



Risk factors and prevention of colonic diseases: Modern approaches and perspectives (literature review)

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Abstract. Diseases of the large intestine continue to make a significant contribution to population morbidity and mortality, which determines their high medical and social importance. Since the beginning of the 21st century, an increase in the prevalence of colorectal pathology has been observed, along with a shift in the age structure of patients toward younger groups, which highlights the relevance of analysing factors influencing the development of these diseases and approaches to their prevention. The aim of this review was to summarise and systematise

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current scientific evidence on risk determinants and strategies for reducing the burden of major diseases of the large intestine. An analysis of 52 publications was conducted, including systematic reviews, meta-analyses, large cohort studies, and randomised controlled trials retrieved from PubMed, Scopus, Web of Science, and Google Scholar databases. The review included studies published between 1990 and 2025, with 53.8% of the sources dated from 2020 to 2025. The literature analysis demonstrated that dietary patterns, excess body weight, low physical activity, smoking, and alcohol consumption play a leading role in the development of large intestinal diseases. Alterations in gut microbiota composition and chronic inflammation are considered key pathogenic mechanisms. A reduction in disease incidence and mortality has been confirmed for screening strategies based on endoscopic and non-invasive methods, as well as for selected pharmacological and technological approaches, including computer-assisted endoscopic systems. The findings of this review may be applied in clinical practice, planning of screening programs, and development of prevention-oriented interventions within public health systems

Keywords: colorectal cancer; inflammatory bowel disease; diverticular disease; polyposis; lifestyle; screening

Introduction

Large intestine diseases remain one of the most significant problems in contemporary gastroenterology and public health, forming a substantial burden of morbidity and mortality worldwide. This group of pathologies is characterised by considerable medical, social, and economic consequences associated with the prolonged course of diseases, the need for expensive diagnostics and treatment, as well as a reduction in patients' quality of life. This necessitates an in-depth analysis of risk factors and the development of effective prevention measures.

In a review work by N. Keum & E. Giovannucci [1], based on an analysis of global epidemiological data and the results of large cohort studies, it was shown that colorectal cancer (CRC) consistently ranks third in prevalence and second among the causes of cancer mortality. The authors emphasise that approximately 1.9 million new cases of CRC and more than 900,000 deaths are registered annually, with modifiable factors, including dietary patterns, obesity, physical inactivity, and smoking, playing a key role in shaping the risk of the disease. In turn, M. Arnold *et al.* [2], in a multicentre analysis of global trends in CRC incidence and mortality, demonstrated pronounced regional differences in disease dynamics. The authors note a decline in indicators in high-income countries, which is associated with the implementation of screening and prevention programmes, whereas in developing regions, an increase in incidence is observed, linked to urbanisation, changes in eating habits, and lifestyle patterns of the population.

A systematic review by H.N. Humphrey *et al.* [3], devoted to the genetic, epigenetic, and environmental factors of large intestine diseases, showed that a shift is occurring in the age structure of patients with colorectal pathology, including an increase in the proportion of young and middle-aged individuals. The authors emphasise that a key role in this process is played by environmental and lifestyle factors, such as dietary patterns, physical inactivity, and metabolic disorders, which indicates the growing importance of modifiable risk factors in the pathogenesis of large intestine diseases. In a systematic review and meta-analysis by

H. Hua *et al.* [4], data from epidemiological and observational studies directly devoted to risk factors for early-onset colorectal cancer were summarised. The authors showed that early-onset CRC is significantly associated with obesity, metabolic syndrome, chronic inflammation, and changes in eating behaviour, which makes it possible to consider early-onset CRC as a separate clinical-epidemiological phenotype closely linked to modifiable risk factors.

Alongside oncological diseases, inflammatory bowel diseases (IBD), primarily Crohn's disease and ulcerative colitis, are of substantial clinical and social significance. In a review work by J.D. Feuerstein & A.S. Cheifetz [5], data from epidemiological and clinical studies were summarised, according to which IBD is characterised by a chronic relapsing course and increasing prevalence, which leads to prolonged inflammation of the colonic mucosa and the formation of an oncogenic microenvironment. In a systematic review by S.C. Ng *et al.* [6], it was shown that over recent decades, IBD incidence has significantly increased in regions previously considered low-endemic, including Asian countries and developing states. The authors link this increase to urbanisation, changes in eating habits, and exposure to environmental factors, emphasising the globalisation of IBD as a public health problem. In a meta-analysis by M. Zamani *et al.* [7], the risk of developing colorectal cancer in patients with IBD was assessed depending on age of onset and disease duration. It was shown that the greatest increase in CRC risk is observed in patients with early-onset IBD and long disease duration, which underscores the need for early initiation of surveillance and prevention programmes in this group of patients. In a clinical review by S.M. Adams *et al.* [8], it is emphasised that patients with ulcerative colitis, especially those with extensive colonic involvement and long disease duration, require regular endoscopic surveillance due to the increased risk of developing colorectal cancer. The authors indicate that timely detection of dysplasia and early neoplastic changes is a key element of secondary prevention in this category of patients.

Diverticular disease makes a significant contribution to the structure of large intestine diseases, its prevalence progressively increasing with age and reaching 50-70% in individuals over 60 years of age. In a review work by L. Strate & A. Morris [9], diverticular disease is considered as a multifactorial condition in the pathogenesis of which age-related changes, dietary patterns low in dietary fibre, obesity, and chronic low-grade inflammation play an important role. The authors emphasise that although most cases are asymptomatic, complicated forms of the disease, including diverticulitis and intestinal bleeding, impose a significant clinical and economic burden on the healthcare system. In a contemporary review by M. Jacobsson *et al.* [10], diverticular disease is considered in the context of general prevention strategies for large intestine diseases. The authors note that lifestyle modification, including dietary adjustment, increased physical activity, and control of metabolic risk factors, may reduce the frequency of diverticular disease complications and simultaneously have a favourable effect on the risk of developing colorectal cancer.

In the pathogenesis of colorectal cancer, a key role is played by the sequential transformation of benign epithelial neoplasms into malignant tumours, realised through the adenoma-carcinoma cascade. In a review work by R. Monreal-Robles *et al.* [11], the morphological and molecular features of serrated polyps of the large intestine, as well as their significance in an alternative pathway of carcinogenesis, are examined in detail. The authors emphasise that serrated lesions may remain undiagnosed for a long time, which increases the risk of late detection of neoplasia and underscores the importance of timely endoscopic diagnosis. The effectiveness of preventive interventions aimed at detecting and removing precancerous lesions was convincingly demonstrated in a large prospective study by A.G. Zaubler *et al.* [12]. The authors showed that colonoscopic polypectomy is accompanied by a significant reduction in colorectal cancer mortality in the long term, which served as a basis for the formation of contemporary CRC screening and secondary prevention programmes and emphasises the key role of an integrated approach to managing risk factors for large intestine diseases. The aim of the study was to analyse and structure the literature data reflecting contemporary understanding of the factors associated with the development of and opportunities for reducing the burden of large intestine diseases, including colorectal cancer, inflammatory bowel diseases, diverticular disease, and polyposis.

Materials and Methods

The present work is a review of the scientific literature devoted to the risk factors and prevention of large intestine diseases, including colorectal cancer, inflammatory bowel diseases (Crohn's disease and ulcerative colitis), diverticular disease, and polyposis. The search,

selection, and analysis of sources were carried out using the principles of a systematic literature review and elements of the PRISMA methodology. The search for publications was conducted in the international bibliographic databases PubMed/MEDLINE, Scopus, and Web of Science, as well as using Google Scholar to identify additional relevant sources. The review includes articles published between 1990 and 2025. The choice of this time range was determined by the need to cover both fundamental studies that shaped contemporary understanding of the pathogenesis and prevention of large intestine diseases, and current systematic reviews, meta-analyses, and clinical studies reflecting contemporary epidemiological trends and preventive approaches. Of the total of 52 included publications, 28 (53.8%) were published between 2020 and 2025, which meets the requirements of relevance for review studies and makes it possible to reflect contemporary directions of scientific inquiry in gastroenterology and oncology. The search strategy was based on the use of combinations of key terms reflecting nosological forms of diseases, risk factors, and preventive interventions, including: colorectal cancer, inflammatory bowel disease, Crohn's disease, ulcerative colitis, diverticular disease, polyps, risk factors, prevention, screening, diet, obesity, physical activity, microbiota, aspirin, colonoscopy, stool-based tests, and artificial intelligence. The search was conducted without language restrictions; however, the analysis included primarily publications in English.

The selection of sources was carried out in stages. At the initial stage, an analysis of titles and abstracts was conducted to assess the relevance of publications to the topic of the review. Next, a full-text review of articles was performed using predetermined inclusion and exclusion criteria. The review included publications presented in peer-reviewed scientific journals and containing quantitative data on risk factors, preventive interventions, or the effectiveness of screening methods. Priority was given to systematic reviews, meta-analyses, large cohort studies, and randomised controlled trials. The study considered fundamental and experimental studies at the molecular level devoted to the mechanisms of carcinogenesis and inflammatory processes in the large intestine, if their results allowed the interpretation of the influence of risk factors and preventive interventions. Duplicate publications, studies with limited methodological validity, as well as works not corresponding to the aims and objectives of the review, were excluded.

The quality assessment of the included sources was carried out considering the PRISMA recommendations and generally accepted approaches to the critical appraisal of scientific publications. For systematic reviews and meta-analyses, the completeness of the search strategy, the transparency of the selection criteria, the methods for assessing bias, and the adequacy of the statistical analysis were analysed. For original studies,

the study design, characteristics of the study population, correctness of exposure and outcome definitions, as well as the applied methods of statistical data processing were considered. The systematisation of information was carried out by extracting and summarising the methodological characteristics and key directions of analysis from each publication. All sources were grouped according to the main thematic areas: colorectal cancer, inflammatory bowel diseases, diverticular disease, and polyposis. The choice of these nosologies was determined by their high prevalence, significant contribution to the structure of large intestine morbidity, and the presence of partially shared pathogenetic mechanisms associated with chronic inflammation, disturbances of the intestinal microbiota, and the impact of lifestyle factors. At the same time, the largest number of available epidemiological and preventive studies is devoted to colorectal cancer, which determined a more detailed consideration of risk factors and preventive strategies for this nosology within the structure of the review. Within the framework of these directions, the data were analysed considering the types of risk factors (modifiable and non-modifiable), levels of prevention (primary, secondary, and tertiary), as well as the diagnostic and preventive technologies used, which ensured a structured and comprehensive character of the review.

Risk factors for the development of large intestine diseases

According to the global cancer statistics GLOBOCAN 2020, presented by H. Sung *et al.* [13], colorectal cancer

remains one of the leading causes of cancer morbidity and mortality worldwide, with pronounced interregional differences in indicators pointing to the potential role of modifiable risk factors and the availability of preventive programmes. Dietary and behavioural factors are the leading determinants of the risk of large intestine diseases (Table 1). According to a global meta-analysis by N. Keum & E. Giovannucci [1], excessive consumption of red and processed meat is significantly associated with an increased risk of CRC: the relative risk (RR) is 1.16 (95% CI 1.08-1.26) per 50 g of processed meat per day. By the International Agency for Research on Cancer, according to a study presented by V. Bouvard *et al.* [14], a comprehensive analysis of epidemiological, experimental, and mechanistic data on the consumption of red and processed meat was conducted. The authors showed that for processed meat, there is convincing evidence of a carcinogenic effect in humans, as a result of which it was classified as a Group 1 carcinogen. For red meat, the evidence base was recognised as limited, but sufficient to classify it as a probable carcinogen (Group 2A). In a large meta-analysis of prospective studies by Z. Ungvari *et al.* [15], it was shown that consumption of red and processed meat is statistically significantly associated with an increased risk of CRC, as well as separately of colon and rectal cancer. According to the expert report "Diet, nutrition, physical activity and colorectal cancer" [16], diets high in saturated fats and refined carbohydrates were associated with insulin resistance and chronic low-grade inflammation, creating a favourable background for carcinogenesis.

Table 1. Main risk factors for diseases of the large intestine

Group of factors	Specific factors	Mechanisms of influence / clinical characteristics	Level of evidence (according to review data)
Dietary	Excessive consumption of red and processed meat	Formation of nitrosamines, heterocyclic amines, oxidative stress, DNA damage of the epithelium	RR = 1.16 for every 50 g of processed meat per day (WCRF/AICR)
	Low consumption of fibre and wholegrain products	Slowed transit, reduced binding of carcinogens, decreased production of short-chain fatty acids	Reduction in CRC risk by approximately 10-20% with an increase in fibre intake by 10 g/day
	High consumption of saturated fats and refined carbohydrates	Insulin resistance, chronic inflammation	Moderate association with CRC and obesity
Behavioural	Smoking	Increased risk of Crohn's disease and CRC, activation of inflammation, mutagenic effect	Risk of IBD is 2-2.5 times higher in smokers
	Alcohol abuse (>30 g ethanol/day)	Mucosal damage, folate deficiency, acetaldehyde-induced stress	Approximately 20% increase in CRC risk
	Low physical activity	Slowing of peristalsis, hyperinsulinaemia, obesity	Regular exercise ≥150 min/week reduces risk by 20-25%
Metabolic	Obesity (BMI > 30)	Increased IL-6, TNF-α, IGF-1, stimulation of proliferation and inhibition of apoptosis	Approximately 30% increase in CRC risk
Genetic	Hereditary syndromes (Lynch syndrome, FAP)	APC, KRAS, TP53 mutations; adenoma-carcinoma cascade	5% of CRC cases are hereditary
Age-related	Older than 50 years	Accumulation of mutations, weakened DNA repair	Incidence increases 4-5-fold compared with individuals <40 years
Microbiological / inflammatory	Microbiota dysbiosis (↑ <i>Fusobacterium</i> , ↓ <i>Bifidobacterium</i>)	Activation of NF-κB, STAT3, increased IL-6 and TNF-α, epithelial damage	10-fold increase in <i>F. nucleatum</i> concentration in tumour tissues

Source: developed by the authors based on [1,5,8,16-24]

In the same report, it was shown that, in contrast to dietary patterns high in red and processed meat, a high level of consumption of dietary fibre and whole grains is associated with a pronounced preventive effect with respect to colorectal cancer. It was shown that an increase in dietary fibre intake of every 10 g per day is accompanied by a reduction in CRC risk of approximately 10%, whereas regular consumption of ≥ 90 g of whole grains per day is associated with a decrease in disease risk of 15-20%. Similar findings are presented in the review work by N. Keum & E. Giovannucci [1], where a dose-dependent relationship between fibre consumption and CRC risk reduction is emphasised. The biological mechanisms of the protective effect of dietary fibre are examined in detail in the work of P. Louis & H.J. Flint [25], who showed that the fermentation of dietary fibre by the gut microbiota leads to the formation of short-chain fatty acids, primarily butyrate and propionate. According to the authors, these metabolites accelerate intestinal transit, reduce the contact time of the colonic mucosa with potential carcinogens, and possess pronounced anti-inflammatory and antiproliferative properties with respect to the colonic epithelium, playing an important role in the modulation of carcinogenesis. Similarly, M.E. Arayici *et al.* [26] presented data in a systematic review and meta-analysis indicating that higher consumption of dietary fibre (including soluble and insoluble) is associated with a reduced risk of colorectal cancer.

According to a meta-analysis by C. Simillis *et al.* [27], extreme values of body mass index, in particular pronounced underweight, were associated with worse long-term outcomes after surgical treatment of colorectal cancer, which underscores the prognostic significance of nutritional status in patients with CRC. At the same time, obesity is considered as an independent risk factor for the development of colorectal cancer: in a large population study by K. Bhaskaran *et al.* [19], it was shown that with a body mass index >30 , the risk of CRC increases by approximately 30% compared to individuals with normal body weight [19]. In the review work by N. Keum and E. Giovannucci [1], it was emphasised that excess adipose tissue functions as an active endocrine organ, producing pro-inflammatory cytokines (including IL-6 and TNF- α) and increasing levels of insulin and insulin-like growth factor-1 (IGF-1), which promotes increased proliferation and suppression of apoptosis of colonic epithelial cells. The level of physical activity plays a significant role in modifying CRC risk. In a systematic review by R.E. Eyl *et al.* [17], it was shown that regular aerobic physical activity, equivalent to at least 150 minutes of moderate exercise per week, is associated with an improvement in the metabolic profile and functional status of patients, and according to epidemiological studies – with a reduction in the risk of developing colorectal cancer by 20-25%.

Smoking is an established risk factor not only for colorectal cancer but also for inflammatory bowel diseases. In clinical reviews by J.D. Feuerstein & A.S. Cheifetz [5] as well as S.M. Adams *et al.* [8], it was shown that smoking increases the risk of developing Crohn's disease by 2-2.5 times and is associated with a more severe course of the disease, whereas smoking cessation helps to reduce the frequency of relapses and exacerbations. Alcohol abuse (consumption of more than 30 g of ethanol per day) is also considered as a factor increasing the risk of colorectal cancer. According to an expert report conducted by "Diet, nutrition, physical activity and colorectal cancer" [16], regular alcohol consumption is associated with an increase in CRC risk by approximately 20%, especially in conditions of concomitant folate deficiency.

Dysbiosis of the gut microbiota is currently considered as a key pathogenetic link not only in inflammatory bowel diseases but also in colorectal cancer. In a review work by J. Gagnière *et al.* [28], it was shown that an imbalance in the gut microbiota is accompanied by a reduction in protective commensals and an increase in the proportion of opportunistic microorganisms, contributing to chronic inflammation and colon carcinogenesis. Similar conceptual provisions are presented in a fundamental review by W.S. Garrett [29], where the role of the microbiota as an active regulator of immune and oncogenic processes in the intestine is emphasised. A decrease in the production of short-chain fatty acids, primarily butyrate and propionate, leads to a loss of the anti-inflammatory and antiproliferative effects of the microbiota and increases the vulnerability of the colonic epithelium to DNA damage. Against this background, an increase in the representation of *Fusobacterium nucleatum*, *Escherichia coli*, and *Bacteroides fragilis* is associated with the activation of NF- κ B and STAT3 signalling pathways, increased production of pro-inflammatory cytokines (IL-6, TNF- α), and the formation of a pro-oncogenic microenvironment. In a systematic review by G. Tabowei *et al.* [30], it was shown that dysbiosis of the gut microbiota in colorectal cancer is characterised by a decrease in microbiota diversity, a reduction in the proportion of protective commensals (in particular, *Bifidobacterium* and *Lactobacillus*), and a relative increase in potentially pro-oncogenic taxa, including *Fusobacterium nucleatum* and toxigenic strains of *Escherichia coli*. These observations were further confirmed in a meta-analysis of metagenomic datasets by N. Avuthu & C. Guda [31], where CRC microbial signatures, including enrichment of *Fusobacterium*, *Parvimonas*, and *Peptostreptococcus*, as well as a reduction in the representation of butyrate-producing bacteria such as *Faecalibacterium prausnitzii*, were reproducibly identified across different populations. A systematic review and meta-analysis by C. Gethings-Behncke *et al.* [32] confirmed a consistent association between the presence/increased

representation of *Fusobacterium nucleatum* and risk indicators and/or prognosis in colorectal cancer. Earlier, A. Kostic *et al.* [24] showed that the concentration of *F. nucleatum* in tumour tissue of CRC patients exceeds control values by approximately 10 times, and this microorganism can inhibit the cytotoxic activity of T cells, activating the β -catenin signalling pathway, and accelerating tumour growth.

In patients with diverticular disease and inflammatory bowel diseases, characteristic disturbances in the composition of the gut microbiota are also identified. In a review work by L. Strate & A. Morris [9], it was emphasised that in diverticular disease, a decrease in the number of short-chain fatty acid-producing bacteria, primarily *Faecalibacterium prausnitzii*, and a relative increase in opportunistic anaerobes, including representatives of *Clostridium* and the family *Enterobacteriaceae*, are noted, which is accompanied by a disruption of the barrier function of the mucous membrane and increased inflammation. S. Costello *et al.* [33], in a randomised clinical trial, showed that preventive correction of the microbiota through diet therapy, prebiotics, probiotics, and faecal transplantation is considered as a promising direction for reducing inflammatory activity and restoring the barrier function of the mucous membrane in inflammatory bowel diseases and inflammatory forms of diverticular disease, which is confirmed by data from randomised clinical trials. X. Zhang *et al.* [34], in a systematic review and meta-analysis, showed that probiotics, prebiotics, and synbiotics may contribute to achieving/maintaining remission of IBD and are accompanied by microbiota shifts, including an increase in the proportion of “beneficial” taxa (e.g., *Bifidobacterium*).

Genetic predisposition makes a significant contribution to the risk of CRC. According to a study by E.R. Fearon and B. Vogelstein [20], carcinogenesis occurs through the sequential accumulation of mutations in the APC, KRAS, and TP53 genes, forming the adenoma-carcinoma cascade. According to L. Nguyen *et al.* [21], approximately 5% of colorectal cancer was associated with hereditary syndromes, primarily Lynch syndrome and familial adenomatous polyposis (FAP), which justifies the need for early and personalised screening in this category of patients. Age remains one of the most significant non-modifiable risk factors for colorectal cancer. M. Baretti & D. Le [23] indicated that microsatellite instability (MSI), caused by a deficiency

of the DNA mismatch repair (MMR) system, is a molecular marker of Lynch syndrome and is associated with the development of hereditary forms of colorectal cancer, which are often diagnosed before the age of 50. Family history also plays a significant role. In the recommendations of D. Rex *et al.* [35], it was emphasised that the presence of CRC or advanced adenomas in first-degree relatives significantly increases the individual risk of the disease. In such cases, the authors recommended started screening at age 40 or 10 years before the age of the earliest diagnosis of colorectal cancer in the family, which reflects the principles of a personalised and risk-oriented approach to prevention.

Increasing attention is being paid to the role of social determinants of health in shaping the risk of colorectal cancer. In a systematic review by S. Coughlin [36], covering studies from developed countries, it was shown that education level, income, social isolation, and access to medical care have a significant impact not only on the risk of developing CRC but also on the stage of disease detection and survival rates. The author emphasises that unfavourable socioeconomic conditions are associated with later diagnosis and worse cancer outcomes, which indicates the need to integrate social factors into colorectal cancer prevention and screening strategies. Taken together, the presented data confirm that the risk factors for large intestine diseases form a complex and interconnected system, including behavioural, metabolic, inflammatory, and microbiota components. This underscores the need for an integrated approach to the prevention and early detection of this group of diseases.

Prevention of large intestine diseases: contemporary approaches and prospects

A balanced diet is the cornerstone of primary prevention of large intestine diseases (Table 2). According to the report [16], up to 40% of colorectal cancer (CRC) cases could be prevented through dietary and lifestyle modification. In a review work by M. Hull [37], it was shown that up to 50% of CRC cases are potentially preventable through correction of diet, body weight, and physical activity level. The author emphasises that, alongside screening, nutritional interventions and chemoprevention, including the use of calcium, vitamin D, and omega-3 polyunsaturated fatty acids, play an important role, which opens up prospects for multi-level prevention of large intestine diseases.

Table 2. Main directions for the prevention of diseases of the large intestine

Level of prevention	Measures and recommendations	Key effect / evidence base	Risk reduction / benefit (%)
Primary	Increased intake of fibre (≥ 30 g/day), vegetables, fruits, and wholegrain products	Acceleration of transit, binding of carcinogens, increased production of short-chain fatty acids	Reduction in CRC risk by 17-20%
	Reduction of red and processed meat consumption (< 350 g/week)	Reduced intake of carcinogens	Reduction in CRC risk by approximately 18%

Table 2. Continued

Level of prevention	Measures and recommendations	Key effect / evidence base	Risk reduction / benefit (%)
Primary	Weight control, regular physical activity (≥ 150 min/week)	Reduction of insulin levels and inflammation, improvement of peristalsis	Risk reduction by 20-30%
	Smoking cessation and alcohol restriction	Reduction of inflammation and mutagenic effects	Reduction in IBD relapses by 40% after smoking cessation
	Adequate intake of Ca (1-1.2 g/day) and vitamin D (≥ 800 IU/day)	Reduced proliferation and adenoma risk	Reduction in CRC risk by approximately 10-15%
Secondary	Screening from the age of 45 years: colonoscopy, FIT, CT colonography, stool DNA testing	Early detection of adenomas and tumours	Reduction in mortality by 30-53% with regular screening
	Chemoprevention in high-risk groups (low-dose acetylsalicylic acid)	Inhibition of cyclooxygenase-1 and 2, reduction of inflammation and proliferation	Reduction in CRC risk in selected patient groups by 20-30%
Tertiary	Follow-up after CRC treatment (CEA monitoring, CT, colonoscopy)	Early detection of recurrences, improved survival	Reduction in mortality by 20-30%
	Probiotics and faecal microbiota transplantation (FMT)	Restoration of microbiota, remission in IBD and after CRC	Remission in 24-30% of cases
	AI-assisted endoscopy (CADe)	Increased adenoma detection, reduction in missed polyps	Increase in ADR by 9 percentage points, reduction in missed lesions by 30%

Source: developed by the authors based on [5,8,12,14,16-17,19,33-35,38-44]

High consumption of dietary fibre (≥ 30 g/day) and whole grains is associated with a reduction in colorectal cancer risk of 17-20%, whereas regular consumption of vegetables and fruits – by 10-15%. These findings are based on the expert report “Diet, nutrition, physical activity and colorectal cancer” [16] and are confirmed by data from a systematic review by R. Eysenck *et al.* [17] analysing the influence of dietary and behavioural factors on long-term outcomes in CRC patients. The mechanisms of the protective action of dietary fibre showed that the fermentation of indigestible carbohydrates by the gut microbiota leads to the formation of short-chain fatty acids, primarily butyrate and propionate, which possess anti-inflammatory and antiproliferative effects, and also contribute to a reduction in intestinal transit time and a decrease in the contact of the mucosa with carcinogens.

Regarding the fat component of the diet, regular consumption of fish rich in omega-3 fatty acids is associated with a moderate reduction in CRC risk (approximately 12% when consuming ≥ 2 servings per week) [16]. In contrast, V. Bouvard *et al.* [14] showed that systematic consumption of processed meat in amounts exceeding 50 g/day increases the risk of colorectal cancer by 18%, and red meat (>100 g/day) – by 17%, which served as a basis for recommendations to limit the consumption of meat products and avoid processed products. Overweight and obesity are also considered as significant risk factors for colorectal cancer, diverticular disease, and inflammatory bowel diseases. In a large population study by K. Bhaskaran *et al.* [19], it was shown that each increase in body mass index of 5 kg/m² is accompanied by an increase in CRC risk of 18% in men and 14% in women, which underscores the role of metabolic disorders in colon carcinogenesis.

B. Singh *et al.* [45], in a systematic review and meta-analysis, showed that physical training in CRC patients is generally safe and feasible, and improves several clinically significant indicators (quality of life, fatigue, etc.). In the report “Diet, nutrition, physical activity and colorectal cancer” [16], it was noted that regular physical activity reduces CRC risk by 20-25%, and in individuals with obesity – up to 30% when combined with diet [17]. The most pronounced effect was observed with moderate-intensity aerobic exercise of at least 150 min/week or equivalent strength exercises. The mechanisms included a reduction in insulin levels, improved glucose sensitivity, reduced inflammation, and accelerated intestinal peristalsis. Moderate alcohol consumption (<20 g of ethanol per day) was not associated with an increased risk, but when this level was exceeded, CRC risk increased by 20% [16]. Thus, smoking cessation and control of alcohol consumption had a significant preventive effect. Adequate intake of calcium (1-1.2 g/day) and vitamin D (>800 IU/day) reduces the risk of adenomas and CRC. Vitamin D regulates the expression of apoptosis and proliferation genes, while calcium binds bile acids and fatty acids in the intestinal lumen. In addition, antioxidants (vitamins C, E, beta-carotene, polyphenols) reduce oxidative stress and inflammation, although the evidence base is currently moderate. Secondary prevention plays an important role in reducing the risk of complications of large intestine diseases. In a randomised clinical trial by Ø. Holme *et al.* [38], it was shown that endoscopic screening can significantly reduce colorectal cancer morbidity and mortality, which underscores the importance of early detection of precancerous changes. Regular screening is considered as the most effective approach to early detection of CRC and its precursors.

Colonoscopy is the gold standard, allowing not only diagnosis but also simultaneous removal of polyps. A study by A. Zauber *et al.* [12] showed that colonoscopy with polypectomy reduces CRC mortality by 53%. Non-invasive and less invasive approaches are used as alternative screening methods. In the recommendations of D. Rex *et al.* [35], it was emphasised that the faecal immunochemical test (FIT), performed annually, remains one of the most accessible tools for population screening, demonstrating a sensitivity of about 79% and a specificity of up to 94% for detecting colorectal cancer. T. Imperiale *et al.* [41], in a large prospective study, showed that the multitarget stool DNA test (Cologuard), based on the detection of KRAS gene mutations and hypermethylation of the NDRG4 and BMP3 genes, had a high sensitivity for detecting colorectal cancer (about 92%), but its sensitivity for advanced precancerous lesions was approximately 42%.

The promise of molecular stool tests based on DNA methylation analysis was confirmed by a systematic review and meta-analysis by M. Gachabayov *et al.* [46], which showed that these methods are characterised by high specificity and satisfactory sensitivity and can be used for risk stratification and selection of patients in need of colonoscopy. F. Caldera *et al.* [47], in a clinical update of the American Gastroenterological Association, considered CT colonography as a non-invasive alternative to endoscopic screening methods and indicated that this method has a high sensitivity for detecting large polyps larger than 10 mm, reaching 86-93%, which makes it an acceptable examination option for patients with contraindications to colonoscopy or those who refuse it.

Overall, as emphasised in systematic reviews and recommendations of expert communities, the combined use of various screening methods makes it possible to reach different risk groups and significantly increase the effectiveness of secondary prevention, ensuring a reduction in colorectal cancer mortality by 30-40% and morbidity by 20-25% [34,37-38]. The use of low doses of aspirin in the range of 75-325 mg/day is associated with a reduction in the risk of developing colorectal cancer by approximately 20-30% with long-term regular use. D. Drew *et al.* [39], in a large epidemiological analysis, showed that long-term aspirin use is associated with a significant reduction in the risk of colorectal cancer and other gastrointestinal neoplasias. J. Chia *et al.* [40], analysing current data on colorectal cancer chemoprevention, also noted the protective effect of long-term use of acetylsalicylic acid, especially in individuals with an increased risk of developing the disease. The proposed mechanisms of the chemopreventive action of aspirin include inhibition of cyclooxygenase-1 and cyclooxygenase-2, reduction of chronic inflammation, and suppression of colonic epithelial proliferation [39-40]. The role of non-steroidal anti-inflammatory drugs in the

prevention of colorectal adenomas and cancer was also confirmed by a meta-analysis by B. Cole *et al.* [48], showing a reduction in the frequency of adenomas, but the clinical use of these drugs is limited by the risk of gastrointestinal and cardiovascular side effects. In this regard, chemoprevention is considered primarily for selected high-risk groups.

After surgical or combined treatment of colorectal cancer, patients are advised to undergo regular follow-up for early detection of recurrence and metachronous tumours. Surveillance colonoscopy should be performed 1 year after surgery, then after 3 years, and thereafter every 5 years in the absence of pathological findings. In addition to endoscopic surveillance, the authors recommended dynamic determination of carcinoembryonic antigen (CEA) levels every 3-6 months during the first 2 years after treatment, as well as regular CT examinations of the chest and abdominal cavities, which contributes to earlier detection of recurrences and metastases. Probiotics and faecal microbiota transplantation (FMT) are considered as promising methods for maintaining remission in patients with inflammatory bowel diseases. In a randomised clinical trial, S. Costello *et al.* [33] showed that the use of FMT in patients with ulcerative colitis was associated with the achievement of clinical remission in 24-30% of patients over 8 weeks of follow-up. Similar findings are presented in a systematic review by M. Kasapoglu *et al.* [42], which emphasised the potential role of microbiome interventions in reducing inflammation activity and maintaining remission in IBD. At the same time, the use of FMT in patients after surgical treatment of colorectal cancer currently remains the subject of experimental research and requires further clinical validation.

Contemporary strategies for the prevention of large intestine diseases are gradually shifting from universal approaches to personalised models based on individual risk assessment. In a meta-analysis, A. Sardari & H. Usefi [49] proposed a "machine learning-based meta-analysis" approach to identify genes/biomarkers associated with CRC and IBD, with validation of the results on independent datasets, which underscores the promise of computational methods for risk stratification and personalisation of prevention. According to a systematic review and meta-analysis by M. Sulbaran *et al.* [50], second-generation colon capsule endoscopy demonstrated acceptable diagnostic efficacy for detecting colorectal cancer and large adenomas and was considered as a promising non-invasive screening method, especially in conditions of limited availability of endoscopic examinations. At the same time, the method remains limited by the inability to perform biopsy and therapeutic interventions, as well as by the requirements for high-quality bowel preparation. AI-assisted endoscopy using computer-aided detection (CADE) systems improves the quality of colonic mucosal visualisation,

helps to reduce “blind spots”, and increases the adenoma detection rate (ADR). In a prospective randomised clinical trial, P. Wang *et al.* [43] showed that the use of CAdE systems during colonoscopy leads to a significant increase in ADR from 20.3% to 29.1%, as well as a reduction in the frequency of missed polyps, mainly due to more effective detection of small adenomas.

In a meta-analysis by C. Hassan *et al.* [51], it was shown that the use of AI in colonoscopy is associated with an increase in adenoma and polyp detection rates compared to routine practice. In a meta-analysis of randomised controlled trials by D. Huang *et al.* [52], it was shown that the use of artificial intelligence systems in colonoscopy significantly increased adenoma detection rate (ADR) and polyp detection rate (PDR) compared to standard colonoscopy. The authors also noted that the positive effect of AI-assisted colonoscopy is maintained in subgroups of neoplasms of different sizes and localisations. Thus, the prevention of large intestine diseases in contemporary conditions is a multi-level system, including lifestyle modification, the implementation of screening programmes, and the use of personalised prevention strategies. The combination of behavioural, diagnostic, and technological approaches makes it possible to influence the key links of pathogenesis, ensuring early detection of precancerous conditions and reducing the risk of disease progression. The comprehensive implementation of these measures contributes to reducing the epidemiological burden of colorectal diseases, improving clinical outcomes, and improving the quality of life of the population.

Conclusions

Large intestine diseases, including colorectal cancer, inflammatory bowel diseases, diverticular disease, and polyposis, remain among the most pressing problems in contemporary gastroenterology and public health. Their significance is determined not only by their high prevalence but also by their multifactorial nature, formed under the influence of genetic, metabolic, inflammatory, and behavioural determinants. The conducted review of the scientific literature confirms that the combination of non-modifiable factors with a wide range of lifestyle factors plays a key role in shaping the risk of large intestine diseases. An analysis of contemporary studies has shown that the leading modifiable risk factors are dietary patterns, overweight, low physical activity, smoking, alcohol abuse, and chronic inflammation accompanied by disturbances in the composition of the gut microbiota. The combination of these factors contributes to the formation of an

unfavourable metabolic and immune microenvironment, creating conditions for the progression of inflammatory processes and the development of neoplastic changes in the mucous membrane of the large intestine. At the same time, it has been convincingly shown that lifestyle modification and rational nutrition possess a pronounced preventive potential, which emphasises the priority importance of primary prevention.

Secondary prevention, based on the early detection of precancerous conditions and initial forms of the disease, plays a significant role in reducing the burden of large intestine diseases. Regular endoscopic surveillance and timely removal of precancerous lesions were considered as one of the most effective tools for preventing adverse outcomes, especially in high-risk groups. Thus, contemporary prevention strategies should be comprehensive in nature and combine measures aimed at reducing the impact of risk factors with effective screening and surveillance programmes. The summarised data indicated that the further development of the prevention of large intestine diseases is associated with a transition to personalised approaches that take into account the individual characteristics of the patient. Promising directions for future research are an in-depth study of the role of the gut microbiota in carcinogenesis and inflammatory processes, the development of risk stratification models based on a combination of clinical-epidemiological and biological parameters, as well as the assessment of the effectiveness of digital and intelligent technologies in early diagnosis. The implementation of these directions will make it possible to increase the targeting of preventive measures and to form more effective strategies for the prevention of large intestine diseases at the population and individual levels.

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Conflict of Interest

None.

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