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BOVINE COLOSTRUM SUPPLEMENTATION IN INFLAMMATORY BOWEL DISEASE: BENEFITS AND ITS ROLE IN GASTROINTESTINAL HEALTH

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ABSTRACT

Growing evidence suggests that many gastrointestinal and some systemic diseases involve dysfunction of the mucosal barrier. Colostrum is the milk produced during the first few days after birth. Bovine colostrum, a nutrient-rich fluid containing growth factors, hormones, and paracrine substances, shows promise in promoting mucosal healing in various inflammatory, infectious, and injury-related conditions. This review outlines the structure and function of the intestinal barrier and how its disruption contributes to diseases such as inflammatory bowel disease (IBD).
IBD comprises chronic, relapsing disorders with unclear causes, and current treatments -targeting immune dysregulation and microbiota imbalances - remain inadequate. Therapies include anti-inflammatory drugs, immunosuppressants, biologics, and supportive interventions like diet and supplements. We explore bovine colostrum as a potential complementary therapy for IBD, reviewing its active components and their gastrointestinal effects based on in vitro and in vivo studies, while also considering its benefits.

KEYWORDS

Bovine Colostrum, Inflammatory Bowel Disease, IBD, Treatment, Gastrointestinal, Immunoglobulins, Leukocytes, Cytokines, Growth Factors, Lactoferrin

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1. Introduction

A wide range of gastrointestinal diseases is associated with the loss of mucosal integrity. This include macroscopic ulceration like IBD. The failure of the intestinal barrier, often observed in IBD [1], results in microbial translocation, where bacteria, viruses, and their pathogen-associated molecular patterns (PAMPs) migrate from the gut lumen into the systemic circulation and lymphatic system [2,3]. This translocation subsequently triggers systemic inflammation [2,3,4].

IBD refers to a group of chronic, relapsing disorders of the gastrointestinal (GI) tract, with Crohn's disease (CD) and ulcerative colitis (UC) being the primary conditions [5]. The pathogenesis of IBD involves a complex interaction of genetic, environmental, and microbiota-related factors, along with an abnormal immune response [6,7,8]. As a result, current therapeutic approaches primarily focus on addressing immune system dysfunctions that contribute to the development of chronic intestinal inflammation [7]. Moreover, recent studies suggest that the pathogenesis of IBD can be influenced by dietary habits (such as those associated with the modern Western diet) and a high-stress lifestyle [9,10].

Damage to the intestinal epithelium, one of the body's key physical barriers, which is covered by a mucous layer and exposed to external agents (such as food antigens or bacteria), may lead to intestinal inflammation [11]. Dysfunction in the intestinal epithelium is also linked to nutrient malabsorption. Furthermore, epithelial cells are capable of synthesizing antimicrobial peptides, and research has shown that these peptides exhibit defective expression in CD patients [12,13].

Current treatment strategies for IBD include anti-inflammatory drugs like aminosalicylates and corticosteroids, immunosuppressive drugs (e.g., methotrexate, azathioprine), antibiotics, and biologic therapies (e.g., infliximab, vedolizumab) [7,14,15]. Supportive treatments involve a balanced and personalized diet, a healthy lifestyle, and stress management [9,10]. However, the existing therapeutic options for IBD patients remain inadequate, emphasizing the need for new treatment strategies with novel mechanisms of action.

One promising alternative is bovine colostrum (BC), which is the milk produced by female mammals during the first three days after giving birth before it transitions into mature milk [16]. Numerous studies have confirmed that the components of BC may positively influence the clinical progression of gastrointestinal diseases, including IBD [17].

2. The Mucosal Barrier in the Gut

The main role of the gastrointestinal tract is digestion and absorption of nutrients from food. In addition to this, the gut serves as a habitat for a vast and diverse microorganisms - including bacteria, viruses, protozoa, and fungi - collectively known as the microbiota. Food entering the digestive system may also carry various microbes, some of which can be harmful. The mucosal barrier functions as both a structural and physiological interface that separates the host from the external environment contained within the gut lumen.

The intestinal barrier consists of several key components:

- **Gastric acid**, which acts as the first line of defense by restricting microbial entry into the intestines.
- **The mucus layer** [18], which establishes a diffusion gradient filled with antimicrobial substances - such as nitric oxide, defensins, and other antimicrobial peptides [19] - as well as immunoglobulins, primarily secretory IgA.
- **The epithelial lining**, composed of a single layer of cells connected by tight junctions and other adhesion structures [20], which is constantly renewed from intestinal crypts.
- **Intraepithelial lymphocytes (IELs)**, a population of resident immune cells located within the epithelial layer.
- **Lymphocytes and macrophages in the lamina propria**, which migrate to the mesenteric lymph nodes and contribute to memory immune responses.
- **Further downstream**, liver macrophages (Kupffer cells) act as an additional defense mechanism, neutralizing pathogens and PAMPs that bypass the upstream barrier components; this element will not be elaborated on here.
- **The gut microbiota**, which plays a protective role by preventing colonization by harmful external microorganisms.

However, pathogens and toxins are able to penetrate the mucosal barrier, particularly when exposure levels are high, but the gastrointestinal mucosa has developed various mechanisms to prevent severe outcomes [18].

3.Gastrointestinal Repair Mechanisms

The epithelial lining of the gut has one of the fastest turnover rates among all the cell types in the human body. With a turnover period of 3-5 days, cells damaged by pathogens or toxins are quickly replaced. Their replacement allows for clinical recovery. The rapid turnover of the intestinal epithelium is dependent on the availability of luminal nutrients, and a lack of them (such as in starvation) can lead to atrophy and a weakened barrier function [21]. This process is partly regulated by glucagon-like peptide 2 (GLP2), a product of the glucagon gene, which has important trophic effects on the intestine. Therapeutic administration of GLP2 can stimulate mucosal hypertrophy even in the absence of luminal nutrients [22]. GLP2's actions are primarily mediated through the secretion of insulin-like growth factor-1 (IGF-1) by neural and myofibroblast cells [23].

Ulceration refers to a disruption in the epithelial surface. The factors that allow ulcers to persist are not fully understood, but the process of wound healing (restitution) in epithelial monolayers has been well-researched. When a monolayer is damaged, cell migration from the edges and increased cell turnover help close the wound and restore integrity [24]. In vivo, injury leads to dedifferentiation and the appearance of injury-related cell types, such as surface mucosal cells, ulcer-associated cells, mucosal neck cells, spasmolytic polypeptide-expressing metaplasia (SPEM), and pyloric metaplasia [25]. Interestingly, many of these metaplastic cells produce mucins, indicating that mucus layer restoration is a key part of the healing process. Polyamines promote epithelial restitution through calcium signaling [26,27]. Growth factor peptides are also necessary for these processes [28], including hormones (LnRH), cytokines, IGF-1, EGF, TGFα and β, PDGF, GH, GHRF, and milk fat globule proteins [29]. Colostrum contains many of these factors and supports epithelial restitution [30].

This offers a compelling reason to explore the potential therapeutic use of colostrum in conditions involving loss of epithelial integrity. It was observed years ago that the expression of receptors for many growth factors is limited to the basolateral surface of enterocytes, suggesting that trophic factors only act on the epithelium when its integrity is compromised, a phenomenon known as luminal surveillance [31]. Many of these factors also have anti-inflammatory effects [32]. Recently, innate immune receptors have been shown to play a role in restitution and repair [33]. While EGF was once considered the primary driver of epithelial repair, recent evidence suggests that other peptides, such as Neuregulin-1, which signal through EGFR-related pathways, may be more potent in promoting this process [34].

4. Constituents of Bovine Colostrum

Bovine colostrum (BC) consists of over 250 functional components, including immune-boosting peptides and antimicrobial substances [35,36]. Key ingredients in BC include macronutrients, immunoglobulins, leukocytes, cytokines, growth factors, lactoferrin (LF), lysozyme (LZ), casein, proline-rich polypeptides, glycomacropeptide (GMP), lactalbumin (LA), and enzymes such as lactoperoxidase (LPO) (Table 1). Additional components include vitamins, macro- and microelements, hormones, nucleotides, and gangliosides [37,38]. Colostrum plays an essential role in the development of the immune system in infants and aids in the growth, maturation, and repair of various tissues. As a result, BC contains significantly higher concentrations of growth-promoting factors than mature milk [16,35,37]. Commercially available colostrum comes in various forms, including powder, concentrate, lozenges, fortified milk and beverages, yogurts, butter, and even chewing gums. These products may vary in terms of compound quality, quantity, and bioavailability [39,40].

4.1 Immunoglobulins

Bovine colostrum (BC) contains five types of immunoglobulins: IgG, IgA, and IgM in significant amounts, along with trace levels of IgD and IgE. These immunoglobulins provide protection against various pathogens, including bacteria, viruses, parasites, and fungi [41]. In the gastrointestinal tract, their main function is to bind to microorganisms, thereby blocking their interaction with the intestinal epithelium and preventing their entry into the bloodstream [42]. Immunoglobulins are particularly vital for ruminants, as their syndesmochorial placenta does not allow the transfer of antibodies to the fetus during gestation [43].

4.2 Leukocytes

Bovine colostrum contains approximately 10^6 leukocytes per milliliter [44], predominantly made up of colostral mononuclear cells (CMCs) like macrophages and lymphocytes, along with some polymorphonuclear and epithelial cells [45,46]. Research has shown that CMCs have the ability to present antigens, which allows them to influence the immune response and help regulate the balance between immune tolerance and allergic reactions [47].

4.3 Cytokines

In addition to cytokines released by leukocytes found in colostrum, various cytokines are also synthesized in the mammary glands and subsequently secreted into colostrum [35,48]. These cytokines contribute to the development of the infant's immune system and help regulate inflammatory responses [49,50]. They can have either pro-inflammatory or anti-inflammatory effects, playing a role in defending the body against viral, bacterial, and fungal infections [35,51,52]. Under normal conditions, cytokines like IL-1β, IL-6, and TNF-α are commonly detected, and their levels are significantly higher in bovine colostrum than in mature milk [52].

4.4 Growth factors

Colostrum contains several key growth factors, including insulin-like growth factors (IGF) 1 and 2, transforming growth factors (TGF) β1 and 2, fibroblast growth factors (FGF) 1 and 2, epidermal growth factor (EGF), β-cellulin (BTC), platelet-derived growth factor (PDGF), and vascular endothelial growth factor (VEGF). Among these, IGF-1 and IGF-2 are the most abundant. IGF-1 plays a crucial role in promoting cell growth, proliferation, repair processes, and the metabolism of macronutrients.[53]

4.5 Lactoferrin

Lactoferrin (LF) is a glycoprotein that binds iron. Its biological effects include anti-infective, immune-modulating, and potentially pro- or anti-inflammatory actions, depending on the immune status of the host [54,55]. LF is highly effective against a broad range of viruses, as well as several species of bacteria, fungi, and protozoa. Additionally, it has the ability to influence the intestinal microbiota [56,57]. LF also interacts with lymphocytes, macrophages, granulocytes, and natural killer (NK) cells, affecting their functions such as cytokine production, proliferation, maturation, migration, activation, and cytotoxicity [56,58]. For example, LF can enhance the activity of NK cells, promote Th1 cell immune responses, and increase cytokine secretion to prevent viral infections [56].

Colostrum also contains other compounds that contribute to its beneficial effects on gut regeneration. These include lysozyme, glycomacropeptide, proline-rich polypeptide complex, lactoperoxidase and vitamins. As mentioned earlier, the components of colostrum possess antimicrobial and immunomodulatory properties that could influence inflammatory processes in IBD.

5. Inflammatory Bowel Disease (IBD)

Inflammatory bowel disease (IBD) is a persistent and recurring inflammatory condition of the gastrointestinal tract that affects individuals across all age groups [59,60,61]. The most prevalent forms are ulcerative colitis and Crohn’s disease, though other less common types - such as collagenous colitis and lymphocytic colitis - can also occur. Although the precise etiology of IBD remains unclear, it is thought to result from complex interactions between host genetics, intestinal microbiota, and environmental influences, which lead to an abnormal immune response within the gut. Therapeutic options currently include aminosalicylates, corticosteroids, antibiotics, immunosuppressive agents, stem cell therapy, and surgical procedures [62,63,64]. Despite this variety, no single treatment has proven universally effective.

Bovine colostrum (BC) has demonstrated encouraging anti-inflammatory and symptom-relieving effects in both experimental colitis models and clinical studies. Additional advantages have been noted in children with Crohn’s disease who were administered TGF-β-enriched nutritional formulations. These interventions were linked to improvements in disease activity scores (PCDAI), body mass index, and systemic inflammation markers [65,66]. Since TGF-β is a major bioactive component of bovine colostrum, these findings point to its potential as a supportive therapy for patients with IBD.

Conclusions

Bovine colostrum (BC) contains a wide array of bioactive components that could potentially improve the clinical progression of inflammatory diseases, including IBD. This complex biological fluid is rich in growth factors, nutrients, hormones, and paracrine factors, all of which are thought to play a role in promoting mucosal healing in various inflammatory, infectious, and injury-related conditions. Increasing evidence suggests that these properties may be beneficial for both treating and preventing several disorders. Every element of colostrum provides valuable health benefits by supplying vital components to key systems in the body. It demonstrates anti-inflammatory, antioxidant, antibacterial, prebiotic, and antiviral properties. The leukocytes in BC, particularly colostral mononuclear cells, contribute to immune regulation by presenting antigens and balancing immune responses. Immunoglobulins are crucial for immune defense, protecting against bacterial and viral infections, as well as modulating immune system activation. Lactoferrin encourages cellular growth, supports the proliferation of beneficial bacteria, boosts immune defense, and has promising therapeutic potential. Cytokines present in BC assist in developing the immune system and modulating inflammatory responses, with higher concentrations found in colostrum compared to mature milk. Growth factors such as IGF-1 and IGF-2 support cell growth, repair, and metabolism. Additionally, compounds like lysozyme, glycomacropeptide, and lactoperoxidase further contribute to BC's beneficial effects on gut regeneration. Overall, BC presents promising therapeutic potential for immune modulation and gut health.

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REFERENCES

1. Aldars-García, L., Chaparro, M., & Gisbert, J. (2021). Systematic review: The gut microbiome and its potential clinical application in inflammatory bowel disease. *Microorganisms, 9*(5), 977. https://doi.org/10.3390/microorganisms9050977
2. Luján, J. A., Rugeles, M. T., & Taborda, N. A. (2019). Contribution of the microbiota to intestinal homeostasis and its role in the pathogenesis of HIV-1 infection. *Current HIV Research, 17*(1), 13–25. https://doi.org/10.2174/1570162X17666190311114808
3. Marchetti, G., Tincati, C., & Silvestri, G. (2013). Microbial translocation in the pathogenesis of HIV infection and AIDS. *Clinical Microbiology Reviews, 26*(1), 2–18. https://doi.org/10.1128/CMR.00050-12
4. Fukui, H. (2016). Increased intestinal permeability and decreased barrier function: Does it really influence the risk of inflammation? *Inflammatory Intestinal Diseases, 1*(3), 135–145. https://doi.org/10.1159/000447252
5. Holtmann, G. J., Ford, A. C., & Talley, N. J. (2016). Pathophysiology of irritable bowel syndrome. *The Lancet Gastroenterology & Hepatology, 1*(2), 133–146. https://doi.org/10.1016/S2468-1253(16)30023-1
6. Bernstein, C. N., Eliakim, A., Fedail, S., Fried, M., Gearry, R., Goh, K. L., Hamid, S., Khan, A. G., Khalif, I., Ng, S. C., et al. (2016). World Gastroenterology Organisation global guidelines: Inflammatory bowel disease. *Journal of Clinical Gastroenterology, 50*(10), 813–818. https://doi.org/10.1097/MCG.0000000000000660
7. Zhang, Y. Z., & Li, Y. Y. (2014). Inflammatory bowel disease: Pathogenesis. *World Journal of Gastroenterology, 20*(1), 91–99. https://doi.org/10.3748/wjg.v20.i1.91
8. Xu, H., Liu, M., Cao, J., Li, X., Fan, D., Xia, Y., Lu, X., Li, J., Ju, D., & Zhao, H. (2019). The dynamic interplay between the gut microbiota and autoimmune diseases. *Journal of Immunology Research, 2019*, 1–14. https://doi.org/10.1155/2019/7548254
9. Lee, D., Albenberg, L., Compher, C., Baldassano, R., Piccoli, D., Lewis, J. D., & Wu, G. D. (2015). Diet in the pathogenesis and treatment of inflammatory bowel diseases. *Gastroenterology, 148*(6), 1087–1106. https://doi.org/10.1053/j.gastro.2015.01.007
10. Sigall-Boneh, R., Levine, A., Lomer, M., Wierdsma, N., Allan, P., Fiorino, G., Gatti, S., Jonkers, D., Kierkus, J., Katsanos, K. H., et al. (2017). Research gaps in diet and nutrition in inflammatory bowel disease: A topical review by D-ECCO Working Group (Dietitians of ECCO). *Journal of Crohn's and Colitis, 11*(12), 1407–1419. https://doi.org/10.1093/ecco-jcc/jjx109
11. Salim, S. Y., & Söderholm, J. D. (2011). Importance of disrupted intestinal barrier in inflammatory bowel diseases. *Inflammatory Bowel Diseases, 17*(1), 362–381. https://doi.org/10.1002/ibd.21403
12. Wehkamp, J., Harder, J., Weichenthal, M., Mueller, O., Herrlinger, K. R., Fellermann, K., Schroeder, J. M., & Stange, E. F. (2003). Inducible and constitutive beta-defensins are differentially expressed in Crohn's disease and ulcerative colitis. *Inflammatory Bowel Diseases, 9*(4), 215–223. https://doi.org/10.1097/00054725-200307000-00001
13. Owczarek, D., Rodacki, T., Domagała-Rodacka, R., Cibor, D., & Mach, T. (2016). Diet and nutritional factors in inflammatory bowel diseases. *World Journal of Gastroenterology, 22*(3), 895–905. https://doi.org/10.3748/wjg.v22.i3.895
14. Fichna, J. (2016). Inflammatory bowel disease treatment. *Pharmacological Reports, 68*(4), 787–788. https://doi.org/10.1016/j.pharep.2016.05.010
15. Yamamoto-Furusho, J. K. (2012). Treatment of inflammatory bowel disease. *Revista de Gastroenterología de México, 77*(1), 39–41. https://doi.org/10.1016/j.rgmx.2011.10.007
16. Langer, P. (2009). Differences in the composition of colostrum and milk in eutherians reflect differences in immunoglobulin transfer. *Journal of Mammalogy, 90*(2), 332–339. https://doi.org/10.1644/08-MAMM-R-287.1
17. Bodammer, P., Maletzki, C., Waitz, G., & Emmrich, J. (2011). Prophylactic application of bovine colostrum ameliorates murine colitis via induction of immunoregulatory cells. *Journal of Nutrition, 141*(6), 1056–1061. https://doi.org/10.3945/jn.110.137059
18. Schroeder, B. O. (2019). Fight them or feed them: How the intestinal mucus layer manages the gut microbiota. *Gastroenterology Reports, 7*(1), 3–12. https://doi.org/10.1093/gastro/goy052
19. Bevins, C. L., & Salzman, N. (2011). Paneth cells, antimicrobial peptides and maintenance of intestinal homeostasis. *Nature Reviews Genetics, 9*(5), 356–368. https://doi.org/10.1038/nrmicro2546

20. Paradis, T., Bègue, H., Basmaciyan, L., Dalle, F., & Bon, F. (2021). Tight junctions as a key for pathogen invasion in intestinal epithelial cells. *International Journal of Molecular Sciences, 22*(5), 2506. https://doi.org/10.3390/ijms22052506
21. Kelly, P. (2020). Starvation and its effects on the gut. *Advances in Nutrition, 12*(3), 897–903. https://doi.org/10.1093/advances/nmaa135
22. Brubaker, P. L. (2011). Glucagon-like peptide-2 and the regulation of intestinal growth and function. *Comprehensive Physiology, 8*(3), 1185–1210. https://doi.org/10.1002/cphy.c170055
23. Fesler, Z., Mitova, E., & Brubaker, P. L. (2020). GLP-2, EGF, and the intestinal epithelial IGF-1 receptor interactions in the regulation of crypt cell proliferation. *Endocrinology, 161*(6), bqaa040. https://doi.org/10.1210/endocr/bqaa040
24. Galura, G. M., Chavez, L. O., Robles, A., & McCallum, R. (2019). Gastroduodenal injury: Role of protective factors. *Current Gastroenterology Reports, 21*(5), 34. https://doi.org/10.1007/s11894-019-0701-x
25. Goldenring, J. R. (2018). Pyloric metaplasia, pseudopyloric metaplasia, ulcer-associated cell lineage and spasmolytic polypeptide-expressing metaplasia: Reparative lineages in the gastrointestinal mucosa. *The Journal of Pathology, 245*(2), 132–137. https://doi.org/10.1002/path.5066
26. Rao, J. N., Rathor, N., Zhuang, R., Zou, T., Liu, L., Xiao, L., Turner, D. J., & Wang, J.-Y. (2012). Polyamines regulate intestinal epithelial restitution through TRPC1-mediated Ca^{2+} signaling by differentially modulating STIM1 and STIM2. *American Journal of Physiology – Cell Physiology, 303*(3), C308–C317. https://doi.org/10.1152/ajpcell.00120.2012
27. Timmons, J., Chang, E. T., Wang, J.-Y., & Rao, J. N. (2012). Polyamines and gut mucosal homeostasis. *Journal of Gastrointestinal & Digestive System, 2*(S7), 1. https://doi.org/10.4172/2161-069X.S7-001
28. Playford, R., & Weiser, M. (2021). Bovine colostrum: Its constituents and uses. *Nutrients, 13*(1), 265. https://doi.org/10.3390/nu13010265
29. Chatterton, D. E., Nguyen, D. N., Bering, S. B., & Sangild, P. T. (2013). Anti-inflammatory mechanisms of bioactive milk proteins in the intestine of newborns. *International Journal of Biochemistry & Cell Biology, 45*(8), 1730–1747. https://doi.org/10.1016/j.biocel.2013.04.028
30. Blais, M., Pouliot, Y., Gauthier, S., Boutin, Y., & Lessard, M. (2014). A gene expression programme induced by bovine colostrum whey promotes growth and wound-healing processes in intestinal epithelial cells. *Journal of Nutritional Science, 3*, e57. https://doi.org/10.1017/jns.2014.56
31. Playford, R. J., Macdonald, C. E., & Johnson, W. S. (2000). Colostrum and milk-derived peptide growth factors for the treatment of gastrointestinal disorders. *American Journal of Clinical Nutrition, 72*(1), 5–14. https://doi.org/10.1093/ajcn/72.1.5
32. Asaro, J. A., Khan, Z., Brewer, M., Klose, K., Pesce, C., Schanler, R. J., & Codipilly, C. N. (2021). Relationship between milk fat globule-epidermal growth factor 8 and intestinal cytokines in infants born preterm. *Journal of Pediatrics, 230*, 71–75.e1. https://doi.org/10.1016/j.jpeds.2020.11.014
33. Stenson, W. F., & Ciorba, M. A. (2021). Nonmicrobial activation of TLRs controls intestinal growth, wound repair, and radioprotection. *Frontiers in Immunology, 11*, 617510. https://doi.org/10.3389/fimmu.2020.617510
34. Jardé, T., Chan, W. H., Rossello, F. J., Kahlon, T. K., Theocharous, M., Arackal, T. K., Flores, T., Giraud, M., Richards, E., Chan, E., et al. (2020). Mesenchymal niche-derived neuregulin-1 drives intestinal stem-cell proliferation and regeneration of damaged epithelium. *Cell Stem Cell, 27*(4), 646–662.e7. https://doi.org/10.1016/j.stem.2020.06.021
35. Menchetti, L., Traina, G., Tomasello, G., Casagrande-Proietti, P., Leonardi, L., Barbato, O., & Brecchia, G. (2016). Potential benefits of colostrum in gastrointestinal diseases. *Frontiers in Bioscience (Scholar Edition), 8*, 331–351. https://doi.org/10.2741/s465
36. Puppel, K., Gołębiewski, M., Grodkowski, G., Slósarz, J., Kunowska-Slósarz, M., Solarczyk, P., Łukasiewicz, M., Balcerak, M., & Przysucha, T. (2019). Composition and factors affecting quality of bovine colostrum: A review. *Animals, 9*(12), 1070. https://doi.org/10.3390/ani9121070
37. Lönnerdal, B. (2013). Bioactive proteins in breast milk. *Journal of Paediatrics and Child Health, 49*(Suppl 1), 1–7. https://doi.org/10.1111/jpc.12104
38. Playford, R. J., Floyd, D. N., Macdonald, C. E., Calnan, D. P., Adenekan, R. O., Johnson, W., Goodlad, R. A., & Marchbank, T. (1999). Bovine colostrum is a health food supplement which prevents NSAID-induced gut damage. *Gut, 44*(5), 653–658. https://doi.org/10.1136/gut.44.5.653
39. Christiansen, S., Guo, M., & Kjelden, D. (2010). Chemical composition and nutrient profile of low-molecular-weight fraction of bovine colostrum. *International Dairy Journal, 20*(9), 630–636. https://doi.org/10.1016/j.idairyj.2010.02.008
40. Silva, E., Rangel, A., Mürmam, L., & Bezerra, M. F. (2019). Bovine colostrum: Benefits of its use in human food. *Food Science and Technology, 39*(Suppl 1), 355–362. https://doi.org/10.1590/fst.04318
41. Mehra, R., Marnila, P., & Korhonen, H. (2006). Milk immunoglobulins for health promotion. *International Dairy Journal, 16*(11), 1262–1271. https://doi.org/10.1016/j.idairyj.2006.06.011

42. Detzel, C. J., Horgan, A., Henderson, A. L., Petschow, B. W., Warner, C. D., Maas, K. J., & Weaver, E. M. (2015). Bovine immunoglobulin/protein isolate binds pro-inflammatory bacterial compounds and prevents immune activation in an intestinal co-culture model. *PLoS ONE, 10*(4), e0120278. https://doi.org/10.1371/journal.pone.0120278
43. Barrington, G. M., & Parish, S. M. (2001). Bovine neonatal immunology. *Veterinary Clinics of North America: Food Animal Practice, 17*(3), 463–476. https://doi.org/10.1016/S0749-0720(15)30015-5
44. Riedel-Caspari, G. (1993). The influence of colostral leukocytes on the course of an experimental *Escherichia coli* infection and serum antibodies in neonatal calves. *Veterinary Immunology and Immunopathology, 35*(3–4), 275–288. https://doi.org/10.1016/0165-2427(93)90021-R
45. Yang, T. J., Ayoub, I. A., & Rewinski, M. J. (1997). Lactation-stage-dependent changes of lymphocyte subpopulations in mammary secretions: Inversion of $CD4^{+}/CD8^{+}$ T-cell ratios at parturition. *American Journal of Reproductive Immunology, 37*(5), 378–383. https://doi.org/10.1111/j.1600-0897.1997.tb00248.x
46. Platt, R., Burdett, W., & Roth, J. A. (2006). Induction of antigen-specific T-cell subset activation to bovine respiratory disease viruses by a modified-live virus vaccine. *American Journal of Veterinary Research, 67*(7), 1179–1184. https://doi.org/10.2460/ajvr.67.7.1179
47. Le Jan, C. (1996). Cellular components of mammary secretions and neonatal immunity: A review. *Veterinary Research, 27*(4–5), 403–417. https://hal.archives-ouvertes.fr/hal-00902432
48. Hagiwara, K., Kataoka, S., Yamanaka, H., Kirisawa, R., & Iwai, H. (2000). Detection of cytokines in bovine colostrum. *Veterinary Immunology and Immunopathology, 76*(3–4), 183–190. https://doi.org/10.1016/S0165-2427(00)00219-5
49. Meki, A., Saleem, T. H., Al-Ghazali, M. H., & Sayed, A. A. (2003). Interleukins 6, 8 and 10 and tumor necrosis factor-alpha and its soluble receptor I in human milk at different periods of lactation. *Nutritional Research, 23*(7), 845–855. https://doi.org/10.1016/S0271-5317(03)00074-3
50. Gonzalez, D. D., & Dus Santos, M. J. (2017). Bovine colostral cells — The often forgotten component of colostrum. *Journal of the American Veterinary Medical Association, 250*(9), 998–1005. https://doi.org/10.2460/javma.250.9.998
51. Shing, C. M., Peake, J. M., Suzuki, K., Jenkins, D. G., & Coombes, J. S. (2009). Bovine colostrum modulates cytokine production in human peripheral blood mononuclear cells stimulated with lipopolysaccharide and phytohemagglutinin. *Journal of Interferon & Cytokine Research, 29*(1), 37–44. https://doi.org/10.1089/jir.2008.0029
52. Marek, A., Zagierski, M., Liberek, A., Aleksandrowicz, E., Korzon, M., Krzykowski, G., Kamińska, B., & Szlagatys-Sidorkiewicz, A. (2009). TGF-β1, IL-10 and IL-4 in colostrum of allergic and nonallergic mothers. *Acta Biochimica Polonica, 56*(3), 411–414. https://doi.org/10.18388/abp.2009_2460
53. McGrath, B. A., Fox, P. F., McSweeney, P. L. H., & Kelly, A. L. (2016). Composition and properties of bovine colostrum: A review. *Dairy Science & Technology, 96*(2), 133–158. https://doi.org/10.1007/s13594-015-0258-x
54. Legrand, D. (2016). Overview of lactoferrin as a natural immune modulator. *Journal of Pediatrics, 173*(Suppl 1), S10–S15. https://doi.org/10.1016/j.jpeds.2016.02.071
55. Zelechowska, P., Agier, J., & Brzezińska-Błaszczyk, E. (2016). Endogenous antimicrobial factors in the treatment of infectious diseases. *Central European Journal of Immunology, 41*(4), 419–425. https://doi.org/10.5114/ceji.2016.65134
56. Wakabayashi, H., Oda, H., Yamauchi, K., & Abe, F. (2014). Lactoferrin for prevention of common viral infections. *Journal of Infection and Chemotherapy, 20*(11), 666–671. https://doi.org/10.1016/j.jiac.2014.08.003
57. Lauterbach, R., Kamińska, E., Michalski, P., & Lauterbach, J. P. (2016). Lactoferrin — A glycoprotein of great therapeutic potential. *Developmental Period Medicine, 20*(2), 118–125. PMID: 27416630
58. Legrand, D., Elass, E., Carpentier, M., & Mazurier, J. (2005). Lactoferrin: A modulator of immune and inflammatory responses. *Cellular and Molecular Life Sciences, 62*(22), 2549–2559. https://doi.org/10.1007/s00018-005-5370-2
59. Zhang, Y. Z., & Li, Y. Y. (2014). Inflammatory bowel disease: Pathogenesis. *World Journal of Gastroenterology, 20*(1), 91–99. https://doi.org/10.3748/wjg.v20.i1.91
60. Wehkamp, J., Götz, M., Herrlinger, K., Steurer, W., & Stange, E. F. (2016). Inflammatory bowel disease. *Deutsches Ärzteblatt International, 113*(5), 72–82. https://doi.org/10.3238/arztebl.2016.0072
61. Guan, Q. (2019). A comprehensive review and update on the pathogenesis of inflammatory bowel disease. *Journal of Immunology Research, 2019*, 7247238. https://doi.org/10.1155/2019/7247238
62. Lamb, C. A., Kennedy, N. A., Raine, T., Hendy, P. A., Smith, P. J., Limdi, J. K., Hayee, B., Lomer, M. C. E., Parkes, G. C., Selinger, C., et al. (2019). British Society of Gastroenterology consensus guidelines on the management of inflammatory bowel disease in adults. *Gut, 68*(Suppl 3), S1–S106. https://doi.org/10.1136/gutjnl-2019-318484
63. Nadpara, N., Reichenbach, Z. W., Ehrlich, A. C., & Friedenberg, F. (2020). Current status of medical therapy for inflammatory bowel disease: The wealth of medications. *Digestive Diseases and Sciences, 65*(9), 2769–2779. https://doi.org/10.1007/s10620-020-06471-4

64. Seyedian, S. S., Nokhostin, F., & Malamir, M. D. (2019). A review of the diagnosis, prevention, and treatment methods of inflammatory bowel disease. *Journal of Medicine and Life, 12*(2), 113–122. https://doi.org/10.25122/jml-2018-0075
65. Hartman, C., Berkowitz, D., Weiss, B., Shaoul, R., Levine, A., Adiv, O. E., Shapira, R., Fradkin, A., Wilschanski, M., Tamir, A., et al. (2008). Nutritional supplementation with polymeric diet enriched with transforming growth factor-beta 2 for children with Crohn's disease. *Israel Medical Association Journal, 10*(7), 503–507. PMID: 18751621
66. Rubio, A., Pigneur, B., Garnier-Lengliné, H., Talbotec, C., Schmitz, J., Canioni, D., Goulet, O., & Ruemmele, F. M. (2011). The efficacy of exclusive nutritional therapy in paediatric Crohn's disease, comparing fractionated oral vs continuous enteral feeding. *Alimentary Pharmacology & Therapeutics, 33*(12), 1332–1339. https://doi.org/10.1111/j.1365-2036.2011.04662.x