at that time. However, in order to make generalizations for the purpose of comparison or planning, it must be accepted that the observed number of cases is imprecise in the sense that 80 or 100 cases would also have been likely occurrences in the same population, simply due to random chance. Thus the confidence intervals presented in this monograph reflect the uncertainty in the underlying process itself, not in the quality of the data collected.

Confidence intervals for estimates of five-year survival were estimated using the Greenwood variance estimate (Cox, 1984) on the cumulative hazard scale (Link, 1986). For the annual percent change, profile likelihood confidence intervals (McCullagh, 1989) were computed using the Poisson regression model described in the Time trends section. Confidence intervals were calculated for incidence and mortality rate ratios between regions using the approximate bootstrap confidence interval method (Swift, 1995).

**Projections**

The expected number of incident cases of colorectal cancer is presented up to the year 2011 for both males and females. These estimates were calculated by extrapolating age-specific incidence rate trends using data from 1980-1995 and applying these extrapolated rates to projected population estimates made available by the Ontario Ministry of Finance. (Ontario Ministry of Finance, 1994)

**Survival**

Ontario-wide cause-specific survival statistics for colorectal cancer have been estimated from deaths due to cancer among cases registered with colorectal cancer in the OCR from 1979 to 1996. Only microscopically confirmed cases where the first primary cancer was colorectal cancer were included in the survival analysis. Cases were followed up for a maximum of ten years, with censoring at the earlier of December 31, 1997 or the date of diagnosis of any second primary cancer.
Sex-specific one- and five-year survival rates were estimated by age group and year of diagnosis. Survival plots were prepared by anatomic site and morphologic group, and five-year survival rates estimated by morphologic group for each anatomic site. The Kaplan-Meier product limit survival estimator was used for all survival estimates (Kaplan, 1958).

Proportional hazards regression modelling (Cox, 1984) was used to examine the combined effects of age, sex, year, anatomic site and morphology. This approach yielded results consistent with the analysis presented in this monograph, indicating that interactions between, and the confounding of, explanatory variables are minor issues, and do not change the conclusions presented here.

The relative survival approach (the ratio of observed survival in the colorectal cancer cases from all causes of death to the expected survival of a similar cohort from the general population) was used to assess excess mortality from causes other than cancer (or cancer deaths not specified as such on the death certificate). This analysis indicated that there was no excess mortality for the adenocarcinomas; and for the other morphologic groups, only a small portion of total mortality hazard was due to causes other than cancer. Incomplete follow-up of overall mortality resulted in relative survival estimates that became increasingly inconsistent as the number of years of follow-up increased. For this reason, only cause-specific survival estimates were used in this monograph.
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1971-1996

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### Appendix A: ICD-9 Topographic codes for malignant neoplasms of colorectum (WHO, 1977)

<table>
<thead>
<tr>
<th>Location</th>
<th>Codes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Right (upper) colon</strong></td>
<td></td>
</tr>
<tr>
<td>caecum</td>
<td>153.4</td>
</tr>
<tr>
<td>ascending colon</td>
<td>153.6</td>
</tr>
<tr>
<td>hepatic flexure</td>
<td>153.0</td>
</tr>
<tr>
<td>transverse colon</td>
<td>153.1</td>
</tr>
<tr>
<td><strong>Left (lower) colon</strong></td>
<td></td>
</tr>
<tr>
<td>splenic flexure</td>
<td>153.7</td>
</tr>
<tr>
<td>descending colon</td>
<td>153.2</td>
</tr>
<tr>
<td>sigmoid colon</td>
<td>153.3</td>
</tr>
<tr>
<td>colon, other</td>
<td>153.8</td>
</tr>
<tr>
<td>colon, unspecified (NOS)</td>
<td>153.9</td>
</tr>
<tr>
<td><strong>Rectum</strong></td>
<td></td>
</tr>
<tr>
<td>rectosigmoid junction</td>
<td>154.0</td>
</tr>
<tr>
<td>rectum</td>
<td>154.1</td>
</tr>
<tr>
<td><strong>Anus</strong></td>
<td></td>
</tr>
<tr>
<td>anus</td>
<td>154.3</td>
</tr>
<tr>
<td>anal canal</td>
<td>154.2</td>
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<tr>
<td>anorectum</td>
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<td><strong>Appendix</strong></td>
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<td>appendix</td>
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### Appendix B: Cancer directed surgery codes (CoC, 1997)

#### Colon (153.0-153.9)

<table>
<thead>
<tr>
<th>No cancer-directed surgery/unknown</th>
<th>CCP Codes</th>
</tr>
</thead>
<tbody>
<tr>
<td>00 No surgical procedure</td>
<td></td>
</tr>
<tr>
<td>01 Incisional, needle, or aspiration biopsy of other than primary site</td>
<td>66.82; 62.81</td>
</tr>
<tr>
<td>02 Incisional, needle, or aspiration biopsy of primary site</td>
<td>57.93-57.95</td>
</tr>
<tr>
<td>03 Exploratory surgery only (no biopsy)</td>
<td>57.97; 57.99</td>
</tr>
<tr>
<td>04 Bypass surgery or -ostomy only (no biopsy)</td>
<td>58.11-58.14; 58.03</td>
</tr>
<tr>
<td>05 Combination of 03 plus 01 or 02</td>
<td></td>
</tr>
<tr>
<td>06 Combination of 04 plus 01 or 02</td>
<td></td>
</tr>
<tr>
<td>07 Non-cancer-directed surgery NOS</td>
<td></td>
</tr>
<tr>
<td>08 Reconstructive surgery (for subsequent therapy only)</td>
<td></td>
</tr>
<tr>
<td>09 Unknown if surgery done</td>
<td></td>
</tr>
</tbody>
</table>

#### Type of cancer-directed surgery

<table>
<thead>
<tr>
<th>Type of cancer-directed surgery</th>
<th>CCP Codes</th>
</tr>
</thead>
<tbody>
<tr>
<td>10 Local cancer destruction without pathology specimen (includes laser surgery, cryosurgery, electrocautery, and fulguration)</td>
<td>57.21</td>
</tr>
<tr>
<td>20 Local surgical excision with pathology specimen (includes polypectomy, snare, or laser surgery)</td>
<td>57.29</td>
</tr>
<tr>
<td>30 Partial/subtotal colectomy (but less than hemicolecctomy - includes segmental resection, e.g. cecectomy, appendectomy, sigmoidectomy, partial resection of transverse colon and flexures, ileoctectomy, enterocolectomy and partial/subtotal colectomy NOS)</td>
<td>57.51:57.52; 57.54; 57.56; 57.59</td>
</tr>
<tr>
<td>40 Hemicolecctomy or greater (but less than total) right/left colectomy (all of right or left colon beginning at mid-transverse)</td>
<td>57.53; 57.55</td>
</tr>
<tr>
<td>50 Total colectomy (beginning with cecum and ending with the sigmoid/rectum or part of rectum)</td>
<td>57.6</td>
</tr>
<tr>
<td>60 Colectomy NOS</td>
<td></td>
</tr>
<tr>
<td>70 Colectomy (subtotal, hemicolecctomy, or total) plus partial or total removal of other organs</td>
<td></td>
</tr>
<tr>
<td>80 Surgery of regional and/or distant site(s)/node(s) only</td>
<td></td>
</tr>
<tr>
<td>90 Surgery NOS</td>
<td></td>
</tr>
</tbody>
</table>
### Appendix B: (continued)

#### Rectosigmoid, Rectum (154.0-154.1)

<table>
<thead>
<tr>
<th>CCP Codes (STC, 1986)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>No cancer-directed surgery/unknown</strong></td>
</tr>
<tr>
<td>00</td>
</tr>
<tr>
<td>01</td>
</tr>
<tr>
<td>02</td>
</tr>
<tr>
<td>03</td>
</tr>
<tr>
<td>04</td>
</tr>
<tr>
<td>05</td>
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<tr>
<td>07</td>
</tr>
<tr>
<td>08</td>
</tr>
<tr>
<td>09</td>
</tr>
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</table>

#### Type of cancer-directed surgery |

<table>
<thead>
<tr>
<th>CCP Codes (STC, 1986)</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
</tr>
<tr>
<td>20</td>
</tr>
<tr>
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<td>40</td>
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<tr>
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<td></td>
</tr>
<tr>
<td>60</td>
</tr>
<tr>
<td>70</td>
</tr>
<tr>
<td></td>
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<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>80</td>
</tr>
<tr>
<td>90</td>
</tr>
</tbody>
</table>
## Appendix C: World and Canadian standard populations, and Ontario 1996 population*

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>0-4</td>
<td>12,000</td>
<td>6946.4</td>
<td>747,717</td>
</tr>
<tr>
<td>5-9</td>
<td>10,000</td>
<td>6945.4</td>
<td>761,924</td>
</tr>
<tr>
<td>10-14</td>
<td>9,000</td>
<td>6803.4</td>
<td>746,766</td>
</tr>
<tr>
<td>15-19</td>
<td>9,000</td>
<td>6849.5</td>
<td>721,674</td>
</tr>
<tr>
<td>20-24</td>
<td>8,000</td>
<td>7501.6</td>
<td>757,574</td>
</tr>
<tr>
<td>25-29</td>
<td>8,000</td>
<td>8994.4</td>
<td>858,206</td>
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<tr>
<td>30-34</td>
<td>6,000</td>
<td>9240.0</td>
<td>1,023,410</td>
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<tr>
<td>35-39</td>
<td>6,000</td>
<td>8338.8</td>
<td>996,706</td>
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<tr>
<td>40-44</td>
<td>6,000</td>
<td>7606.3</td>
<td>874,646</td>
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<tr>
<td>45-49</td>
<td>6,000</td>
<td>5953.6</td>
<td>804,424</td>
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<tr>
<td>50-54</td>
<td>5,000</td>
<td>4764.9</td>
<td>616,522</td>
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<tr>
<td>55-59</td>
<td>4,000</td>
<td>4404.1</td>
<td>503,292</td>
</tr>
<tr>
<td>60-64</td>
<td>4,000</td>
<td>4232.6</td>
<td>462,390</td>
</tr>
<tr>
<td>65-69</td>
<td>3,000</td>
<td>3857.0</td>
<td>433,907</td>
</tr>
<tr>
<td>70-74</td>
<td>2,000</td>
<td>2965.9</td>
<td>378,250</td>
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<tr>
<td>75-79</td>
<td>1,000</td>
<td>2212.7</td>
<td>261,584</td>
</tr>
<tr>
<td>80-84</td>
<td>500</td>
<td>1359.5</td>
<td>170,896</td>
</tr>
<tr>
<td>85+</td>
<td>500</td>
<td>1023.7</td>
<td>132,537</td>
</tr>
<tr>
<td>Total</td>
<td>100,000</td>
<td>100,000</td>
<td>11,252,425</td>
</tr>
</tbody>
</table>

* preliminary postcensal estimates
Appendix D: Cancer Care Ontario Regions (CCORs)
Appendix E: Cancer Care Ontario Regions (CCORs) showing census divisions

Northern Ontario

Census Divisions
48. Nipissing
49. Parry Sound
51. Manitoulin Island
52. Sudbury District
53. Sudbury R.M.
54. Timiskaming
56. Cochrane
57. Algoma
58. Thunder Bay
59. Rainy River
60. Kenora

Southern Ontario

Census Divisions
1. Stormont, Dundas and Glengarry
2. Prescott & Russell
6. Ottawa-Carleton
7. Leeds & Grenville
9. Lanark
10. Frontenac
11. Lennox & Addington
12. Hastings
13. Prince Edward
14. Northumberland
15. Peterborough
16. Victoria
18. Durham
19. York
20. Toronto
21. Peel
22. Dufferin
23. Wellington
24. Halton
25. Hamilton-Wentworth
26. Niagara
28. Haldimand Norfolk
29. Brant
30. Waterloo
31. Perth
32. Oxford
34. Elgin
36. Kent
37. Essex
38. Lambton
39. Middlesex
40. Huron
41. Bruce
42. Grey
43. Simcoe
44. Muskoka
46. Haliburton
47. Renfrew