# National Cancer Control Programme Colorectal Cancer 3.4. Polyposis and other risk factors

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Early detection of colorectal cancer (crc) is best based on an individual's personal and familial risk.

#### <u>Age</u>

The risk for crc increases slightly at age 40 and more sharply at age 50, doubling thereafter with each decade (see 2.1). Individuals who are at risk by virtue of age only and who have no other associated high risk factors are considered at average risk, the others at high risk (Table 1.).

The lifetime risk for crc is about 5% for men and women in western industrialized countries.

# Adenomatous polyps

The concept that most crc evolve from benign adenomas (adenomatous polyps) is now widely accepted (1). A colorectal polyp is defined as a visible protrusion above the surface of the surrounding normal large bowel mucosa (2). Colorectal adenomas are common in the general population: 22 to 61 percent prevalence in autopsy studies; 25 to 41 percent prevalence in colonoscopy studies of asymptomatic, average-risk individuals with no personal or family history (2). The National Polyp Study has demonstrated that colonoscopic removal of adenomatous polyps significantly reduces the risk of developing crc (3). Adenomas, which are potentially malignant, account for approximately two thirds of the polyps removed by colonoscopy. The remaining polyps are usually overgrowths of normal mucosa or other polyps with no malignant potential. All adenomas, by definition have some degree of dysplasia. Approximately two thirds of adenomas are tubular; they have less premalignant potential than adenomas with villous features, which become more frequent with increasing size.

## Familial syndromes

The inherited syndromes of crc can be separated into two categories: the polyposis syndromes (familial adenomatous polyposis - FAP, Gardner's syndrome etc.) and the hereditary nonpolyposis colorectal cancer syndrome (hereditary nonpolyposis colorectal cancer - HNPCC) - see 6.3. In addition to such rare syndromes, there is evidence that partially penetrant inherited factors play a role in most cases of crc (see 6.2).

## Inflammatory bowel disease

The etiology of chronic colitis remains unknown, but evidence has accumulated from studies of different ethnic groups, families of patients, and twin pairs to implicate genetic susceptibility in pathogenesis (see 3.3). Genes of the major histocompatibility complex are implicated as inherited determinants of susceptibility to ulcerative colitis and may also influence the pattern of disease (4).

## Family history

A family history of adenoma or crc is associated with an increased risk of adenoma and crc (see 6.2).

## Patient history

Individuals presenting with an adenoma have a 40% to 50% likelihood of additional (synchronous) adenomas. Patients who have had an adenoma removed from their colon have a 30% increased risk of a subsequent adenoma (5,6). Prevalence rates of adenomas correlate with crc incidence rates (2).

Synchronous crc occur in 3% to 5 % of patients (7). The risk for crc is double the risk of the general population after a first crc (8). The relative risk for subsequent breast, ovarian or uterine cancer ranges from 1 to 2 in crc patients (9).

Pelvic radiation doubles the risk for rectal cancer (10).

The clinician should consider the diagnosis of crc when a person reports one or more of the following <u>symptoms</u>: one episode of blood in the feces, on the feces or on the toilet paper; change in bowel habits for more than 3 weeks or unusual abdominal, pelvic or back pain. Doctors should explain to their clients what melena is.

#### References

- Toribara N.W. and Sleisenger M.H.: Screening for colorectal cancer. N. Engl. J. Med. 331: 1669-1674, 1994.
- Markowitz A.J. and Winawer S.J.: Management of colorectal polyps. CA Cancer J. Clin. 47: 93-112, 1997.
- Winawer S.J., Zauber A.G., Ho / M.N. et al.: Prevention of colorectal cancer by colonoscopic polypectomy. N. Engl. J. Med. 329: 1977-1981, 1993.
- Satsangi J., Welsh K.I., Bunce M., Julier C., Farrant J.M., Bell J.I. and Jewell D.P.: Contribution of genes of the major histocompatibility complex to susceptibility and disease phenotype in inflammatory bowel disease. Lancet 347: 1212-1217, 1996.
- Winawer S.J., O'Brien M.J., Waye J.D. et al.: Risk and surveillance of individuals with colorectal polyps. Bull. World Health Organ. 68: 789-795, 1990.
- O'Brien M.J., Winawer S.J., Zauber A.G. et al.:
   The National Polyp Study: patient and polyp characteristics associated with high-grade dysplasia in colorectal adenomas.
   Gastroenterology 98: 371-379, 1990.
- 7. Langevin J.M., Nivatvongs S.: The true incidence of synchronous cancer of the large bowel. Ann. J. Surg. 147: 330-333,1984.
- 8. Haar S.K., Wilson J., Blot W.J., Mc Laughlin J.K., Winn D.M. and Kantor A.F.: Second cancer following cancer of the digestive system in Connecticut, 1935-82. Natl. Cancer inst. Monogr. 68: 49-82, 1985.
- NCI: Multiple primary cancers in Connecticul and Denmark. Natl. Cancer Inst. Monogr. 68.
- Storm H.H.: Second primary cancer after treatment for cervical cancer. Late effects after radiotherapy. Cancer 61: 679-688, 1988.

## Table 1. Risk factors for colorectal cancer

# **AVERAGE RISK**

Age: > 50 years, asymptomatic.

### HIGH RISK

Familial adenomatous polyposis

Familial adenomatous polyposis (FAP)

Gardner's syndrome Turcot syndrome I

Peutz-Jeghers syndrome

Juvenile polyposis

Hereditary nonpolyposis colorectal cancer

Hereditary nonpolyposis crc (HNPCC)

Turcot syndrome II Muir-Torre syndrome

Inflammatory bowel disease

Chronic ulcerative colitis

Chronic granulomatous colitis (Crohn's disease)

Family history

Colorectal adenomas

Colorectal cancer

Patient history

Colorectal adenomas

Colorectal cancer

Breast, ovarian, or uterine cancer

Pelvic radiation

**Symptoms**