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# CLINICAL AND PATHOGENETIC FEATURES OF INFLAMMATORY BOWEL DISEASES: A LITERATURE REVIEW

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**Abstract**

**Background & Aims:** Inflammatory bowel diseases (IBD), including ulcerative colitis and Crohn's disease, pose significant challenges and are associated with a rising global incidence. However, research in Kazakhstan is limited and primarily descriptive. This review **aims** to elucidate the clinical and pathogenetic features of IBD in Kazakhstan to improve diagnostic and therapeutic approaches. A systematic review was conducted to identify common associations.

**Objectives:** To evaluate the clinical and pathogenetic features of inflammatory bowel diseases in Kazakhstan and their implications for diagnosis and treatment.

**Methods:** We analyzed systematic reviews and studies published in PubMed, Scopus, MEDLINE, EMBASE, and Cochrane databases from 2014 to the present. This review specifically examined approximately 60 articles from the past decade, focusing on IBD in Kazakhstan, given the limited and primarily descriptive nature of the existing research.

**Conclusions:** Our review underscores the increasing incidence of inflammatory bowel disease (IBD) in Kazakhstan, highlighting significant challenges in understanding its clinical and pathogenetic aspects. Despite advances in diagnostics and therapies, the precise etiology of IBD remains unclear. The complexity of diagnosing and distinguishing between ulcerative colitis and Crohn's disease, along with inconsistent markers for mucosal healing, emphasizes the need for more standardized diagnostic criteria. Large-scale studies with rigorous methodologies are crucial to advancing our understanding of IBD and improving treatment strategies. Addressing these research gaps is essential for enhancing healthcare and patient outcomes in the region.

***Keywords:*** *Inflammatory Bowel Diseases (IBD); Gastrointestinal tract; Ulcerative Colitis; Crohn's Disease.*

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**Резюме**

# КЛИНИКО-ПАТОГЕНЕТИЧЕСКИЕ ОСОБЕННОСТИ ВОСПАЛИТЕЛЬНЫХ ЗАБОЛЕВАНИЙ КИШЕЧНИКА: ОБЗОР ЛИТЕРАТУРЫ

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**Введение и цели:** Воспалительные заболевания кишечника (ВЗК), включая язвенный колит и болезнь Крона, представляют собой серьезную клиническую проблему и демонстрируют рост заболеваемости во всем мире. Однако исследования в Казахстане ограничены и носят в основном описательный характер. **Цель** данного обзора - проанализировать клинические и патогенетические особенности ВЗК в Казахстане для совершенствования диагностики и терапии. Был проведен систематический обзор для выявления общих закономерностей.

**Задачи:** Оценить клинические и патогенетические особенности воспалительных заболеваний кишечника в Казахстане и их значение для диагностики и лечения.

**Методы:** Мы проанализировали систематические обзоры и оригинальные исследования, опубликованные в базах данных PubMed, Scopus, MEDLINE, EMBASE и Cochrane с 2014 года по настоящее время. Особое внимание было уделено 60 статьям, опубликованным за последнее десятилетие и посвящённым ВЗК в Казахстане, учитывая ограниченность и описательный характер имеющихся данных.

**Выводы:** Наш обзор подчеркивает рост заболеваемости воспалительными заболеваниями кишечника (ВЗК) в Казахстане и трудности, связанные с изучением их клинических и патогенетических аспектов. Несмотря на достижения в диагностике и лечении, этиология ВЗК остается недостаточно изученной. Сложности в разграничении язвенного колита и болезни Крона, а также отсутствие унифицированных маркеров слизистой ремиссии, указывают на необходимость стандартизации диагностических критериев. Крупномасштабные исследования с применением строгих методологических подходов необходимы для углубления понимания ВЗК и совершенствования лечебных стратегий. Устранение существующих научных пробелов является важным условием повышения качества медицинской помощи и прогноза для пациентов в регионе.

***Ключевые слова:*** *воспалительные заболевания кишечника (ВЗК); желудочно-кишечный тракт; язвенный колит; болезнь Крона.*

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**Түйіндеме**

# ІШЕК ҚАБЫНУ АУРУЛАРЫНЫҢ КЛИНИКО-ПАТОГЕНЕЗДІК ЕРЕКШЕЛІКТЕРІ: ӘДЕБИ ШОЛУ

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**Кіріспе және мақсаттар:** Асқазан-ішек жолының қабыну аурулары (АІЖҚА), оның ішінде ойық жаралы колит пен Крон ауруы, жаһандық деңгейде денсаулық сақтау жүйесіне елеулі қиындықтар туғызып отыр және олардың таралуы өсуде. Дегенмен, Қазақстанда бұл саладағы зерттеулер шектеулі және негізінен сипаттамалық сипатта. Бұл шолу Қазақстандағы АІЖҚА-ның клиникалық және патогенездік ерекшеліктерін нақтылап, диагностикалық және емдеу тәсілдерін жетілдіруді көздейді. Ортақ заңдылықтарды анықтау үшін жүйелі шолу жүргізілді.

**Мақсаттар:** Қазақстандағы қабыну ішек ауруларының клиникалық және патогенездік ерекшеліктерін бағалап, олардың диагностикасы мен еміне әсерін анықтау.

**Әдістер:** 2014 жылдан бастап бүгінгі күнге дейін PubMed, Scopus, MEDLINE, EMBASE және Cochrane мәліметтер базаларында жарияланған жүйелі шолулар мен ғылыми зерттеулер талданды. Соңғы он жылдықта жарияланған және Қазақстандағы ішек қабыну ауруларына арналған 60 мақалаға ерекше назар аударылды, себебі қолжетімді деректер шектеулі әрі сипаттамалық сипатта.

**Қорытындылар:** Бұл шолу Қазақстанда қабыну ішек ауруларының таралуының артып келе жатқанын және олардың клиникалық және патогенездік жақтарын түсінудегі елеулі қиындықтарды айқындайды. Диагностика мен емдеудегі жетістіктерге қарамастан, АІЖҚА-ның нақты этиологиясы әлі де белгісіз. Ойық жаралы колит пен Крон ауруының ажыратылуындағы қиындықтар, сондай-ақ шырышты қабаттың жазылу маркерлерінің бірізді болмауы, диагностикалық критерийлерді стандарттаудың маңыздылығын көрсетеді. Қатаң әдіснамалық талаптарға сай ауқымды зерттеулер АІЖҚА туралы түсінікті тереңдетуге және емдеу стратегияларын жетілдіруге қажет. Бұл ғылыми олқылықтарды жою – аймақтағы денсаулық сақтау сапасы мен науқастардың өмір сүру сапасын арттыру үшін өте маңызды.

***Түйінді сөздер:*** *Ішектің қабыну ауруы (АІЖҚА); асқазан-ішек жолы; ойық жаралы колит; Крон ауруы.*

***Дәйексөз үшін:*** *Жаппаркулова А.Б., Абилов Т.С., Нурдинов Н.С., Аймаханов М.С., Нускабаева Г.О., Садыкова К.Ж., Тулешова Э.Ж., Тастемирова А.Б.* Ішек қабыну ауруларының клинико-патогенездік ерекшеліктері: әдеби шолу // ғылым және Денсаулық. 2025. Т.27 (2), Б. 188-197. doi 10.34689/SH.2024.27.2.021

**Introduction**

Inflammatory bowel diseases (IBD), such as ulcerative colitis and Crohn's disease, are significant concerns in gastroenterology due to their complex and multifactorial nature [1], [2], [3]. These chronic conditions, with unclear causes and intricate pathogenesis, frequently lead to relapses and severe complications, often resulting in long-term disability. Despite the rising global prevalence, research on IBD in Kazakhstan remains limited and predominantly descriptive.

Although IBD was once primarily associated with Western countries, it has now emerged as a global concern, affecting regions across Asia, Africa, and South America. The incidence of IBD varies significantly by region, with Europe reporting the highest rates of ulcerative colitis at 24.3 per 100,000 in 2017, followed by North America at 19.2, and Asia at 6.3 [3]. For Crohn's disease, North America has the highest incidence at 20.2 per 100,000, followed by Europe at 12.2, and Asia and the Middle East at 5. This upward trend, particularly in Europe and North America, presents considerable medical and social challenges [5], [6], [7].

In Kazakhstan, the Ministry of Health has reported a prevalence of Crohn's disease (CD) and ulcerative colitis (UC) at 6.3 and 31.5 per 100,000 individuals, respectively. However, the first comprehensive report on IBD prevalence in Central Asia reveals a higher age- and sex-adjusted prevalence of 113.9 per 100,000, including 84.4 for ulcerative colitis and 29.5 for Crohn's disease. These findings underscore the need for more targeted health planning and resource allocation for IBD in the region [8]. A description of the demographic characteristics and regional distribution of Kazakhstan's population is presented in Table 1.

**Materials and methods**

When preparing the literature review, information from internationally recognized and widely used biomedical databases was utilized. This article was prepared within the framework of the project of the Scientific Committee of the Ministry of Science and Higher Education of the Republic of Kazakhstan (Grant No. BR24992814).

**Search strategy:**

A comprehensive literature review was conducted using international English- and Russian-language databases, including PubMed, Scopus, Google Scholar, and eLibrary. The search covered publications from the last 10 years, from 2014 to 2024. The literature search was performed using scientific terms relevant to the research topic, such as “Inflammatory Bowel Diseases (IBD)”, “Gastrointestinal tract”, “Ulcerative Colitis”, and “Crohn's Disease”.

*Geographic scope:* The review includes research conducted in Kazakhstan, and other countries, reflecting a broad international perspective on the topic.

*Inclusion criteria:* It would have been possible to select articles from the only last 5 years, but given the limited research conducted in Kazakhstan and the corresponding small number of scientific articles, we decided to consider the full 10-year range for the study.

*Exclusion criteria:* Articles without full text, duplicate publications, or studies lacking relevant abstracts. .

*Table 1.*

**Characteristics of IBD types by age, gender, location and regional prevalence in Kazakhstan population (modified from Kaibullaeva's article [8].**

| Characteristics of IBD by Age, Sex, and Area | IBD | | UC | | CD | |
|---|---|---|---|---|---|---|
| | Per 100,000 | 95% CI | Per 100,000 | 95% CI | Per 100,000 | 95% CI |
| ***Age group (yr)*** | | | | | | |
| 18–29 | 129.5 | 83.3–175.7 | 89.0 | 50.2–127.9 | 40.5 | 13.0–67.9 |
| 30–39 | 155.5 | 100.9–210.1 | 117.8 | 69.8–165.7 | 37.7 | 8.5–66.9 |
| 40–49 | 117.0 | 69.4–164.7 | 74.9 | 36.0–113.8 | 42.1 | 11.7–72.5 |
| 50–59 | 75.1 | 41.5–108.7 | 53.6 | 24.7–82.5 | 21.4 | 1.6–41.3 |
| ≥60 | 83.8 | 41.7–125.9 | 69.0 | 30.4–107.6 | 14.8 | 0–36.4 |
| Total | 110.8 | 91.5–130.1 | 79.6 | 63.2–96.1 | 31.2 | 20.7–41.6 |
| ***Sex*** | | | | | | |
| Male | 129.5 | 94.3–164.6 | 107.9 | 75.7–140.1 | 21.6 | 6.0–37.2 |
| Female | 100.2 | 77.1–123.3 | 63.6 | 45.1–82.2 | 36.6 | 22.3–50.9 |
| ***Area*** | | | | | | |
| Urban | 114.1 | 92.2–136.0 | 81.8 | 63.2–100.4 | 32.3 | 20.4–44.2 |
| Rural | 97.1 | 54.8–139.5 | 70.6 | 33.9–107.3 | 26.5 | 2.0–51.0 |
| **Types of IBD and its prevelance by Regions Region** | **IBD** | | **UC** | | **CD** | |
| | **Per 100,000** | **95% CI** | **Per 100,000** | **95% CI** | **Per 100,000** | **95% CI** |
| ***Region (Crude rates)*** | | | | | | |
| Almaty | 120.2 | 84.3–156.1 | 72.1 | 43.9–100.3 | 48.1 | 24.7–71.5 |
| East | 114.5 | 71.5–157.5 | 94.7 | 55.3–134.2 | 19.7 | 0–40.2 |
| West | 108.0 | 42.0–174.0 | 72.0 | 16.3–127.8 | 36.0 | 0–79.2 |
| North | 106.5 | 67.8–145.2 | 79.0 | 45.4–112.7 | 27.5 | 6.2–48.8 |
| South | 87.5 | 31.3–143.7 | 79.6 | 25.6–133.6 | 8.0 | 0–35.0 |
| Total | 110.8 | 91.5–130.1 | 79.6 | 63.2–96.1 | 31.2 | 20.7–41.6 |

**Search results**

The initial search yielded 122 548 articles from PubMed, 1 932 from Scopus, and 193 000 from Google Scholar. After applying additional filtering by key terms, relevance, and systematic sorting, the selection was narrowed to 11 998 articles from PubMed, 1,450 from Scopus, and 26 700 from Google Scholar. Further refinement, including removal of duplicates and unavailable full texts, resulted in a final selection of 80 articles from PubMed, 27 from Scopus, and 85 from Google Scholar. These articles were systematically reviewed, and as a result, 60 articles that met specific criteria from the PubMed database were selected for in-depth analysis (Figure 1).

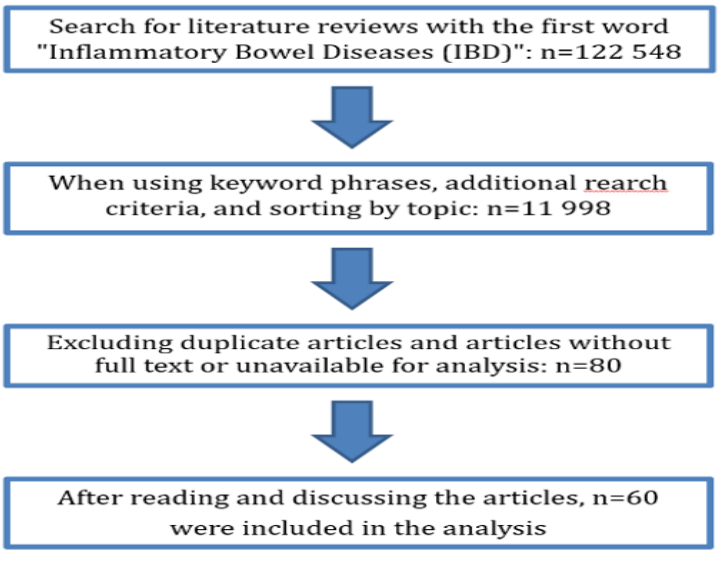


**Figure 1. Table of selection of articles included in the analysis.**

**Discussion**

Understanding IBD involves complex interactions of genetic, immunological, and environmental factors. More than 100 genes have been linked to Crohn's disease, including the NOD2/CARD15 gene, which affects inflammation. NOD2 is the most significant gene predisposing to Crohn's disease and is associated with stricture formation, ileal involvement, and increased risk of surgery [9]. Additional genetic modifications associated with autophagy, such as ATG16L1 (autophagy-related 16-like 1), LRRK2 (leucine-rich repeat kinase 2), and IRGM (immune-related GTPase M), may also increase susceptibility to IBD [10]. Environmental factors, including diet and nicotine use, further influence the risk of developing IBD [11], [12], [13].

This review aims to synthesize current knowledge on IBD mechanisms and epidemiology, with a particular focus on Kazakhstan, and to identify research gaps to guide future studies and clinical practices.

**Comprehensive Analysis of Toll-like Receptors in Ulcerative Colitis and Crohn's Disease**

Toll-like receptors (TLRs), essential components of innate immunity, recognize pathogen-associated molecular patterns. Humans have 11 TLRs, each detecting specific pathogens and influencing immune responses. Dysfunctions in TLRs can lead to diseases, with research focusing on their role in IBD [14].

*Abubakirova's* study revealed an increase in TLR2 and TLR4 expression in the terminal sections of the colon in patients with ulcerative colitis. This suggests that impaired expression in various segments of the intestinal mucosa may contribute to the development of IBD [15].

Studies show that increased TLR2 and TLR4 expression in ulcerative colitis may contribute to the disease. Variations in TLR expression and genetic activity further suggest a role in IBD pathogenesis. Elevated TLR8 expression in biopsies supports its involvement. Additionally, antimicrobial peptides activated by TLRs protect the mucosa and support immune function, with lactoferrin showing potential for modulating inflammation and reducing pro-inflammatory cytokines [16].

Research on the genetic aspects of inflammatory bowel disease (IBD) in Kazakhstan and Central Asia is currently limited. A 2017 study conducted in Kazakhstan focused on the prevalence and patient awareness of IBD, reporting an age- and sex-adjusted prevalence of 113.9 per 100,000 population (Table 1) [8]. However, this study did not delve into genetic factors such as TLR or NOD2 polymorphisms. While specific studies on TLR2, TLR3, TLR4, TLR5, and NOD2 polymorphisms in IBD patients within Kazakhstan or Central Asia are lacking, broader research has identified associations between these genetic factors and IBD in other populations. For instance, TLR4 Asp299Gly polymorphism has been linked to an increased risk of Crohn's disease and ulcerative colitis in Caucasian populations, though such associations were not observed in Asian cohorts [17].

Given the absence of region-specific genetic studies, there is a significant opportunity for research in Central Asia to explore the role of these polymorphisms in IBD susceptibility and progression.

**Role of Lactoferrin in Inflammatory Bowel Diseases**

Lactoferrin plays a key role in directing leukocytes to inflamed gut areas, activating immune cells, and exerting anti-inflammatory properties by binding free iron and neutralizing toxic radicals. It is a cationic glycoprotein of the innate immune system that provides numerous benefits [18]. Elevated lactoferrin levels in blood and other fluids correlate with the presence and severity of IBD, serving as a marker for treatment efficacy. Studies show that fecal lactoferrin levels are higher in IBD patients compared to those with non-inflammatory gut conditions, suggesting it as a primary diagnostic tool and a potential alternative to invasive methods like video capsule endoscopy [19], [20].

Research by Djansugurova L. demonstrated that fecal lactoferrin levels were significantly higher in IBD patients [21]. The sensitivity of fecal lactoferrin is 81% for ulcerative colitis and 82% for Crohn's disease, aiding in monitoring treatment and mucosal healing [22]. High post-surgical levels may indicate relapse, though normal levels do not guarantee complete recovery. Researchers emphasized the importance of lactoferrin in ileal diseases and its correlation with endoscopic activity in ulcerative colitis and histological indices in Crohn's disease [20].

The gut mucosa, a natural barrier, relies on both innate (lactoferrin, mucus, lysozyme, cytokines) and acquired (secretory immunoglobulin A, SIgA) immunity. SIgA, produced by B cells in the mucosa, binds pathogens and indicates local immunity and therapy effectiveness. Elevated SIgA levels in serum signal active IBD and synthesis in damaged cells.

**Low Immunoglobulin Levels and Their Implications**

Low immunoglobulin levels can indicate food allergies, celiac disease, or autoimmune predispositions [23], [24]. Research on immunoglobulin A (IgA) in IBD is limited. Recent studies found lower levels in ulcerative colitis biopsies [25]. Another study observed that immunoglobulin levels in IBD patients resemble those in connective tissue diseases [26]. However, researchers reported significantly higher secretory immunoglobulin levels in IBD patients compared to celiac disease patients [27].

IBD involves genetic and environmental factors as well as interactions between innate and adaptive immunity [28], highlighting the need to consider microbiota changes in this context.

**Modern Therapy for Inflammatory Bowel Diseases**

The treatment of inflammatory bowel diseases (IBD) lacks a universal approach due to their unclear pathogenesis. Traditional methods aim to alleviate symptoms, prevent flare-ups, and achieve remission and mucosal healing. Standard treatments include aminosalicylates, glucocorticoids, immunosuppressors, antibiotics, and probiotics [29].

Aminosalicylates are ineffective in 30% of cases, prompting alternatives like immunosuppressors and biological modifiers, which are effective in 40-60% and 40% of cases, respectively. However, biological modifiers are costly and often poorly tolerated. Antibiotics such as rifaximin and ciprofloxacin are used for symptom management and treating perianal fistulas [30]. Probiotics may stabilize gut flora and support immune responses, potentially reducing relapse rates and side effects, though evidence from human trials remains limited. Combining probiotics with traditional therapies may enhance treatment outcomes.

**Diagnosis of Inflammatory Bowel Diseases**

The diagnosis of inflammatory bowel diseases (IBD), encompassing ulcerative colitis and Crohn's disease, has advanced with the integration of various diagnostic modalities. Traditional methods include endoscopic, histological, radiological, and biochemical assessments, each with inherent limitations.

**Endoscopic Techniques:** Colonoscopy remains the gold standard for visualizing the colonic mucosa and obtaining biopsy samples. However, it is invasive and limited in scope. Video capsule endoscopy provides a non-invasive alternative and is especially useful for examining hard-to-reach areas, but it is expensive and cannot collect tissue samples.

**Radiological Imaging:** Cross-sectional imaging techniques such as computed tomography (CT), magnetic resonance imaging (MRI), and ultrasonography are pivotal in evaluating IBD. These modalities offer detailed insights into bowel wall characteristics and extraintestinal manifestations [31].

**Non-Invasive Biomarkers:** Non-invasive methods, such as measuring neutrophil elastase, interleukins, and other markers, are also used but are hindered by the rapid degradation of these substances by fecal enzymes [32]. Fecal calprotectin has emerged as a valuable non-invasive marker for intestinal inflammation, aiding in distinguishing IBD from functional gastrointestinal disorders [33]. Its utility in monitoring disease activity and predicting relapses is well-recognized. IBD presents with variable severity, from mild to debilitating symptoms, and delays in diagnosis, particularly in Crohn's disease, often lead to adverse outcomes. Early detection and timely treatment initiation are crucial for improving patient outcomes. Identifying red flags and referring patients early for specialist evaluation can aid in prompt diagnosis. While treatment options continue to expand, many patients do not respond to first-line therapy, making appropriate selection critical [34], [35]. Disease severity can be stratified using clinical, demographic, and serologic markers to optimize treatment choices. Personalized medicine approaches, incorporating clinical decision support tools, genomics, and predictive biomarkers, are shaping future treatment strategies. Once therapy begins, confirming remission through objective markers and adjusting treatment based on surrogate biomarkers ensures long-term inflammation control [27], [36].

Fecal tests for granulocyte markers assess inflammation but are limited by high costs and potential radiation exposure [33]. Advancements in non-invasive diagnostic methods continue to evolve, aiming to enhance early detection and reduce patient discomfort associated with traditional procedures.

**Fecal Calprotectin as a Diagnostic Tool**

Fecal calprotectin (FCP) is a calcium-binding protein (S100A8/A9) derived from neutrophils [37]. [38], [39]. Its levels can increase 5–40 times during inflammation and correlate with disease activity [40]. FCP levels are significantly higher in patients with inflammatory bowel disease (IBD) than in those with other [41], [42]. However, daily fluctuations and the presence of elevated levels in some healthy individuals limit its use as a sole diagnostic method. Nevertheless, incorporating FCP testing into clinical practice for evaluating lower gastrointestinal symptoms can help reduce unnecessary colonoscopies, thereby lowering costs for both patients and the healthcare system [40]. Nikitin et al. introduced the 'PhiCal ELISA' method, which initially required 5 grams of feces for analysis and set a positive threshold at 10 mg/L [43]. The test distinguishes IBD from irritable bowel syndrome (IBS) with high sensitivity, reaching 100% at 150 μg/g in some studies. N.Zaletova's study of 89 children demonstrated that FCP levels correlate with the degree of endoscopic and histological activity in ulcerative colitis and with both the prevalence and endoscopic activity in Crohn's disease [44]. Additionally, FCP levels in newborns are higher than in older children and adults, though the effect of diet on calprotectin levels in infants is still under investigation. Zinkevich O.D.'s study revealed that FCP levels were elevated in 49% of IBD patients' relatives compared to 13% of spouses, indicating a strong genetic predisposition. However, only 5-10% of relatives showed clear clinical signs, highlighting its utility in genetic risk assessment [45]. Orlinskaya N.Y. conducted a study with 220 patients and found that FCP had 82% sensitivity and 83% specificity for differentiating IBD from IBS, with specificity increasing to 97% for Crohn's disease. In a larger study involving 602 patients, average FCP levels were 50 mg/kg in IBD and 4 mg/kg in IBS, demonstrating high sensitivity (89%) and specificity (79%) compared to other markers like C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) [46]. Kessaeva I. noted that while FCP levels are elevated in microscopic colitis, they are significantly lower than in IBD. In colitis cases, FCP levels were 12 times higher, with 83% sensitivity and 90% specificity [47]. [47]. Maleeva N. highlighted elevated FCP levels in children with celiac disease and allergic colitis, which normalized after dietary intervention. FCP sensitivity and specificity were 64% and 80%, respectively [48].

Despite its diagnostic utility, the practical application of FCP analysis in Kazakhstan remains limited. Additional research is necessary to understand how inflammation location and disease duration influence FCP levels.

**The Role of Genes and Immune Response in Inflammatory Bowel Diseases**

Inflammatory bowel diseases (IBD) result from autoimmune inflammation that affects local gastrointestinal immunity. Relatives of IBD patients have a 20% risk of developing ulcerative colitis or Crohn's disease, with a tenfold higher risk among first-degree relatives. Specific genetic factors, including tumor necrosis factor genes (e.g., CD14 and CTLA4) and loci such as NOD2/CARD15 on chromosome 16, play a significant role in IBD development. Concordance rates for inflammatory bowel disease (IBD) in monozygotic twins vary by type. Ulcerative colitis exhibits rates of 17.6%, while Crohn's disease shows higher rates of 65%, underscoring the combined influence of genetic and environmental factors [49].

**Immune Dysregulation in Ulcerative Colitis**

Ulcerative colitis is primarily driven by an inadequate immune response to gut microbiota antigens. Normally, immune tolerance to commensal microbiota develops before the immune system fully matures. In IBD, this tolerance is disrupted, resulting in inflammation. Antibodies targeting gut mucosal components and resident flora indicate a loss of this tolerance [50].

The immune response in the colon is mediated by lymphoid follicles containing antigen-presenting cells, essential for immune activation. Disruption of immune tolerance shifts the cytokine balance, which is a hallmark of IBD. Key cytokines include:

1. Pro-inflammatory cytokines: Produced by Th1 cells, these promote inflammation.
2. Anti-inflammatory cytokines: Produced by Th2 cells, these inhibit Th1 responses and mitigate inflammation [51].

Cytokine imbalance leads to sustained inflammation, with differing profiles observed in ulcerative colitis and Crohn's disease (Fig. 2)

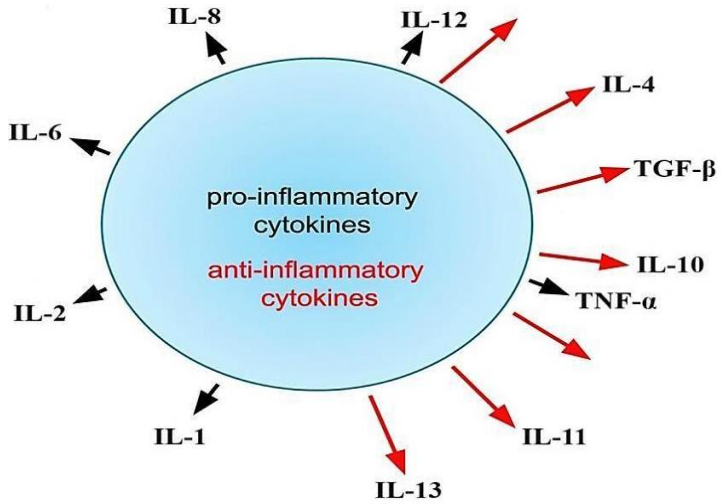


**Figure 2. Cytokines that trigger either pro-inflammatory or anti-inflammatory responses (figure created by the author using BioRender.com)** [51].

**TNF-Alpha's Role in IBD Pathogenesis**

TNF-alpha is a key molecule in IBD, significantly influencing macrophage function and granuloma formation. The imbalance in the immune response results in persistent T-cell activation and chronic inflammation [52], [53].

The study by Zhetpisbayeva K. revealed that the pathogenesis of ulcerative colitis involves not only CD4+ T-lymphocytes but also cytotoxic lymphoid cells, such as CD8+ T-lymphocytes and TNK cells with a pro-inflammatory focus. Additionally, non-specific factors, including oxidase systems in phagocytic cells, contribute to the release of reactive oxygen species (ROS) and macrophage-derived pro-inflammatory cytokines, exacerbating tissue damage and chronic inflammation [54].

The immune response to endogenous gastrointestinal bacteria is a pivotal factor in IBD. Disruption of the intestinal mucosal barrier, coupled with increased IgG production and altered microbiota, triggers inflammation. In IBD, macrophages, lymphocytes, and plasma cells infiltrate the lamina propria of the colon. Elevated IgG levels and metalloproteinase activity further compromise the integrity of the intestinal wall.

Dendritic cells also proliferate in the inflamed gut, recognizing microbial pathogens and producing pro-inflammatory cytokines (Figure 2) [55].

**Symptoms of Inflammatory Bowel Diseases**

The inflammation seen in IBD exhibits unique characteristics compared to other organ systems:

1. Endothelial Dysfunction: This results in microvascular complications and excessive leukocyte adhesion.
2. Thrombotic Risks: Both arterial and venous thromboses are more prevalent due to ischemia, vascular remodeling, and slower mucosal healing, especially in ulcerative colitis and Crohn's disease.

Inflammatory bowel disease (IBD) impacts both the central and peripheral nervous systems, leading to autonomic dysfunction. Studies have shown that autonomic nervous system function can predict the inflammatory response over time in ulcerative colitis patients [56]. Additionally, functional gastrointestinal disorders (FGIDs) significantly increase the risk of developing IBD. Research indicates that individuals with FGIDs have a higher likelihood of developing IBD compared to healthy individuals [57].

Psychological stress affects around 40% of ulcerative colitis patients, correlating with increased IBD incidence due to impaired immune responses and unchecked growth of pathogenic flora [58], [59], [60].

**Conclusions**

Recent advancements in IBD management have led to a better understanding of its pathogenesis, non-invasive diagnostic tools, and novel treatments. However, IBD remains a complex disease influenced by immune dysfunction, with unclear etiology complicating diagnosis and management. Differentiating ulcerative colitis from Crohn’s disease continues to be a challenge.

Mucosal healing is a critical indicator of remission but lacks universal standards. Achieving remission requires both mucosal healing and enhanced patient well-being. Comprehensive clinical data are essential for accurate diagnosis and treatment planning.

Further research is vital to clarify the pathogenetic features of IBD and develop innovative therapeutic approaches. Large-scale projects and systematic research efforts are necessary to address the gaps in knowledge,

particularly in Kazakhstan, where improving prevention and treatment strategies is crucial.

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