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A Review On: Role of Natural Enzymes in Modulating Intestinal Inflammation: Implications for Inflammatory Bowel Disease Therapy

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ABSTRACT

Inflammatory bowel disease (IBD), encompassing Crohn's disease and ulcerative colitis, is a chronic and relapsing inflammatory disorder of the gastrointestinal tract characterized by dysregulated immune responses, impaired intestinal barrier function, and alterations in gut microbiota. Despite advances in conventional therapies, many patients experience limited efficacy, adverse effects, or loss of response over time, highlighting the need for safer and more effective therapeutic alternatives. Natural enzymes derived from plant, microbial, and animal sources have gained increasing attention for their potential role in modulating intestinal inflammation. These enzymes exhibit anti-inflammatory, antioxidant, immunomodulatory, and mucosal protective properties by influencing pro-inflammatory cytokine signalling, oxidative stress pathways, epithelial integrity, and microbiota composition. This review summarizes current evidence on the mechanisms by which natural enzymes regulate intestinal inflammation and evaluates their therapeutic potential in the management of IBD. Furthermore, it discusses the advantages, limitations, and challenges associated with enzyme-based therapies, including issues related to stability, bioavailability, and clinical translation. Overall, natural enzymes emerge as promising adjunctive or alternative strategies for IBD therapy, warranting further preclinical and clinical investigations.

Keywords: Natural enzymes; Intestinal inflammation; Inflammatory bowel disease; Immunomodulation; Gut microbiota; Enzyme-based therapy

1. INTRODUCTION:-

The inflammatory bowel diseases (IBDs) primarily include Crohn's disease and ulcer-ative colitis. Crohn's disease is an IBD that causes inflammation anywhere along the lining of the digestive tract, while ulcerative colitis causes long-lasting inflammation in some part of the digestive tract (mainly the colon). The exact aetiology of IBD is not well known. There are several factors that have been postulated to have an effect on the development of this group of diseases, which include but are not limited to bacterial contamination, a change in the immune system, and genetic variations. For instance, a mutation in the NOD2 gene is associated with an increase susceptibility to IBD via production of proinflammatory cytokines.1 While genetic predisposition plays a key role in immune-mediated diseases, the major influence appears to be due to environmental factors. Indeed, current research suggests that autoimmune diseases are most prevalent in highly industrialized nations but rare in less developed countries.3 Moreover, studies have shown that increased consumption of milk protein, animal protein, and polyunsaturated fatty acids can increase the risk for IBD,4 and that consumption of tobacco increases the risk of Crohn's disease.5The major subtypes of IBD, including Crohn's disease and ulcerative colitis, have a high prevalence rate in the world, with North America noting the highest frequency of people suffering with Crohn's disease. In addition, statistics show that an estimated 129,000 people live with the disease in Canada. Although the onset of the disease usually occurs during adulthood, children are increasingly being diagnosed with IBD. Treating IBD often involves use of medications that can diminish the symptoms and decrease the inflammation in the colon lining. A group of anti-inflammatory drugs including 5-aminosalicylic acid is commonly used to treat IBD.6 Other drugs such as infliximab are also indicated in patients who have failed conventional therapy and are hospitalized with severe IBD. Infliximab is a chimeric monoclonal antibody against tumor necrosis factor alpha (TNF-α), a cytokine involved in intestinal inflammation. Several other immunomodulatory drugs, such as thalidomide, can also be used to treat a patient with severe IBD. Formerly used as a sedative and hypnotic, this synthetic drug has been shown to significantly reduce the inflammation associated with IBD.7 However, under certain circumstances, when medical therapy fails, surgery may be considered. This operation is known as colectomy and involves removal of the large intestine. While ulcerative colitis is cured upon removal of the colon, Crohn's disease unfortunately can still recur after surgery. While medication is commonly used to treat IBD, most pharmaceutical compounds have side effects such as head-ache, diarrhea, and nausea, which can reduce patient compliance and result in worsening of the condition. Therefore,

appropriate delivery systems must be developed in order to overcome the limitations and issues associated with the currently available treatments for IBD. Artificial cell micro-encapsulation is a promising tool in scientific research that allows for targeted delivery of pharmaceutical compounds in a time-dependent fashion.7 Current research aims at developing such a platform in order to deliver anti-inflammatory drugs to the areas of the gastrointestinal tract most affected by IBD.

Types:-

I. Crohn's Disease:-

- **Affected Location** - Can affect any part of the GI tract (from the mouth to the anus) Most often it affects the portion of the small intestine before the large intestine/colon.

- **Damaged Areas** -Damaged areas appear in patches that are next to areas of healthy tissue.

- **Inflammation-** Inflammation may reach through the multiple layers of the walls of the GI tract.

II. Ulcerative Colitis:-

- **Affected Location** - Occurs in the large intestine (colon) and the rectum.

- **Damaged Areas** -Damaged areas are continuous (not patchy) – usually starting at the rectum and spreading further into the colon.

- **Inflammation**- Inflammation may reach through the multiple layers of the walls of the GI tract.

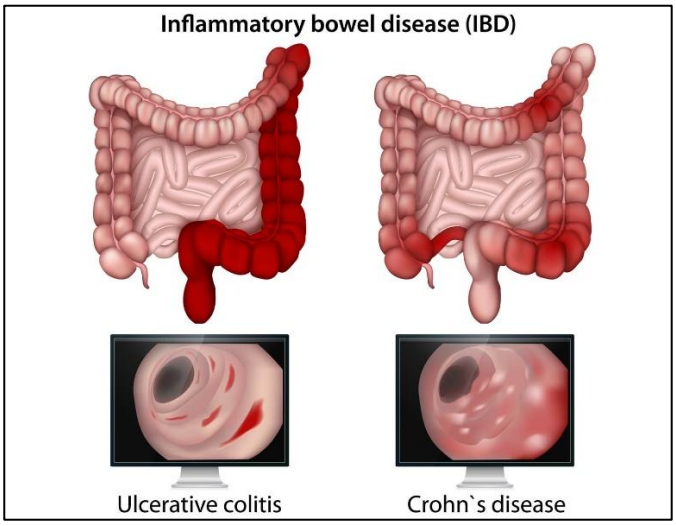

Fig 1: Ulcerative colitis and crohn's disease.

1. SYMPTOMS:-

IBD causes a range of problems in the colon and rectum, but can also affect other parts of the body. The symptoms may come and go. People with IBD may experience flare-ups followed by periods with no symptoms. The first signs of IBD can appear after exposure to something that irritates the intestines, such as a medication (including aspirin, ibuprofen and antibiotics) or a GI infection. The irritation or infection goes away, but the immune system keeps responding.

Common IBD symptoms include:

Abdominal pain (pain in the stomach area), Diarrhea, Rectal bleeding, Weight loss Fever, Anemia.

Malnutrition and delayed growth in people who develop IBD as children -

Anxiety and depression- The condition can also cause swelling or masses, due to inflammation in the intestines. Your doctor may notice these on X-rays and other tests as you are being evaluated for your symptoms.

If inflammation is not controlled, over time IBD can damage the intestines, causing:

Abscesses: pockets of infection that can result in tearing of the intestinal wall.

Strictures: areas of narrowing in the bowel.

Fistulas: abnormal passageways between two organs or vessels that normally do not connect. Fistulas happen when inflammation and pressure inside the bowel break down tissue, and can cause bowel contents to leak into the bladder, urethra or vagina.

Eyes: redness and inflammation due to episcleritis (inflammation between the inner eyelids and the white of the eye) or uveitis (inflammation inside the eye). Experts estimate that 10% to 43% of people with IBD develop eye problems, and regular visits to the eye doctor are important.

Mouth: inflammation (stomatitis), mouth sores and ulcers

Liver: fat in the liver (steatosis)

Biliary tract: gallstones and inflammation of the bile duct system (sclerosing cholangitis)

Kidneys: kidney stones, hydronephrosis (swollen kidneys caused by a backup of urine), fistulas and urinary tract infections.

Skin: erythema nodosum (tender, red bumps on the shins), pyoderma gangrenosum, a rare condition that causes severe skin ulcers on the legs.

Joints and spine: spondylolysis (stress fracture of the vertebrae), sacroiliitis (inflammation of the joints connecting the lower spine with the pelvis) and arthritis in the limbs Blood circulation, including phlebitis (inflammation of blood vessels).

2. CAUSES:-

Researchers are still trying to determine why some people develop IBD. Three factors appear to play a role:

Genetics: As many as 1 in 4 people with IBD have a family history of the disease.

Immune system response: The immune system typically fights off infections. In people with IBD, the immune system mistakes foods as foreign substances. It releases antibodies (proteins) to fight off this threat, causing IBD symptoms.

Environmental triggers: People with a family history of IBD may develop the disease after exposure to an environmental trigger. These triggers include smoking, stress, medication use and depression.

3. EPIDEMIOLOGY OF IBD:-

Disease Burden of UC and CD in India- There are two population-based studies which have looked into the burden of UC in India. The first such study was conducted by in 1984 in Haryana in North India. The study included 21,971 participants and noted a prevalence of 42.8 UC patients per 100,000 people. The second study, conducted 15 years later by Sood et al. from Punjab (this state neighbours Haryana), employed a cluster sampling method and calculated age-standardized prevalence rates after screening a population of 51,910 people of which two thirds lived in rural parts of Punjab and the rest in urban parts. Overall, 23 patients were diagnosed with UC leading to a prevalence rate of 44.3/100,000. The incidence was calculated again during a second visit to the same area 1 year later and was reported to be 6.02/100,000. These data again indicate that UC is not rare in India. However, these findings go against the general belief that the burden of IBD is on the rise in Asian countries, especially when data from other regions suggest so. A recent epidemiological study, entitled "The Asia-Pacific Crohn's and Colitis Epidemiologic Study. Who studied incident IBD cases diagnosed between April 2011 and March 2012, reported the incidence and prevalence of IBD from 8 Asian regions and Australia. The crude overall incidence of IBD, UC, and CD from Asia was 1.37, 0.76, and 0.54 per 100,000, respectively, whereas in Australia it was 23.67, 7.33, and 14.00, respectively. Among the Asian countries, it was highest in mainland China (Guangzhou: 3.4/ 100,000) followed by Hong Kong (3.06/100,000), and Macau (2.2/100,000). Data from Western Asia including

studies from Kuwait, Turkey an incidence rate of 2.8/100,000 and 5.04/100,000 in Kuwait and Israel, respectively, and a prevalence of 4.9/100,000 and 167/100,000 in Turkey and Israel, respectively.

Estimated Disease Burden of IBD in India

We have recently compared the overall disease burden between the West and the East, and came to the staggering observation that the overall disease burden (when both prevalence and population were taken into account) was among the highest in India. The present review employed figures from one geographical area as representative of the entire country, and CD disease burden (where not available) was taken as one third of UC prevalence. The overall estimated IBD population in India in 2010 came out to be 1.4 million, which was the second highest number after the USA (with 1.64 million). Therefore, although the disease prevalence in India is lower than in the West, with a population of more than 120 million, the total IBD population in India is among the largest across the globe.

4. ROLE OF NATURAL ENGYMES FOR IBD:-

Papaya (Carica papaya) biological source – Papain is the dried and purified latex of the green fruits and leaves of ***Carica papaya L***. belonging to family ***Caricaceae*** is a rich source of proteolytic enzymes, collectively known as papaya proteases. These enzymes are predominantly obtained from papaya latex, unripe fruit, leaves, and seeds. The most important proteases include: Papain Chymopapain Caricain Glycyl endopeptidase Among these, papain is the most extensively studied and biologically active enzyme with therapeutic significance and Classification and Biochemical Properties Papaya proteases belong to the cysteine protease Active site contains cysteine–histidine–asparagine triad Broad substrate specificity Optimal activity at pH 5.0–7.0 Molecular weight: ~23 kDa (papain) Heat stable and active over a wide pH range these properties make papaya proteases suitable for oral enzyme therapy**.**

Bromelain (Ananas comosus) is a mixture of proteolytic enzymes (proteases) derived from the pineapple plant biological source - bromelain belongs to a group of protein obtained commercially from the fruit or stem of ***herbaceous perennial***. belonging to the family *Bromeliaceae.* It has long been used in traditional medicine for its anti-inflammatory, immunomodulatory, and digestive properties. In recent years, bromelain has attracted attention as a natural enzyme-based adjunct therapy for inflammatory disorders, including Inflammatory Bowel Disease (IBD).Type of enzyme: Cysteine protease Stem bromelain is preferred for therapeutic applications due to its higher proteolytic activity and stability. Biochemical Characteristics Molecular weight: ~24–37 kDa Active site: Cysteine–Histidine catalytic dyad Optimal pH: 5.5–8.0 broad substrate specificity Stable over a wide pH range, making it suitable for oral administration bromelain is often formulated as enteric-coated tablets or capsules to preserve activity in the gastrointestinal tract.

Gingipain: Zingiber officinalis obtain from biological source– Gingipain is a cysteine protease enzyme found in ***Ginger Zingiber officinalis*** belonging to the family ***Zingiberaceae.*** Gingipains are a group of cysteine proteases produced by the Gram-negative anaerobic bacterium Porphyromonas gingivalis, a well-known periodontal pathogen. Gingipains are major virulence factors responsible for tissue destruction, immune dysregulation, and chronic inflammation. Although primarily associated with periodontitis, recent evidence suggests that gingipains may play a role in systemic inflammatory diseases, including Inflammatory Bowel Disease (IBD), through the oral–gut axis**.** Types of Gingipains Gingipain are classified based on substrate specificity: Gingipain Substrate specificity RgpA Arginine-specific RgpB Arginine-specific Kgp Lysine-specific the gingipains are cysteine proteases biochemical characteristics enzyme class: Cysteine protease molecular weight: ~50–60 kDa active site: Cysteine–Histidine dyad strong proteolytic activity resistant to host protease inhibitors these properties allow gingipains to persist and remain active in host tissues Promote inflammatory responses in genetically susceptible individuals studies show higher abundance of periodontal pathogens in some IBD patients.

Ficin: Ficin is a plant the latex of fig trees proteolytic enzyme belonging biological source- ficin is a cysteine protease enzyme that is extracted from the latex of the fig tree ***Ficus Carica*** belonging to the family ***Moraceae***. especially Ficus carica. traditionally, ficin has been used as a digestive, anti-inflammatory, and medicinal enzyme, and recent interest has focused on its potential role in managing chronic inflammatory diseases, including Inflammatory Bowel Disease (IBD). Classification and Biochemical Properties Cysteine protease molecular weight: ~23–26 kDa active site: Cysteine–Histidine–Asparagine catalytic triad optimal pH: 6.5–8.0 These properties make ficin suitable for oral enzyme formulations when appropriately protected. IBD (Crohn's disease and ulcerative colitis) involves: Chronic intestinal inflammation, Dysregulated immune responses, Compromised epithelial barrier, Oxidative stress, Impaired digestion and nutrient absorption, Ficin may address several of these pathological features through multimodal actions.

Actinidin: Actinidia deliciosa is a plant-derived proteolytic enzyme biological source- actinidin is a proteolytic enzyme obtained from kiwi fruit ***Actinidia deliciosa*** belonging to the ***Actinidiaceae***. It is most abundantly found in kiwifruit and is responsible for the fruit's strong protein-digesting activity. Due to its anti-inflammatory, immunomodulatory, and digestive properties, actinidin has

gained attention as a potential natural enzyme-based adjunct therapy for inflammatory disorders, including Inflammatory Bowel Disease (IBD). classification and biochemical properties Enzyme class: Cysteine protease Molecular weight: ~23–25 kDa Active site: Cysteine–Histidine–Asparagine catalytic triad Optimal pH: 3.0–7.0 Substrate specificity: Broad (effective against muscle proteins, collagen, and dietary proteins) these characteristics make actinidin suitable for oral digestive and anti-inflammatory formulations. IBD (Crohn's disease and ulcerative colitis) involves: Persistent intestinal inflammation Dysregulated immune responses Compromised epithelial barrier integrity Increased oxidative stress impaired digestion and nutrient absorption Actinidin may help mitigate these pathological features via multi-target mechanisms.

Cardosins : Cynara Cardosins are a group of aspartic proteases isolated from the flower tissues of cardoon and biological source-cardosin is an spartic protease present is large amount in the pistils of cardoon flower ***Cynara Cardunculus*** belonging to the family ***Asteraceae***. A plant traditionally used in Mediterranean medicine and cheese-making. Recent studies suggest that cardosins possess proteolytic, anti-inflammatory, and immunoregulatory properties, making them potential candidates for natural enzyme-based adjunct therapy in inflammatory diseases such as Inflammatory Bowel Disease (IBD). Floral extracts Major Types Cardosin A Cardosin B These enzymes are synthesized as inactive precursors and become active under acidic conditions. Classification and Biochemical Properties Aspartic protease Molecular weight: ~30–35 kDa Catalytic residues: Two aspartic acid residues its IBD is characterized by: Chronic intestinal inflammation, Dysregulated immune responses, Protease imbalance in the gut, Epithelial barrier dysfunction Aspartic proteases like cardo sins may help regulate protease–antiprotease balance, which is often disturbed in IBD. Mechanisms of Action of Cardosin in IBD Anti-Inflammatory Effects Modulates inflammatory mediator processing Reduces activation of pro-inflammatory pathways may indirectly suppress cytokines such as TNF-α and IL-6.

5. CONCLUSION:-

Natural enzymes represent a promising and emerging therapeutic approach for the management of intestinal inflammation and inflammatory bowel disease (IBD). Accumulating experimental and clinical evidence indicates that enzymes derived from plant, microbial, and animal sources can modulate key inflammatory pathways involved in IBD pathogenesis. These enzymes exert their beneficial effects by regulating pro-inflammatory cytokines, reducing oxidative stress, enhancing epithelial barrier integrity, and restoring immune homeostasis within the intestinal microenvironment. In addition, certain natural enzymes contribute to maintaining gut microbiota balance, which is increasingly recognized as a critical factor in IBD progression and remission. Compared with conventional pharmacological therapies, natural enzymes may offer advantages such as improved safety profiles, reduced systemic side effects, and potential for long-term use as adjunctive or supportive treatments. Their multi-targeted mechanisms of action align well with the complex and multifactorial nature of IBD. However, despite encouraging preclinical findings, clinical evidence remains limited, and challenges such as enzyme stability, bioavailability, optimal dosing, and standardization need to be addressed. In conclusion, natural enzymes hold significant potential as novel modulators of intestinal inflammation and as complementary strategies in IBD therapy. Future well-designed clinical trials and mechanistic studies are essential to validate their efficacy, establish therapeutic guidelines, and facilitate their integration into evidence-based treatment regimens for patients with inflammatory bowel disease.

6 Bibliography:

1. Fuss, I. J., & Mannon, P. J. (2007). The fundamental basis of inflammatory bowel disease. *The Journal of Clinical Investigation, 117*(3), 514–521.
2. Rashid, M., Kaur, V., Hallan, S. S., Sharma, S., & Mishra, N. (2016). Microparticles as controlled drug delivery carrier for the treatment of ulcerative colitis: A brief review. *Saudi Pharmaceutical Journal, 24*(4), 458–472.
3. Rogler, G. (2014). Chronic ulcerative colitis and colorectal cancer. *Cancer Letters, 345*(2), 235–241.
4. Navaneethan, U., Jegadeesan, R., Gutierrez, N. G., Venkatesh, P. G., Hammel, J. P., Shen, B., & Kiran, R. P. (2013). Progression of low-grade dysplasia to advanced neoplasia based on the location and morphology of dysplasia in ulcerative colitis patients with extensive colitis under colonoscopic surveillance. *Journal of Crohn's and Colitis, 7*(12), e684–e691.
5. Kedia, S., & Ahuja, V. (2017). Epidemiology of inflammatory bowel disease in India: The great shift east. *Inflammatory Intestinal Diseases, 2*(2), 102–115.
6. Ray, G. (2018). Long-term maintenance treatment of ulcerative colitis in eastern India: A 15-year follow-up. *Tropical Doctor, 48*(3), 207–213.
7. Ng, S. C., Tang, W., Ching, J. Y., Wong, M., Chow, C. M., Hui, A. J., Wong, T. C., Leung, V. K., Tsang, S. W., Yu, H. H., & Li, M. F. (2013). Incidence and phenotype of inflammatory bowel disease based on results from the Asia-Pacific Crohn's and Colitis Epidemiology Study. *Gastroenterology, 145*(1), 158–165.
8. Kucharzik, T., & Asthana, A. K. (2019). Medical management 2: Conventional. In *Inflammatory bowel disease nursing manual* (pp. 81–90). Springer.
9. Rizzello, F., Salice, M., Calabrese, C., Mazza, M., Calafiore, A., Calandrini, L., Hrustemovic, H. P., Campieri, M., & Gionchetti, P. (2019). Medical treatment of ulcerative colitis: Does traditional therapy still have a role? In *Ulcerative colitis* (pp. 93–104).

Springer.
10. Managlia, E., Katzman, R. B., Brown, J. B., & Barrett, T. A. (2013). Antioxidant properties of mesalamine in colitis inhibit phosphoinositide 3-kinase signaling in progenitor cells. *Inflammatory Bowel Diseases, 19*(10), 2051–2060.
11. Tripathi, K., & Feuerstein, J. D. (2019). New developments in ulcerative colitis: Latest evidence on management, treatment, and maintenance. *Drugs in Context, 8*, 1–12.
12. Zipporah, I. E., Kaur, L., Morris, G., Bickston, P., & Anton, A. (2019). The use of probiotics in the maintenance of remission in ulcerative colitis: A systematic review. *American Journal of Gastroenterology, 114*(Suppl. 1), S1.
13. Floch, M. H. (2018). The role of prebiotics and probiotics in gastrointestinal disease. *Gastroenterology Clinics of North America, 47*(1), 179–191.
14. Kapoor, D., Maheshwari, R., Verma, K., Sharma, S., Ghode, P., & Tekade, R. K. (2020). Coating technologies in pharmaceutical product development. In *Drug delivery systems* (pp. 665–719). Academic Press.
15. Noreen, A., Akram, J., Rasul, I., Mansha, A., Yaqoob, N., Iqbal, R., Tabasum, S., Zuber, M., & Zia, K. M. (2017). Pectins functionalized biomaterials: A new viable approach for biomedical applications. *International Journal of Biological Macromolecules, 101*, 254–272.
16. Kang, R. K., Mishra, N., & Rai, V. K. (2020). Guar gum microparticles for targeted co-delivery of doxorubicin and metformin HCl for improved specificity and efficacy against colon cancer: In vitro and in vivo studies. *AAPS PharmSciTech, 21*(2), 1–14.
17. Singh, M. N., Hemant, K. S., Ram, M., & Shivakumar, H. G. (2010). Microencapsulation: A promising technique for controlled drug delivery. *Research in Pharmaceutical Sciences, 5*(2), 65–77.
18. Nidhi, Dadwal, A., Hallan, S. S., Sharma, S., & Mishra, N. (2017). Development of enteric-coated microspheres of embelin for their beneficial pharmacological potential in ulcerative colitis. *Artificial Cells, Nanomedicine, and Biotechnology, 45*(6), 1092–1100.
19. Badhana, S., Garud, N., & Garud, A. (2013). Colon-specific drug delivery of mesalamine using Eudragit S100-coated chitosan microspheres for the treatment of ulcerative colitis. *International Current Pharmaceutical Journal, 2*(3), 42–48.
20. Sharma, A., Kaur, N., Sharma, S., Sharma, A., Rathore, M. S., Ajay, K., & Mishra, N. (2018). Embelin-loaded guar gum microparticles for the management of ulcerative colitis. *Journal of Microencapsulation, 35*(2), 181–191.
21. Varshosaz, J., Emami, J., Tavakoli, N., Minaiyan, M., Rahmani, N., Dorkoosh, F., & Mahzouni, P. (2012). Pectin film-coated pellets for colon-targeted delivery of budesonide: In vitro and in vivo evaluation in induced ulcerative colitis in rats. *Iranian Journal of Pharmaceutical Research, 11*(3), 733–743.
22. Sureshkumar, R., Munikumar, M., Ganesh, G. N., Jawahar, N., Nagasamy Venkatesh, D., Senthil, V., Raju, L., & Samantha, M. K. (2014). Formulation and evaluation of pectin–hydroxypropyl methylcellulose coated curcumin pellets for colon delivery. *Asian Journal of Pharmaceutics, 8*(3), 145–152.
23. Fan, L., Zuo, S., Tan, H., Hu, J., Cheng, J., Wu, Q., & Nie, S. P. (2020). Preventive effects of pectin with various degrees of esterification on ulcerative colitis in mice. *Food & Function, 11*, 2886–2897.
24. Avachat, A. M., & Shinde, A. S. (2016). Feasibility studies of concomitant administration of optimized formulation of probiotic-loaded vancomycin hydrochloride pellets for colon delivery. *Drug Development and Industrial Pharmacy, 42*(1), 80–90.
25. Podolsky, D. K. (2002). Inflammatory bowel disease. *New England Journal of Medicine, 347*(6), 417–429.
26. Kaser, A., Zeissig, S., & Blumberg, R. S. (2010). Inflammatory bowel disease. *Annual Review of Immunology, 28*, 573–621.
27. Danese, S., & Fiocchi, C. (2011). Ulcerative colitis. *New England Journal of Medicine, 365*(18), 1713–1725.
28. Abraham, C., & Cho, J. H. (2009). Inflammatory bowel disease. *New England Journal of Medicine, 361*(21), 2066–2078.
29. Barrett, J. C., Hansoul, S., Nicolae, D. L., Cho, J. H., Duerr, R. H., Rioux, J. D., … Daly, M. J. (2008). Genome-wide association defines more than 30 distinct susceptibility loci for Crohn's disease. *Nature Genetics, 40*(8), 955–962.
30. Parkes, M., & Jewell, D. (2001). Ulcerative colitis and Crohn's disease: Molecular genetics and clinical implications. *Expert Reviews in Molecular Medicine, 2001*, 1–18.
31. Bousvaros, A., Antonioli, D. A., Colletti, R. B., Dubinsky, M. C., Glickman, J. N., Gold, B. D., … North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition. (2007). Differentiating ulcerative colitis from Crohn disease in children and young adults. *Journal of Pediatric Gastroenterology and Nutrition, 44*(5), 653–674.
32. Wu, F., Dassopoulos, T., Cope, L., Maitra, A., Brant, S. R., Harris, M. L., … Bayless, T. M. (2007). Genome-wide gene expression differences in Crohn's disease and ulcerative colitis. *Inflammatory Bowel Diseases, 13*(7), 807–821.
33. Xavier, R. J., & Podolsky, D. K. (2007). Unravelling the pathogenesis of inflammatory bowel disease. *Nature, 448*(7152), 427–434.
34. Jalley, T. N., Abreu, M. T., Achkar, J. P., Bernstein, C. N., Dubinsky, M. C., Hanauer, S. B., … Sandborn, W. J. (2011). An evidence-based systematic review on medical therapies for inflammatory bowel disease. *American Journal of Gastroenterology, 106*(Suppl. 1), S2–S25.*

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