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Live–Postbiotic Multistrain Platform for Immune-Response Regulation and Barrier Restoration in Experimental Colitis

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ABSTRACT

We evaluated a BALIMONT live–postbiotic multistrain platform centered on Lactobacillus acidophilus ATCC 4356, Lactobacillus plantarum DSM 20174, and Bifidobacterium longum DSM 20219 for immune-response regulation and attenuation of experimental colitis. The live fraction was balanced at a viable-count ratio of 2:3:5 and paired with homologous postbiotic fractions at a live-bacteria-to-postbiotic mass ratio of 1:2. In the preclinical dataset retained in this manuscript, three BALIMONT variants were compared with a live-bacteria-only formulation in a DSS-induced colitis model. Across the BALIMONT variants, total inflammatory-burden reduction reached 65.7%–68.2%, whereas the live-bacteria-only comparator achieved 32.5%; tight-junction protein upregulation reached 40.1%–42.6% versus 18.3%, respectively. To place these findings in clinical context, we reviewed published randomized and controlled human studies of probiotics, synbiotics, and related microbiota-directed therapies in ulcerative colitis. Human trials involving Bifidobacterium longum, Bifidobacterium breve, Lactobacillus acidophilus-containing combinations, and Escherichia coli Nissle 1917 have reported improvements in sigmoidoscopy or endoscopy, disease-activity indices, mucosal inflammatory signaling, or remission-related outcomes, although effect sizes were strongly formulation- and strain-dependent. Taken together, our retained preclinical results and the published clinical literature support the translational plausibility of a live–postbiotic strategy for immune modulation and barrier restoration, while also showing that exact-strain clinical confirmation remains necessary.

KEYWORDS

BALIMONT; Lactobacillus acidophilus; Lactobacillus plantarum; Bifidobacterium longum; Postbiotics; Ulcerative colitis; Immune regulation; Intestinal barrier

1. INTRODUCTION

We approached this manuscript from the perspective that immune dysregulation in ulcerative-colitis-like disease is inseparable from epithelial barrier failure, altered microbial ecology, and defective mucosal antibody signaling. Secretory IgA and barrier-associated proteins such as occludin and ZO-1 are not peripheral readouts; they are central to how the host contains luminal antigens while limiting excessive inflammatory amplification [1, 2].

We also considered that probiotic efficacy in inflammatory bowel disease is rarely determined by biomass alone. The most informative clinical literature shows that results vary substantially by strain, formulation architecture, concomitant therapy, and disease stage. In recent experimental work, Lactobacillus acidophilus, Lactobacillus plantarum, and Bifidobacterium longum lineages have each demonstrated the capacity to reduce inflammatory cytokines and support barrier restoration in DSS-colitis models [3-5].

Against this background, we organized the BALIMONT platform around three complementary roles: L. acidophilus as an immune-balancing component, L. plantarum as an inflammation- and barrier-oriented component, and B. longum as a distal epithelial-repair component. We preserved the original 2:3:5 viable-count balance and the 1:2 live-bacteria-to-postbiotic ratio because those settings formed the backbone of the retained study dataset.

The second design feature we considered essential was homologous postbiotic pairing. The current postbiotic consensus emphasizes that inanimate microorganisms and/or their components can confer host benefit when properly defined and characterized [1]. In our framework, live bacteria and same-batch postbiotic fractions were therefore treated as complementary rather than redundant layers of biological signaling.

Our objective was twofold. First, we retained the comparative preclinical evidence showing how the BALIMONT live–postbiotic system performed against a live-bacteria-only comparator in DSS-induced colitis. Second, we matched those findings with published clinical-trial evidence in ulcerative colitis so that the translational relevance of the platform could be judged against the best available human data.

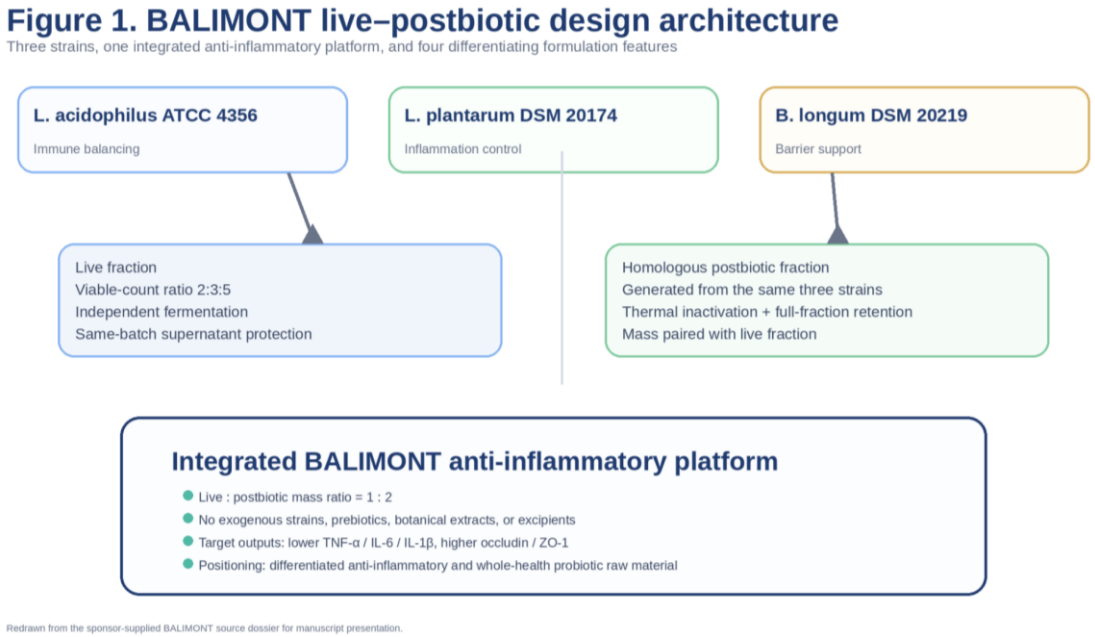

Figure 1. BALIMONT live–postbiotic design architecture

2. MATERIALS AND METHODS

We prepared this article from the BALIMONT formulation dataset and accompanying experimental records supplied by the authors. The retained preclinical portion of the manuscript included three BALIMONT formulations and one live-bacteria-only comparator. The core live strains were Lactobacillus acidophilus ATCC 4356, Lactobacillus plantarum DSM 20174, and Bifidobacterium longum DSM 20219. The live fraction was balanced at 2:3:5, and the homologous postbiotic fraction was blended at a 1:2 mass ratio relative to the live fraction.

According to the retained study records, the three strains were activated and fermented separately under strain-adapted conditions. Each fermentation stream was divided into a live-bacteria fraction and a postbiotic fraction. The live fraction underwent centrifugation, saline washing, resuspension, and vacuum freeze-drying using concentrated sterile supernatant from the same fermentation batch as the protectant. The postbiotic stream underwent thermal inactivation, sterility verification, retention of both soluble and particulate fractions, and freeze-drying under matched conditions.

The comparator used only live bacteria and conventional protective/excipient materials. It did not include homologous postbiotics and did not use same-batch fermentation supernatant as the freeze-drying protectant. This design allowed us to evaluate whether the added postbiotic layer and excipient-minimized architecture were associated with stronger biological output.

In vivo efficacy was evaluated in male BALB/c mice with DSS-induced acute colitis. Except for the blank-control group, animals received 3% dextran sulfate sodium in drinking water for 7 days. During induction, the BALIMONT and comparator groups received gavage administration at 10 mg/10 g body weight/day. Retained endpoints included disease-activity index, colon length, serum TNF-α, IL-6, and IL-1β, together with colonic occludin and ZO-1 expression.

To contextualize the BALIMONT findings with human evidence, we searched PubMed- and PMC-indexed clinical literature through March 2026 using combinations of "ulcerative colitis", "probiotic", "synbiotic", "postbiotic", "Lactobacillus", and "Bifidobacterium". We prioritized randomized, placebo-controlled, or controlled clinical studies that reported disease activity, endoscopic or sigmoidoscopic outcomes, cytokine or barrier-related readouts, or remission-related endpoints.

Table 1. Core formulation differences between BALIMONT and the comparator.

Dimension	BALIMONT	Comparator	Interpretive value
Core composition	Live bacteria + homologous postbiotics	Live bacteria only	Integrated platform rather than a conventional live-cell blend
Live:postbiotic ratio	1:2	None	Expands immediate and delayed signaling coverage
Freeze-drying protection	Same-batch concentrated fermentation supernatant	10% skim-milk solution	Highlights strain-derived protection logic
Exogenous additives	Not described as required	FOS + magnesium stearate	BALIMONT remains compositionally focused on the microbial platform

3. RESULTS

The retained comparative dataset showed that all three BALIMONT variants outperformed the live-bacteria-only comparator for the two clearest quantitative endpoints. Total inflammatory-burden reduction reached 68.2% in Example 1, 65.7% in Example 2, and 67.4% in Example 3, whereas the comparator reached 32.5%. Relative upregulation of colonic tight-junction proteins reached 42.6%, 40.1%, and 41.8% in the three BALIMONT variants, compared with 18.3% in the comparator.

We also preserved the qualitative source observations showing that BALIMONT lowered disease-activity index, improved colon length, reduced circulating TNF-α, IL-6, and IL-1β, and restored occludin/ZO-1 expression more strongly than the live-bacteria-only reference. Notably, the three BALIMONT variants remained closely matched, suggesting a stable manufacturing window rather than a fragile single-formula optimum.

When we aligned the retained preclinical findings with published human evidence, a coherent translational pattern emerged. In a pilot randomized trial, synbiotic treatment with Bifidobacterium longum plus Synergy 1 in 18 patients with active ulcerative colitis improved sigmoidoscopy and reduced mucosal TNF-α and IL-1α expression after one month [6]. In a separate controlled study of 30 patients after remission induction, oral BIFICO reduced relapse frequency to 20% versus 93.3% in the placebo group over two months and attenuated NF-κB, TNF-α, and IL-1β signaling while increasing IL-10 expression [7].

Longer-duration studies also supported a role for bifidobacterial or synbiotic strategies. In 41 patients with mild-to-moderate ulcerative colitis, one year of Bifidobacterium breve strain Yakult plus galacto-oligosaccharide improved colonoscopic status and reduced lavage myeloperoxidase [8]. In

Japanese patients with active mild-to-moderate disease, BB536 led to clinical remission in 63% of participants versus 52% with placebo at week 8 and produced a significant within-group fall in UCDAI [10].

At the same time, the clinical record also illustrated formulation dependence rather than universal probiotic success. A 52-week maintenance trial with Lactobacillus acidophilus La-5 plus Bifidobacterium animalis subsp. lactis BB-12 in left-sided ulcerative colitis did not show a significant advantage over placebo, although the intervention was well tolerated [9]. This negative finding is important because it cautions against over-generalizing from species names alone.

More recent trials reinforced the relevance of formulation architecture and add-on context. In a multicenter placebo-controlled study of 133 patients with mild-to-moderate ulcerative colitis receiving 5-ASA, Escherichia coli Nissle 1917 significantly improved 4-week clinical response and 8-week endoscopic remission relative to placebo, despite a neutral primary quality-of-life endpoint [11]. A 2024 updated meta-analysis of 45 randomized trials further concluded that probiotics significantly increased the odds of clinical remission in ulcerative colitis and that multi-strain formulations outperformed comparators more consistently than single-strain approaches [12].

Table 2. Core outcome summary from the BALIMONT comparative system.

Group	Core composition	Live: postbiotic	Inflammatory-burden reduction	Tight-junction increase
Example 1	Three-strain live + homologous postbiotic	1:2	68.2%	42.6%
Example 2	Three-strain live + homologous postbiotic	1:2	65.7%	40.1%
Example 3	Three-strain live + homologous postbiotic	1:2	67.4%	41.8%
Comparator	Live bacteria only	None	32.5%	18.3%

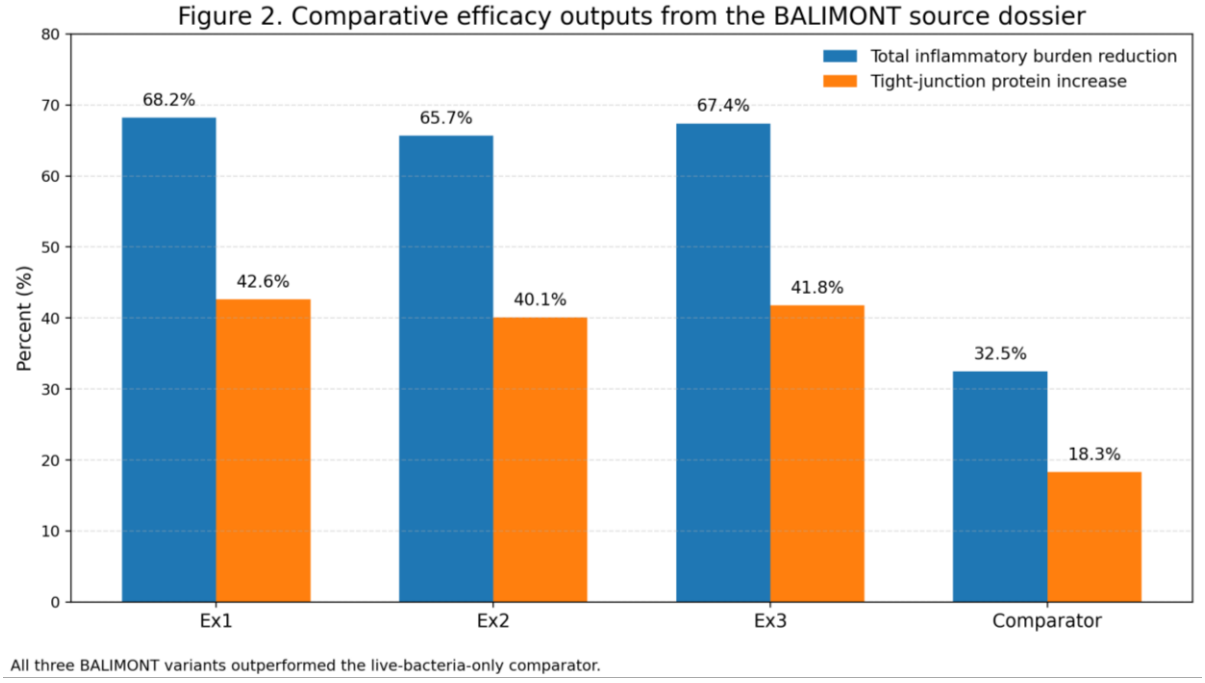

Figure 2. Comparative efficacy outputs extracted from the BALIMONT study records

Table 3. Published clinical trial evidence relevant to BALIMONT's immune–barrier positioning

Study	Patients / duration	Intervention	Key reported outcomes	Relevance to BALIMONT
Furrie 2005 [6]	Active UC; n=18; 4 weeks	B. longum + Synergy 1	Sigmoidoscopy improved; TNF-α and IL-1α decreased; epithelial inflammation reduced	Supports microbiota-directed reduction of mucosal inflammatory signaling
Cui 2004 [7]	UC after remission induction; n=30; 8 weeks	BIFICO vs placebo	Relapse 20% vs 93.3%; NF-κB, TNF-α, and IL-1β decreased; IL-10 increased	Aligns with BALIMONT cytokine-lowering and barrier-oriented rationale
Ishikawa 2011 [8]	Mild–moderate UC; n=41; 1 year	B. breve + GOS	Colonoscopic status and lavage MPO improved; fecal pH and Bacteroidaceae decreased	Shows long-duration synbiotic benefit in clinical disease control
Wildt 2011 [9]	UC in remission; n=32; 52 weeks	L. acidophilus La-5 + BB-12	No significant maintenance benefit vs placebo; intervention well tolerated	Demonstrates strain- and formulation-specific heterogeneity
Tamaki 2016 [10]	Active UC; n=56; 8 weeks	BB536 vs placebo	Clinical remission 63% vs 52%; significant within-group UCDAI decrease	Provides randomized human evidence for bifidobacterial efficacy in active UC
Park 2022 [11]	Mild–moderate UC on 5-ASA; n=133 randomized; 8 weeks	EcN add-on vs placebo	Clinical response at week 4: 39.7% vs 21.7%; endoscopic remission at week 8: 46.4% vs 27.1%	Supports probiotic add-on benefit in standard-of-care context

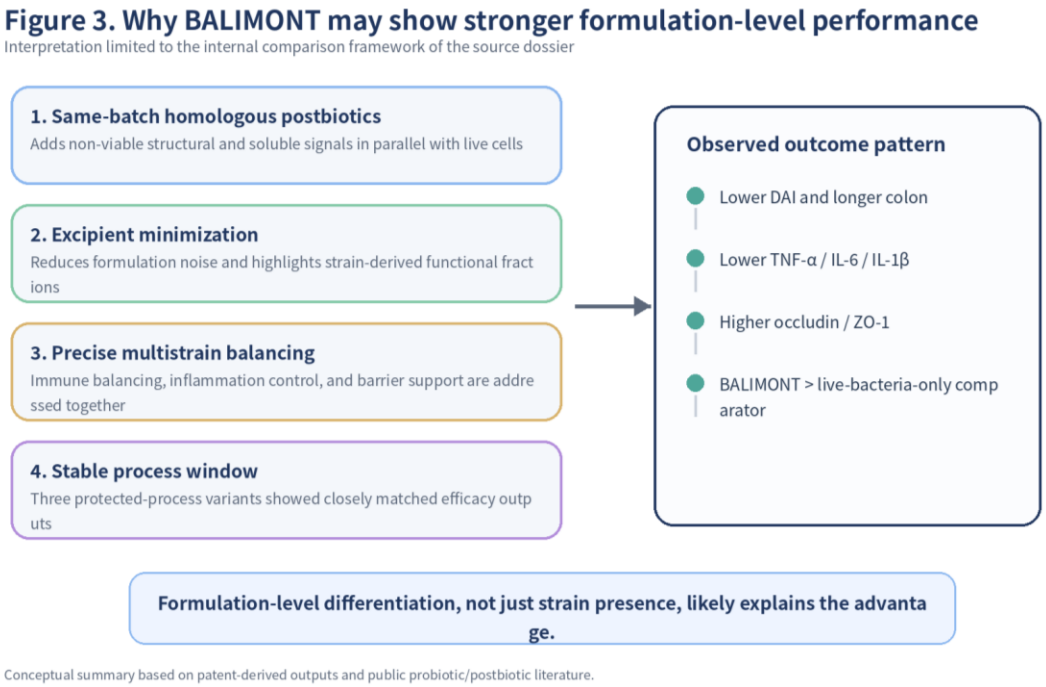

Figure 3. Formulation-level interpretation of BALIMONT differentiation

4. DISCUSSION

We interpret the BALIMONT advantage primarily as a formulation-level effect. The live–postbiotic pairing adds a rapid, non-viability-dependent signaling layer on top of the longer-duration ecological pressure created by viable cells. Within the retained dataset, this architecture was associated with approximately two-fold stronger inflammatory-burden reduction and more than two-fold stronger tight-junction improvement than the live-bacteria-only comparator.

This interpretation is compatible with current knowledge of probiotic and postbiotic biology. Mucosal immune conditioning depends on more than colonization. Structural components, cell-wall fragments, metabolites, and host–epithelium crosstalk can all influence epithelial APRIL-associated pathways, inflammatory tone, and barrier repair [1, 2]. Our retained BALIMONT data therefore fit a plausible mechanistic model in which live cells and homologous postbiotic fractions act on overlapping but non-identical timescales.

The published clinical trials we reviewed do not provide direct confirmation for the exact BALIMONT 2:3:5 live–postbiotic system. Nevertheless, they do provide relevant human anchors. Bifidobacterium-centered synbiotic and probiotic interventions have improved endoscopic and inflammatory outcomes in active ulcerative colitis [6,8,10]. Add-on probiotic therapy can improve response or endoscopic remission when used with 5-ASA in mild-to-moderate disease [11, 12]. At the same time, not every Lactobacillus/Bifidobacterium formulation is effective, as illustrated by the neutral La-5/BB-12 maintenance trial [9].

For that reason, we regard the present article as a translationally strengthened manuscript rather than definitive clinical proof. The retained murine data justify the barrier-centered and anti-inflammatory positioning of BALIMONT, and the reviewed human studies support the broader clinical plausibility of microbiota-directed therapy in ulcerative colitis. However, exact-strain and exact-formulation validation remains the next necessary step.

A well-designed human program for BALIMONT should therefore include clinical activity indices, fecal calprotectin, endoscopic healing, mucosal cytokine profiling, stool microbiome analysis, and barrier-linked biomarkers. The literature suggests that multi-strain systems and adjunctive use with standard therapy may be especially informative design choices [11, 12].

5. CONCLUSION

We conclude that the BALIMONT live–postbiotic multistrain platform demonstrates a consistent barrier-restorative and anti-inflammatory signal in the retained experimental-colitis dataset, with all three BALIMONT variants outperforming a live-bacteria-only comparator. When these retained results are viewed alongside published randomized and controlled ulcerative-colitis trials, the translational rationale becomes stronger: probiotic and synbiotic interventions can improve mucosal inflammatory activity, endoscopic outcomes, and remission-related measures in humans, but success depends heavily on strain balance, formulation design, and treatment context. BALIMONT should therefore be advanced as a differentiated immune-regulation and barrier-support candidate whose exact clinical value now requires prospective human confirmation.

REFERENCES

[1] Salminen S, Collado MC, Endo A, et al. The International Scientific Association of Probiotics and Prebiotics (ISAPP) consensus statement on the definition and scope of postbiotics. Nat Rev Gastroenterol Hepatol. 2021; 18(9):649-667. doi:10.1038/s41575-021-00440-6.

[2] Gutzeit C, Magri G, Cerutti A. Intestinal IgA production and its role in host-microbe interaction. Immunol Rev. 2014; 260(1):76-85. doi:10.1111/imr.12189.

[3] Hu T, Wang H, Xiang Y, et al. Preventive effect of Lactobacillus acidophilus XY27 on DSS-induced ulcerative colitis in mice. Drug Des Devel Ther. 2020; 14:5977-5991.

[4] Wang H, Sun Y, Ma X, et al. The Lactobacillus plantarum P-8 probiotic microcapsule prevents DSS-induced colitis through improving intestinal integrity and reducing colonic inflammation in mice. Nutrients. 2024; 16(7):1055. doi:10.3390/nu16071055.

[5] Li M, Ding J, Stanton C, et al. Bifidobacterium longum subsp. infantis FJSYZ1M3 ameliorates DSS-induced colitis by maintaining the intestinal barrier, regulating inflammatory cytokines, and modifying gut microbiota. Food Funct. 2023; 14(1):354-368. doi:10.1039/D2FO03263E.

[6] Furrie E, Macfarlane S, Kennedy A, et al. Synbiotic therapy (Bifidobacterium longum/Synergy 1) initiates resolution of inflammation in patients with active ulcerative colitis: a randomised controlled pilot trial. Gut. 2005; 54(2):242-249. doi:10.1136/gut.2004.044834.

[7] Cui HH, Chen CL, Wang JD, et al. Effects of probiotic on intestinal mucosa of patients with ulcerative colitis. World J Gastroenterol. 2004; 10(10):1521-1525. doi:10.3748/wjg.v10.i10.1521.

[8] Ishikawa H, Matsumoto S, Ohashi Y, et al. Beneficial effects of probiotic bifidobacterium and galacto-oligosaccharide in patients with ulcerative colitis: a randomized controlled study. Digestion. 2011; 84(2):128-133. doi:10.1159/000322977.

[9] Wildt S, Nordgaard I, Hansen U, Brockmann E, Rumessen JJ. A randomised double-blind placebo-controlled trial with Lactobacillus acidophilus La-5 and Bifidobacterium animalis subsp. lactis BB-12 for maintenance of remission in ulcerative colitis. J Crohns Colitis. 2011; 5(2):115-121. doi:10.1016/j.crohns.2010.11.004.

[10] Tamaki H, Nakase H, Inoue S, et al. Efficacy of probiotic treatment with Bifidobacterium longum 536 for induction of remission in active ulcerative colitis: a randomized, double-blinded, placebo-controlled multicenter trial. Dig Endosc. 2016; 28(1):67-74. doi:10.1111/den.12553.

[11] Park SK, Hong H, Oh K, et al. Additive effect of probiotics (Mutaflor) on 5-aminosalicylic acid therapy in patients with ulcerative colitis. Korean J Intern Med. 2022; 37(5):949-957. doi:10.3904/kjim.2021.458.

[12] Estevinho MM, González-Martínez C, María N, et al. Efficacy and safety of probiotics in IBD: An overview of systematic reviews and updated meta-analysis of randomized controlled trials. United European Gastroenterol J. 2024; 12(8):960-977. doi:10.1002/ueg2.12654.

[13] Huang C, Fan L, Hu B, et al. Probiotics for the treatment of ulcerative colitis: a review of experimental research from 2018 to 2022. Front Microbiol. 2023; 14:1211271. doi:10.3389/fmicb.2023.1211271.