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The Role of Health Information Management and Medical Secretaries in Improving Early Detection and Public Health Surveillance of Colorectal Cancer.

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Abstract

Background: Colorectal cancer (CRC) is a major global health concern and remains an important cause of cancer-related morbidity and mortality. Its development involves genetic susceptibility, dietary habits, lifestyle behaviors, metabolic disorders, and chronic inflammatory conditions. Differences in population characteristics, screening practices, and healthcare access also contribute to variations in CRC incidence and outcomes.

Aim: This review examines the epidemiology, survival patterns, and major risk factors associated with CRC, with particular attention to findings from Japan. It also discusses the contributions of public health initiatives, health information systems, and medical secretarial services to prevention and early detection.

Methods: Relevant literature was reviewed to evaluate global and Japanese CRC incidence and mortality trends, stage-specific survival, hereditary predispositions, dietary and lifestyle exposures, inflammatory bowel disease, diabetes, and socioeconomic determinants. Evidence concerning screening programs, cancer registries, emerging technologies, and research initiatives was also considered.

Results: CRC incidence and mortality vary across geographical regions and populations. Survival is substantially higher when cancer is diagnosed at an early stage, emphasizing the importance of screening and timely treatment. Major risk factors include Lynch syndrome, familial adenomatous polyposis, processed meat consumption, obesity, alcohol intake, smoking, inflammatory bowel disease, and type 2 diabetes. Vitamin D, calcium, and fermented dairy products have demonstrated potential protective associations. Japanese studies have also identified population-specific dietary and metabolic associations. Public health interventions, integrated cancer registries, electronic health records, and machine learning may improve screening participation, risk identification, and follow-up. Medical secretaries support these processes through appointment coordination, documentation, referral management, and patient communication.

Conclusion: CRC prevention requires coordinated strategies combining risk reduction, population-based screening, accurate health information management, and timely clinical follow-up. Integrating public health services, information technology, and administrative coordination may improve early detection and equitable access to care.

Keywords: Colorectal Cancer; Early Detection; Health Information Management; Medical Secretaries; Public Health Surveillance; Cancer Screening; Risk Factors; Cancer Registries; Japan.

Introduction

Colorectal cancer (CRC) represents a major global health concern, ranking as the third most commonly diagnosed cancer and the second leading cause of cancer-related deaths worldwide [1]. Its incidence and mortality vary across geographical regions, reflecting differences in socioeconomic development, dietary habits, lifestyle factors, population structure, and access to healthcare services. Countries with high human development

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index (HDI) values report higher incidence rates, partly associated with dietary patterns characterized by increased consumption of red and processed meat, sugary beverages, and high-calorie foods, alongside physical inactivity and obesity. However, countries with low and middle HDI values are experiencing increasing CRC incidence as urbanization, population aging, and the adoption of Western dietary habits continue to influence disease patterns. Japan provides an example of this epidemiological transition, with changes in dietary habits and lifestyle contributing to the increasing burden of CRC compared with earlier periods when traditional dietary patterns were more prevalent. Other established risk factors include older age, family history of CRC, hereditary cancer syndromes, inflammatory bowel disease, smoking, alcohol consumption, and metabolic disorders. The interaction between genetic susceptibility and environmental exposures further contributes to individual variations in disease risk. Despite improvements in screening programs, diagnostic techniques, surgical procedures, chemotherapy, and targeted treatment, CRC remains associated with substantial morbidity and mortality. Early detection is particularly important because identifying precancerous lesions and localized malignancies can improve treatment outcomes and reduce disease-related deaths. Nevertheless, inequalities in screening participation, diagnostic capacity, healthcare infrastructure, and public awareness continue to affect prevention and early diagnosis, particularly in resource-limited settings [1]. Effective CRC prevention requires coordinated approaches that combine clinical services, population-based screening, health education, and reliable disease surveillance. Health informatics can support these efforts through electronic health records, cancer registries, data management, and analysis of epidemiological trends. Public health professionals contribute through risk assessment, screening initiatives, prevention campaigns, and evaluation of population-level outcomes. Medical secretaries also support this process by maintaining accurate patient records, coordinating appointments and screening referrals, organizing clinical documentation, and facilitating communication between patients and healthcare teams. Together, these roles can strengthen CRC surveillance, improve continuity of care, and support evidence-based prevention strategies.

Global Trends

Colorectal cancer (CRC) is the third most commonly diagnosed malignancy worldwide, with approximately 1.93 million new cases recorded in 2020, representing nearly 10% of all cancer diagnoses [1]. Its geographical distribution varies considerably according to socioeconomic development, lifestyle patterns, population characteristics, and access to healthcare. Countries with high human development index (HDI) values, particularly in Europe, North America, and Australia, report high age-standardized incidence rates (ASIRs), sometimes exceeding 40 cases per 100,000 individuals. Organized screening programs, including colonoscopy, have contributed to earlier diagnosis and improved survival in these regions. In the United States, screening has also contributed to declining CRC incidence and mortality. However, screening effectiveness depends on population participation, public awareness, healthcare accessibility, and socioeconomic circumstances [2]. In Asia, CRC incidence has increased alongside urbanization, population aging, and the adoption of Western dietary habits. Japan, South Korea, and Singapore report incidence rates comparable to those in Western countries, whereas India and Indonesia have lower reported rates but are experiencing epidemiological transitions. Among BRICS countries, China has experienced a substantial increase in CRC incidence, while Russia continues to report high rates. Brazil and South Africa demonstrate intermediate disease burdens, with variations between urban and rural populations. CRC also represents the second leading cause of cancer-related mortality worldwide, with approximately 935,000 deaths reported in 2020 [1]. The global age-standardized mortality rate (ASMR) is approximately 8.9 per 100,000 individuals. Eastern Europe and Eastern Asia experience particularly high mortality, reflecting disease incidence and differences in screening, diagnostic services, and treatment availability. Russia and Poland report mortality rates exceeding 15 per 100,000 individuals [2]. The consequences extend beyond patients, affecting family well-being, household income, and community productivity. In parts of Africa and Southeast Asia, limited healthcare infrastructure and late-stage diagnosis contribute to unfavorable outcomes [3].

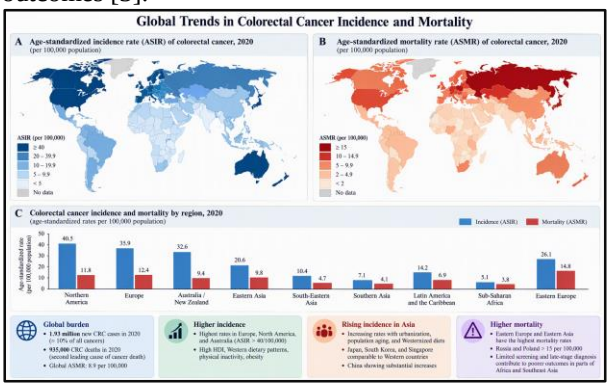


Figure-1: Global Trends, Epidemiology, Incidence, and Mortality of CRC.

Trends in Japan: Incidence

Japan provides an important example of CRC epidemiological trends. Before 2016, cancer incidence estimates relied primarily on prefectural registries. The introduction of the national cancer registry improved national data collection and reporting. Recent statistics recorded 147,725 CRC

cases, including 82,809 men and 64,915 women, accounting for 15.6% of all cancer diagnoses. Colon cancer represented 66.5% of cases, with a higher proportion among women than men [4]. Incidence increases from approximately 40 years of age and becomes more frequent in older populations. Regional differences are also evident, with Aomori, Okinawa, Akita, Iwate, and Tochigi reporting high incidence rates. These variations may reflect differences in dietary patterns, alcohol consumption, and other lifestyle exposures. Historical registry data indicate that CRC incidence increased until the late 1990s, followed by a declining trend. This pattern was more pronounced for colon cancer than rectal cancer [5]. Japan recorded 53,088 CRC deaths, representing 13.7% of all cancer-related deaths. Men accounted for 28,099 deaths and women for 24,989. Colon cancer represented 70.1% of CRC deaths, with a higher proportion among women [4]. Mortality increases with age, particularly after 50 years. Aomori, Okinawa, Akita, Hokkaido, and Kyoto report high age-adjusted mortality rates, with Aomori demonstrating a substantial burden in both incidence and mortality. Between 1979 and 2022, colon cancer mortality increased until the late 1990s before declining. Rectal cancer mortality also demonstrated a gradual decrease [4]. These trends suggest improvements in prevention, screening, and clinical management, although persistent regional and age-related differences indicate the need for continued surveillance and equitable access to diagnostic and treatment services.

Survival Rates for CRC

Survival outcomes for colorectal cancer (CRC) have improved over recent decades, largely because of advances in screening, diagnostic techniques, surgical procedures, and systemic treatment. Survival varies considerably according to the stage at diagnosis, highlighting the importance of early detection and timely intervention. The 5-year relative survival rate is approximately 90% for stage I CRC, compared with around 14% for stage IV disease [6]. This substantial difference reflects the influence of disease extent on treatment response and long-term prognosis. Screening programs contribute to improved survival by identifying precancerous lesions and detecting malignancies before they progress to advanced stages. In addition, improvements in surgical techniques, chemotherapy, radiotherapy, and multidisciplinary cancer care have expanded treatment options and improved disease control. Japanese cancer registry data demonstrate progressive improvements in CRC survival. Reports covering patients diagnosed between 1993 and 2011 show increases in 5-year relative survival for both colon and rectal cancer. For colon cancer, overall survival increased from 71.3% to 72.8% among men and from 66.1% to 69.4% among women between the 1993–1996 and 2009–2011 diagnosis periods. These findings indicate gradual improvements in outcomes across successive diagnostic periods [7, 8].

Stage-specific findings provide further evidence of progress. Among patients with localized colon cancer, the 5-year relative survival rate increased from 96.6% in 1993–1996 to 98.2% in 2009–2011. For localized rectal cancer, survival improved from 93.0% to 95.7%. Greater improvements were observed among patients with regional disease, including lymph node involvement or invasion of adjacent organs. Survival increased from 64.8% to 76.0% for colon cancer and from 55.3% to 74.0% for rectal cancer. Although prognosis remains poor in metastatic disease, survival also improved in this group. For distant-stage colon cancer, the 5-year relative survival rate increased from 8.2% to 16.2%, while rectal cancer survival increased from 8.1% to 19.7% [7, 8]. These trends emphasize the value of continued improvements in screening, staging, treatment access, and coordinated cancer care.

Risk Factors for CRC

Colorectal cancer (CRC) develops through interactions between genetic susceptibility, environmental exposures, metabolic abnormalities, and lifestyle factors. Identifying these risk factors is essential for determining individual risk, developing prevention strategies, and establishing appropriate screening schedules. Genetic predisposition and several modifiable factors contribute to CRC incidence and mortality, with varying degrees of evidence supporting their associations.

Genetic Predispositions

Hereditary factors account for a proportion of CRC cases and can substantially increase lifetime risk. Lynch syndrome is the most common hereditary CRC syndrome, accounting for approximately 3% of all cases. It results from pathogenic mutations in DNA mismatch repair genes, leading to microsatellite instability and an increased likelihood of malignant transformation. Affected individuals have an estimated 50–80% lifetime risk of developing CRC. Familial adenomatous polyposis (FAP), caused by mutations in the APC gene, is characterized by the development of numerous adenomatous polyps throughout the colon and rectum. Without preventive intervention, nearly all affected individuals develop CRC, commonly before the age of 40. In Japan, the increasing use of multigene cancer panel testing has improved the identification of hereditary cancer syndromes, including Lynch syndrome through secondary findings. Genetic counseling and regular surveillance are therefore important for individuals with suggestive family histories or confirmed pathogenic variants [9].

Dietary and Metabolic Factors

Dietary habits and obesity are established modifiable risk factors. High consumption of processed meat is associated with increased CRC risk, with each additional 50 g/day corresponding to a

relative risk (RR) of 1.17 (95% CI: 1.11–1.22) [10]. Sugar-sweetened beverage consumption has also been associated with early-onset CRC among women (HR = 1.16, 95% CI: 1.01–1.34) [11]. Similarly, every 5 kg/m² increase in body mass index is associated with a 5% increase in CRC risk (RR = 1.05, 95% CI: 1.03–1.07) [12]. Conversely, adequate vitamin D and calcium intake may have protective effects. The highest versus lowest vitamin D intake was associated with an RR of 0.67 (95% CI: 0.54–0.83), while each 300 mg/day increase in calcium intake was associated with an RR of 0.92 (95% CI: 0.89–0.95). Higher fermented dairy consumption was also associated with reduced risk (RR = 0.81, 95% CI: 0.74–0.90) [18, 19].

Alcohol and Smoking

Alcohol consumption demonstrates a dose-related association with CRC. Intake of 12.5–50 g/day was associated with an RR of 1.17 (95% CI: 1.11–1.24), while consumption exceeding 50 g/day was associated with estimates ranging from 1.44 to 1.52 across studies [13, 14]. Smoking also increases CRC risk. Current smokers have higher incidence and mortality than nonsmokers, with reported mortality RRs of 1.32 among men and 1.41 among women [15–17].

Chronic Diseases and Inflammation

Inflammatory bowel disease, particularly ulcerative colitis, increases CRC risk through prolonged intestinal inflammation. One study reported a 2.4-fold increased risk, rising to 5.7-fold among patients with extensive colitis [20]. Type 2 diabetes and insulin resistance are additional risk factors. Diabetes has been associated with increased CRC incidence (RR = 1.30) and mortality (RR = 1.26), while higher C-peptide levels have also demonstrated a positive association with CRC risk [21–23]. These findings support comprehensive risk assessment and targeted prevention strategies.

Diet and Lifestyle Factors in CRC

Dietary habits, lifestyle behaviors, metabolic conditions, and socioeconomic circumstances contribute substantially to colorectal cancer (CRC) development. Although genetic susceptibility and age are important determinants, several modifiable exposures influence CRC incidence and may affect disease progression and mortality. Evidence from international studies and Japanese population-based investigations demonstrates that the relationships between these factors and CRC can vary according to sex, geographical location, dietary patterns, and genetic background. Understanding these differences is important for developing effective prevention strategies and identifying populations that may benefit from targeted screening.

Processed Foods, High-Sugar Beverages, and Obesity

Dietary patterns characterized by high consumption of processed foods, sugar-sweetened beverages, and excessive energy intake are associated with an increased risk of CRC. Processed meat has been classified as a Group 1 carcinogen by the International Agency for Research on Cancer because of sufficient evidence linking its consumption to colorectal carcinogenesis. A comprehensive meta-analysis reported that each additional 50 g/day of processed meat consumption was associated with a 17% increase in CRC risk (RR = 1.17, 95% CI: 1.11–1.22) [10]. Potential mechanisms include exposure to N-nitroso compounds, heterocyclic amines, and polycyclic aromatic hydrocarbons, which may contribute to DNA damage and abnormal cellular proliferation. These compounds can arise during meat processing, preservation, or high-temperature cooking. Sugar-sweetened beverages represent another dietary exposure associated with CRC, particularly early-onset disease. A prospective investigation reported that daily consumption of these beverages was associated with a 16% higher risk of early-onset CRC among women (HR = 1.16, 95% CI: 1.01–1.34) [11]. High consumption may contribute to excessive caloric intake, weight gain, insulin resistance, and metabolic disturbances. These processes may create conditions that promote colorectal carcinogenesis, although the association may also reflect other dietary and lifestyle characteristics.

Obesity is an established modifiable risk factor for CRC. A meta-analysis demonstrated that each 5 kg/m² increase in body mass index was associated with a 5% increase in CRC risk (RR = 1.05, 95% CI: 1.03–1.07) [12]. Adipose tissue can influence cancer development through insulin resistance, increased circulating insulin and insulin-like growth factors, chronic low-grade inflammation, and altered adipokine production. These changes may stimulate cellular proliferation and interfere with normal mechanisms regulating cell growth and apoptosis. Japan provides an important example of population-specific dietary associations. Although Westernized dietary habits, including greater consumption of processed foods and sugary beverages, have become more common, obesity prevalence remains lower than in the United States. Nevertheless, Japan has reported a higher CRC incidence rate than the USA in some comparisons. This difference suggests that obesity and Western dietary patterns alone cannot explain international variations in CRC incidence. Genetic susceptibility, environmental exposures, gut microbiota, population age structures, and screening practices may also contribute. The Japan Public Health Center-based Prospective (JPHC) Study identified differences in the association between meat consumption and CRC according to sex. High red meat consumption was significantly associated with increased colon cancer risk among Japanese women, whereas high total meat consumption was associated with increased colon

cancer risk among men. However, processed meat consumption was not significantly associated with CRC risk in either sex [24]. Similarly, the JPHC investigation of sugar-sweetened beverages found no significant association with overall CRC risk among Japanese men or women. Nevertheless, higher consumption was associated with increased colon cancer risk, particularly proximal colon cancer, among women [25]. These findings demonstrate that dietary exposures may have different effects across populations and reinforce the importance of considering sex-specific and anatomical differences when evaluating CRC risk.

Alcohol Intake

Alcohol consumption is an established risk factor for CRC, with epidemiological evidence supporting a dose-response relationship. Alcohol intake may contribute to carcinogenesis through several mechanisms, including the production of acetaldehyde, a toxic metabolite capable of damaging DNA. Alcohol metabolism can also generate oxidative stress, interfere with folate metabolism, and affect the integrity of the intestinal mucosa. These biological effects may contribute to the development of malignant changes in colorectal tissues. A meta-analysis reported that individuals consuming at least 50 g of alcohol daily had a 52% higher risk of CRC than nondrinkers (RR = 1.52, 95% CI: 1.27–1.81) [13]. Another meta-analysis demonstrated that moderate consumption of 12.5–50 g/day was associated with a 17% increased risk (RR = 1.17, 95% CI: 1.11–1.24), while consumption exceeding 50 g/day was associated with a 44% increase (RR = 1.44, 95% CI: 1.25–1.65) [14]. Although the estimates differ between studies, both findings support a positive association between alcohol intake and CRC risk. Evidence from pooled Japanese cohort studies further demonstrates this relationship. Among Japanese men and women, CRC risk increased by approximately 10% for every additional 15 g of daily alcohol consumption. This association was observed for both colon and rectal cancers [26]. Comparisons with Western populations have suggested that the relationship between alcohol consumption and CRC may be stronger among Japanese individuals. Differences in alcohol-metabolizing enzymes, particularly genetic variations affecting acetaldehyde metabolism, may partly explain this observation. However, environmental exposures, drinking patterns, dietary habits, and other lifestyle characteristics may also contribute. Further population-based research is required to clarify these differences.

Smoking

Tobacco smoking contributes to CRC development and mortality through exposure to carcinogenic compounds that can enter the circulation and reach colorectal tissues. These substances may cause DNA damage, promote oxidative stress, and interfere with cellular repair mechanisms. Smoking may also influence inflammatory pathways and the composition of the intestinal microbiota, potentially contributing to colorectal carcinogenesis. A meta-analysis by Botteri et al. [15] reported that smoking was associated with an approximately 14–17% increase in overall CRC risk. The strength of this association may vary according to smoking intensity, duration, and tumor characteristics, including molecular subtype. Evidence from prospective studies in Japan and the United States supports the relationship between smoking and CRC incidence and mortality [16, 17]. Japanese cohort findings indicated higher CRC incidence among current smokers, particularly men, although the association among women was less consistent. Additional evidence demonstrated increased CRC-related mortality among current smokers compared with never-smokers, with relative risks of 1.32 among men and 1.41 among women [17]. The association between smoking and CRC is influenced by cumulative exposure. Individuals who smoke more cigarettes daily or continue smoking for longer periods may experience greater carcinogenic exposure. Conversely, smoking cessation can reduce several smoking-related health risks over time. Although the extent and timing of CRC risk reduction after cessation require further investigation, tobacco control remains an important component of cancer prevention. Public health strategies should therefore incorporate smoking prevention, cessation counseling, and awareness of the long-term consequences of tobacco exposure.

Vitamin D, Calcium, and Fermented Foods Intake

Certain dietary nutrients and fermented foods may have protective associations with CRC. Vitamin D is involved in cellular differentiation, immune regulation, and the control of cell proliferation. Its potential protective effects have been investigated through studies assessing dietary intake and circulating 25-hydroxyvitamin D [25(OH)D] concentrations. A meta-analysis reported that higher vitamin D intake was associated with a 12% reduction in CRC risk, while higher circulating 25(OH)D levels were associated with a 33% reduction. Furthermore, each 10 ng/mL increase in blood 25(OH)D concentration was associated with a 26% lower CRC risk [18]. These findings suggest a potential relationship between vitamin D status and colorectal health, although observational associations do not establish that supplementation prevents CRC. Calcium intake has also been inversely associated with CRC risk. A meta-analysis reported that each additional 300 mg/day of calcium intake was associated with an 8% reduction in CRC risk (RR = 0.92, 95% CI: 0.89–0.95) [12]. Calcium may contribute to colorectal protection by binding bile acids and free fatty acids in the intestinal lumen, thereby reducing their potential harmful effects on the colonic epithelium. It may also influence cellular signaling, differentiation, and proliferation.

Japanese population-based evidence has produced somewhat different findings. The JPHC Study did not identify a statistically significant association between vitamin D intake and CRC risk among men or women when considered independently. However, among men, participants with high combined vitamin D and calcium intake had a lower CRC risk than those with lower intake of both nutrients [27]. This observation suggests that combined dietary patterns may be relevant when evaluating nutritional factors and CRC risk. Differences in baseline nutrient intake, food sources, environmental exposure, and population characteristics may also explain variations between Japanese and Western studies. Fermented foods have attracted attention because of their potential effects on intestinal microbiota and gastrointestinal health. Products such as yogurt and kefir contain microorganisms and fermentation-derived compounds that may influence microbial diversity, intestinal barrier function, and inflammatory activity. A systematic review and meta-analysis of cohort studies found that individuals with the highest fermented dairy intake had a 19% lower CRC risk than those with the lowest intake (RR = 0.81, 95% CI: 0.74–0.90) [19]. Although these findings support a potentially protective association, further research is needed to identify the specific microorganisms, dietary components, and biological pathways responsible for this relationship.

Inflammatory Bowel Disease

Inflammatory bowel disease (IBD), particularly ulcerative colitis (UC), is an established risk factor for CRC. Persistent inflammation of the intestinal mucosa can promote repeated epithelial injury, cellular regeneration, genetic alterations, and dysplasia. Unlike sporadic CRC, which commonly develops through the adenoma–carcinoma sequence, inflammation-associated CRC may arise through a distinct progression involving chronic inflammatory damage and early molecular abnormalities. A meta-analysis of population-based cohort studies reported a pooled standardized incidence ratio (SIR) of 2.4 (95% CI: 2.1–2.7) among patients with UC, indicating approximately 2.4 times the CRC risk observed in the general population. Approximately 1.6% of patients developed CRC during an average follow-up of 14 years. The risk was higher among men than women, with SIRs of 2.6 and 1.9, respectively. Patients with extensive colitis had a substantially greater risk, with an SIR of 4.8 [20]. These findings emphasize the importance of disease duration, inflammatory extent, and cumulative inflammatory burden in determining CRC risk among individuals with UC. The clinical characteristics and outcomes of IBD-associated CRC may also differ from those of sporadic disease. Birch et al. [28] examined 5,141 patients with IBD-associated CRC and compared them with 385,473 patients with non-IBD CRC. Patients with IBD-associated CRC were diagnosed at a younger median age than those without IBD (66 versus 72 years). They were also more likely to have right-sided tumors and emergency presentations, reported in 25.1% compared with 16.7% of non-IBD cases. Furthermore, synchronous and metachronous tumors were more frequent among patients with IBD-associated CRC, with rates of 3.2% versus 1.6% and 1.7% versus 0.9%, respectively. The reported 2-year survival outcomes also differed between the groups, with rates of 15% for stage I and 70.7% for stage III among IBD-associated CRC patients. These findings highlight the clinical complexity of CRC arising in the context of chronic intestinal inflammation.

At the molecular level, chronic inflammation, genetic mutations, and immune dysregulation contribute to malignant transformation in IBD. Porter et al. [29] discussed the involvement of early TP53 mutations and inflammatory signaling pathways, particularly nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB) and interleukin-6/signal transducer and activator of transcription 3 (IL-6/STAT3). These pathways can promote inflammatory mediator production, epithelial cell survival, proliferation, and resistance to apoptosis. Persistent activation may therefore facilitate the transition from inflammation to dysplasia and invasive carcinoma [29]. Regular surveillance is essential for patients with longstanding or extensive colitis, particularly those with additional risk factors. Colonoscopic assessment allows clinicians to identify dysplasia and early malignant lesions before clinical progression. Surveillance intervals should reflect disease duration, inflammatory extent, previous dysplasia, and individual clinical risk. Effective control of intestinal inflammation is also an important component of long-term CRC risk management.

Diabetes and Insulin Resistance

Type 2 diabetes mellitus and insulin resistance are associated with increased CRC incidence and mortality. Insulin resistance may result in compensatory hyperinsulinemia, which can stimulate insulin and insulin-like growth factor signaling pathways involved in cellular proliferation and survival. Hyperglycemia, chronic low-grade inflammation, and metabolic disturbances may further contribute to an environment that supports colorectal carcinogenesis. A meta-analysis by Larsson et al. [21], involving more than 2.5 million participants from 15 studies, demonstrated that diabetes was associated with a 30% increase in CRC incidence (RR = 1.30, 95% CI: 1.20–1.40). This association was observed in both sexes and across different colorectal subsites. Diabetes was also associated with a 26% increase in CRC-related mortality (RR = 1.26, 95% CI: 1.05–1.50), indicating that its influence may extend beyond cancer

development to disease outcomes [21]. Additional evidence comes from a 20-year English cohort study involving 137,804 individuals with type 2 diabetes. The study reported a standardized mortality ratio of 2.40 (95% CI: 2.26–2.54) for CRC, indicating substantially higher CRC mortality than expected in the general population. The association was particularly evident among older adults [22]. Differences in metabolic control, diabetes duration, obesity, comorbidities, and healthcare access may contribute to the observed variation in outcomes. The JPHC Study Group also investigated the relationship between hyperinsulinemia and CRC using plasma C-peptide concentrations as an indicator of insulin secretion. Among men, increasing C-peptide levels were associated with progressively higher CRC risk, with odds ratios of 2.3, 2.8, and 3.2 across ascending quartiles. The association was stronger for colon cancer than for rectal cancer [23]. These findings support the potential contribution of insulin-related metabolic pathways to colorectal carcinogenesis. They also highlight the importance of addressing diabetes, insulin resistance, and obesity through lifestyle modification, metabolic monitoring, and appropriate clinical management.

Socioeconomic Factors

Socioeconomic conditions influence CRC prevention, diagnosis, treatment, and survival. Income, educational attainment, employment conditions, and access to healthcare can affect an individual's ability to adopt preventive behaviors and participate in screening programs [2, 6]. People with lower socioeconomic status may experience barriers to preventive healthcare, including limited screening availability, financial constraints, reduced health literacy, and difficulties accessing specialist services. These barriers can delay diagnosis and increase the likelihood that CRC is detected at an advanced stage. Educational attainment may influence awareness of CRC symptoms, screening recommendations, dietary risk factors, and the importance of seeking medical assessment. Income and employment conditions can also affect dietary choices, opportunities for physical activity, and access to healthcare facilities. Consequently, socioeconomic disadvantage may interact with behavioral and environmental exposures to produce differences in CRC incidence and outcomes. Within Japan, regional differences in dietary habits, healthcare access, screening participation, and population characteristics may contribute to variations in CRC incidence and mortality. Improving screening coverage, strengthening public health education, and reducing barriers to diagnostic services are important measures for addressing these inequalities. Health policies should prioritize populations with limited access to preventive services and ensure that individuals with elevated risk receive appropriate surveillance and follow-up. Overall, CRC risk reflects the combined influence of inherited susceptibility, dietary exposures, lifestyle behaviors, chronic inflammatory conditions, metabolic disorders, and socioeconomic circumstances. Although genetic predisposition and certain chronic diseases cannot always be prevented, several modifiable factors offer opportunities for risk reduction. Population-based prevention strategies should therefore promote healthy dietary patterns, weight management, smoking cessation, reduced alcohol consumption, physical activity, and appropriate management of metabolic and inflammatory diseases. Integrating these measures with risk-based screening and equitable access to healthcare may contribute to reducing CRC incidence and improving long-term outcomes.

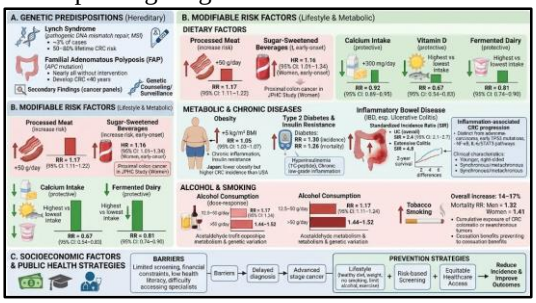


Figure-2: Risk Factors of CRC.

Public Health, Health Information, and Medical Secretary Roles in CRC Prevention

Public health initiatives, health information systems, and medical secretarial services play complementary roles in colorectal cancer (CRC) prevention, screening, and early detection. Effective prevention requires coordinated efforts to increase public awareness, improve screening participation, reduce barriers to healthcare access, and ensure appropriate follow-up. Educational campaigns and community outreach programs, particularly those adapted to cultural needs and supported by community health workers, have improved screening uptake among underserved populations [30]. These interventions are important for encouraging individuals to participate in screening and seek medical assessment when symptoms arise. Japan's population-based CRC screening program has contributed to early cancer detection and reduced mortality. However, further integration of screening records with the national cancer registry is needed to strengthen population surveillance and evaluate program outcomes [31]. Important challenges include suboptimal participation in fecal immunochemical test (FIT) screening, inadequate follow-up among individuals with positive results, and regional differences in screening implementation. Targeted education, smartphone-based reminders, and improved coordination between screening centers and diagnostic facilities may help address these limitations. Artificial intelligence-assisted quality assurance in colonoscopy may also improve examination standards and support the detection of colorectal lesions.

Health information management is essential for the effective organization and evaluation of CRC prevention programs. Electronic health records, cancer registries, screening databases, and clinical reporting systems enable healthcare organizations to collect, store, analyze, and exchange relevant patient information. These systems can help identify individuals who are overdue for screening, monitor positive FIT results, track referrals for colonoscopy, and evaluate diagnostic and treatment outcomes. Integrating screening data with cancer registries also supports epidemiological research by providing information on incidence, stage at diagnosis, geographical distribution, and survival. Furthermore, big data analytics and machine learning can help identify individuals with elevated CRC risk and support the development of targeted screening strategies [33]. However, the effectiveness of these technologies depends on data accuracy, interoperability, confidentiality, and appropriate clinical oversight. Medical secretaries contribute to CRC prevention through administrative coordination and accurate documentation. Their responsibilities may include registering patients, scheduling screening appointments, maintaining medical records, processing referrals, organizing diagnostic reports, and communicating appointment information. They can also support follow-up systems by identifying missed appointments, notifying patients of scheduled investigations, and ensuring that positive screening results are directed to the appropriate clinical teams. Although medical secretaries do not make clinical decisions, their work helps maintain continuity of care and reduces administrative delays that may interfere with timely diagnosis. Research initiatives further support improvements in CRC prevention and early detection. Collaboration between academic institutions, healthcare providers, and industry has advanced screening technologies and precision medicine. The Cancer Moonshot initiative in the United States represents an example of coordinated efforts to accelerate cancer research and improve early detection [32]. Continued investigation into cost-effectiveness, screening participation, artificial intelligence, and personalized risk prediction is necessary to guide future policies. Integrating public health programs, reliable information systems, and effective medical secretarial coordination can strengthen screening pathways, improve follow-up, and promote equitable CRC prevention.

Conclusion:

Colorectal cancer represents a substantial public health challenge influenced by genetic, dietary, metabolic, inflammatory, and socioeconomic factors. Early detection remains essential because survival differs considerably according to disease stage at diagnosis. Population-based screening, lifestyle modification, genetic assessment of high-risk individuals, and appropriate surveillance for patients with inflammatory bowel disease and diabetes are important prevention strategies. Evidence from Japan highlights the influence of population-specific exposures and the need to strengthen screening participation and follow-up. Health information systems and integrated cancer registries can improve surveillance, data accuracy, and identification of individuals requiring further assessment. Medical secretaries also support continuity of care through accurate documentation, appointment scheduling, referral coordination, and follow-up communication. Collaboration among public health professionals, healthcare providers, information specialists, and medical secretaries can strengthen CRC prevention programs and improve access to timely diagnosis.

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