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REVIEW ARTICLE

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Main clinical evidence on nutrients, lifestyle and gut microbiota in the treatment of inflammatory bowel diseases: a concise systematic review

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Abstract

Introduction: The main risk factor for inflammatory bowel diseases (IBD) is a positive family history. The pathogenesis of IBD is linked to genetically susceptible individuals, dysregulated intestinal microbiota (dysbiosis), chronic inflammation and poor dietary patterns. Diet plays an important role in modulating the gut microbiota. **Objective:** It was to carry out a systematic review on the main interactions between nutrition, lifestyle changes, intestinal microbiota and inflammatory bowel diseases, in order to highlight the main clinical outcomes. **Methods:** The present study followed the international systematic review model (PRISMA). This study was carried out from January to March 2025. It included randomized controlled, prospective and retrospective studies. Common descriptive statistical analysis was performed. The Chi-Square (X^2) and One-Way (ANOVA) tests were applied, adopting an α level lower than 0.05 ($p<0.05$) with statistical significance in the 95% confidence interval. The R-sq (X^2) value using the Chi-Square test was analyzed to discover the inaccuracy or heterogeneity of the analyses, adopting the codes of low association ≤ 25%, medium association 25% <X<50% and high association ≥ 50%. The Cohen test was performed to calculate the effect size (Effect Size) and the inverse of the standard error (precision or sample size) for the risk of bias (Funnel Plot). **Results:** A total of 207 were found, 17 clinical studies on the modulation of diet to control IBD were included in this study. These studies showed reductions in persistent intestinal symptoms, improvement in the intestinal microbiota, reduction in markers of inflammation and improvement in quality of life, with statistical significance ($p<0.05$) (95% CI). The studies were homogeneous (X^2 = 92.32%), which increases the reliability of clinical results on the importance of diet in modulating IBD. **Conclusion:** The important role of dietary modulation in controlling and even remitting IBD was evident.

Keywords: Nutrology. Inflammatory bowel disease. Diet therapy. Gut microbiota. Lifestyle.

Introduction

Inflammatory bowel diseases (IBD) have shown an increase in incidence worldwide **[1]**. A positive family history is noteworthy in 10-25% of patients. Crohn's disease (CD) can affect individuals of various ages and with a higher percentage in women **[2]**. Ulcerative colitis (UC) can start at any age and has a higher incidence between 20 and 40 years **[3]**.

The pathogenesis of IBD is that genetically susceptible individuals develop intolerance to the dysregulated gut microbiota (dysbiosis), and chronic inflammation develops as a result of poor dietary triggers **[4-6]**. Thus, diet plays an important role in modulating the gut microbiota and can be applied as a therapeutic tool to improve the course of the disease **[7]**. Therefore, current research in the field of IBD focuses largely on establishing the role of causal variants in gene expression **[8]**.

Despite this, genetic risk loci identified so far explain only a small part of the genetic variation in disease risk, and more factors need to be taken into account to understand this multifactorial pathology **[9]**. In this respect, diet plays a role in regulating intestinal inflammation by modifying and modulating the intestinal microbiota **[10,11]**. In this sense, the

evolution of epigenetics has provided new insights into the mechanisms by which environmental changes induce the expression of pathological genes and influence the cellular phenotype in the context of IBD.

The evolution and clinical manifestation of IBD are related to the interaction between genetic factors, with emphasis on mutations in the NOD2 gene (or CARD15), and also three other main mutations (R 702W, G908 R, and 1007 frameshift) have been described and linked to the disease phenotype, the intestinal microbiota and mucosal immunoregulation **[12-17]**. In this context, metabolism encompasses the interactions between diet, the microbiome, and cellular enzymatic processes that generate the chemical pathways necessary to maintain life. Endogenous metabolites as well as dietary nutrients can directly influence epigenetic enzymes. Epigenetic modifications in DNA and histone proteins alter cell fate by controlling chromatin accessibility and downstream gene expression patterns **[18]**.

In addition to the connection between metabolism and epigenetic pathways, nutrients can impact cellular status by modulating signaling pathway activity. One example is through the mechanistic target of rapamycin (mTOR) signaling pathway and, in particular, the mTOR complex 1 (mTORC1), which regulates cell growth only when nutrients and growth factors are present. Depletion of specific nutrients, including arginine, leucine, and S-adenosylmethionine, prevents growth factor-induced mTORC1 activation by blocking Rag GTPase-mediated mTORC1 recruitment to the lysosome, where it can be activated by Rheb GTPase **[18]**.

Another way that nutrients are detected to impact cellular status is through AMPactivated protein kinase (AMPK), which, at low levels of cellular ATP, phosphorylates substrates to restore the cell's energy balance and in the process, regulates cell growth and autophagy. In addition, transcription factors can be directly regulated by metabolites, such as tryptophan and kynurenine **[18]**. Dietary manipulations and metabolites can affect tissue stem cells and direct cell fate decisions, as highlighted in the small intestine by intestinal stem cells (ISCs). In this case, the enzyme 3-hydroxy-3-methylglutaryl-CoA synthase (Hmgcs2) is highly expressed. Also, sources of ketogenic or high-glucose diets regulate the balance of self-renewal by CTI **[18]**.

All these epigenetic and nutritional mechanisms are of paramount importance, as approximately 70.0 to 80.0% of patients lose weight with IBD, leading to some degree of nutritional impairment, and around 23.0% of outpatients and 85.0% of hospitalized patients with predominant malnutrition **[19,20]**. In this respect, diet also plays a decisive role in modulating the composition of the microbiome **[13]** and influences the inflammatory response **[17]**. Thus, a balanced diet with low fat and fiber content may be important in preventing dysbiosis and preserving the immune system **[21]**.

In this sense, gut microbiota is fundamental for the activation of the immune system, with emphasis on Lactobacillus acidophilus, Lactobacillus bulgaricus, and Lactobacillus casei, increasing IgA for the removal of antigens through a noninflammatory pathway and increasing T and B lymphocytes. Lactobacilli and Bifidobacteria inhibit the growth of exogenous and/or harmful bacteria, stimulate immune functions, aid in the digestion and/or absorption of food ingredients and minerals, and contribute to vitamin synthesis **[22-24]**.

Also, *Faecalibacterium prausnitzii* is one of the most prevalent intestinal bacterial species in healthy adults, being a producer of butyrate **[1]**. The reduction of this bacterium in the intestine may contribute to the appearance or worsening of IBD. Thus, to increase the numbers of this bacterium, it is necessary to eat foods rich in fiber, increase the consumption of fruits, vegetables, legumes, whole grains and cereals, seeds, and nuts **[1,4]**. Thus, short-chain fatty acids, such as butyrate, propionate, and acetate, serve as an energy source for intestinal epithelial cells and induce protective regulatory immune responses **[23]**.

The Gram-negative bacterium Bacteroides fragilis induces the differentiation of CD4+ T cells into Treg cells, leading to the production of anti-inflammatory cytokines, such as interleukin-10 (IL-10) and transforming growth factor beta (TGFβ), abolishing the pro-inflammatory response of Th17 cells **[8]**. The differentiation of Treg cells depends on the recognition by CD4+ T cells of the polysaccharide presented by CD4+ cells. In turn, segmented filamentous bacteria, after contact with antigen-presenting cells, have been shown to induce pro-inflammatory cells, such as Th17 cells **[8]**.

Therefore, the present study conducted a systematic review on the main interactions between nutrients, lifestyle changes, gut microbiota, and inflammatory bowel diseases, in order to highlight the main clinical outcomes.

Methods

Study Design

This study followed an international model for systematic review, adhering to the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines. Available at: http://www.prisma-statement.org/?AspxAutoDetectCookieSupport=1.

Accessed on: 02/11/2025. The methodological quality standards of AMSTAR-2 (Assessing the methodological quality of systematic reviews) were also followed. Available at: https://amstar.ca/. Accessed on: 03/15/2025.

Data Sources and Search Strategy

The literature search process was conducted from January to March 2025 and developed based on Scopus, PubMed, Lilacs, Ebsco, Scielo, and Google Scholar, covering scientific articles from various periods to the present day. The PICOS search strategy model was applied. The descriptors (DeCS/MeSH Terms) were used, employing the Boolean operator "and" between MeSH terms and "or" between historical discoveries, as shown in Table 1.

Table 1. Example of the search structure in PubMed; the same search strategy was used in the other databases.

PubMed	Inflammatory bowel disease **OR** Functional Nutrition **OR** Diet therapy **OR** Nutrological treatment **OR** Gut microbiota **OR** Quality of life
	AND
PubMed	Randomized controlled trial **OR** Prospective study **OR** Retrospective study **OR** Observational/Epidemiological studies
	NOT
PubMed	**OR** Case reports **OR** Editorials **OR** Letters to the editor **OR** Review study **OR** Meta-analysis

Source: Own authorship.

Study Quality and Risk of Bias

Quality was classified as high, moderate, low, or very low regarding risk of bias, clarity of comparisons, precision, and consistency of analyses. The most evident highlight was systematic review articles or meta-analyses of randomized clinical trials, followed by randomized clinical trials. Low quality of evidence was attributed to case reports, editorials, and brief communications, according to the GRADE instrument. The risk of bias was analyzed according to the Cochrane instrument through the analysis of the Funnel Plot (Sample size versus Effect size), using Cohen's d test.

Results and Discussion

Summary of Findings

After the detailed literature search process, the present study found seventeen (17) studies, being 11 randomized controlled clinical trials and 6 prospective studies in the last ten years, out of a total of 87 studies evaluated, showing a high quality of scientific evidence in the studies addressed, with evidence level IA, according to the GRADE criteria. Furthermore, the analyzed studies showed high homogeneity in the results (high association = >50%), presenting 92.32% in relation to the R-sq value (X^2).

Main Findings from Clinical Studies

Most of the studies listed in this study followed a randomized controlled design and were homogeneous in relation to symptom reduction, reduction of inflammatory bowel syndrome (IBS), improvement in quality of life (QoL), and improvement of the gut microbiota, highlighting the important influence of dietary interventions on inflammation and clinical outcomes of IBD (Table 2).

Table 2. Main outcomes of each listed study in relation to symptom reduction, reduction of inflammatory bowel syndrome (IBS), improvement in quality of life (QoL), and improvement of the gut microbiota. Most studies presented $p<0.001$ in relation to the control group of each study, that is, they presented a statistically significant difference.

Authors/ years	*Symptom reduction **	*Reduction of inflammatory bowel syndrome*	*QoL*	*Improvement of the Gut Microbiota*
Cox et al. 2020	1	1	1	1
Cox et al. 2017	1	1	1	**(-)
Pedersen et al. 2017	1	1	1	(-)
Bodini et al. 2019	1	1	1	(-)
Papada et al. 2019	0	0	0	(-)
Jian et al., 2018	1	1	1	(-)
Albenberg et al. 2019	1	1	1	(-)
Svolos et al.,2018	1	1	1	1
Levine et al. 2019	1	1	1	1
Racine et al. 2016	0	0	0	0
Braly et al., 2017	1	1	1	1
Machado et al., 2015	1	1	1	(-)
Brotherton et al., 2014	1	1	1	(-)
Sökülmez et al., 2014	1	1	1	(-)
Kyaw et al., 2014	1	1	1	(-)
Hanai et al., 2012	1	1	1	(-)
Kang et al., 2015	1	1	1	(-)

***** Code 1 means "yes" answer and code 0 means "no" answer in statistical terms. **(-) not reported in studies.

Only 4 studies presented the results of changes in the gut microbiota with dietary interventions (Table 2). There was a prevalence of symptom reduction (88.24%), reduction in IBS (88.24%), improvement in QoL (88.24%), and improvement in gut microbiota (80%), highlighting the important influence of dietary interventions on the clinical outcomes of IBD.

Using the Chi-Square test, it is possible to observe the differences between the observed and expected data, analyzing which variables presented the greatest differences, which may indicate dependence or association between them. In addition, the contributions to the Chi-Square statistic (Pearson and

Likelihood Ratio) can be compared to analyze which variables presented the highest values. As a result, there was a significant dependence between the variables Symptom Reduction (1) vs. QoL increase (1), with contributions to the Chi-Square of 14.336, and with Pearson's test equal to 17.000 and Likelihood Ratio equal to 12.315, with $p=0.019<0.05$.

Also, using the Chi-Square test, the correlation between the variables Reduction of Symptoms vs. Improvement of Gut Microbiota (Improvement_GM) was performed using the Chi-Square method (X^2). As a result, there was a significant dependence between the variables Reduction of Symptoms (1) vs. Improvement_GM (1), with contributions to the Chi-Square of 19.0627 and with Pearson's test equal to 21.000 and Likelihood Ratio equal to 18.415, with $p=0.011<0.05$.

Given these results, the present study found important randomized controlled clinical trials and other clinical studies in the last ten years that highlighted the important role of dietary modulation in the control of IBD **[25-41]**. In this sense, these studies showed important reductions in persistent intestinal symptoms, improvement in the intestinal microbiota, reduction of circulating markers of inflammation and improvement in quality of life.

Finally, many studies have evaluated the ability of diet to modulate the intestinal microbiota and influence epithelial barrier function. Low-fiber diets are associated with IBD, with a postulated mechanism of reduced production of short-chain fatty acids by commensal bacteria whose preferred energy source is fiber. Butyrate, a short-chain fatty acid, is essential for colon health and the main energy source for colonocytes **[42]**.

Conclusion

It was concluded that with nutritional treatment, several micronutrients have the potential to modulate intestinal inflammation. Immunonutrition has demonstrated its importance through vitamins A, C, E, and D, folic acid, beta-carotene, and trace elements such as zinc, selenium, manganese, and iron. Enteral nutrition in pediatric Crohn's disease appears to be the only nutritional intervention currently recommended as first-line therapy. Specific carbohydrate diets, fermentable oligosaccharides, disaccharides, monosaccharides, and polyol diets, and the Mediterranean diet also appear to show strong anti-inflammatory properties and promise to improve the symptoms of inflammatory bowel disease. Dietary modulation can control inflammatory bowel diseases by reducing persistent intestinal symptoms, balancing the gut microbiota, and reducing markers of inflammation. Dietary therapy can improve the quality of life of patients with inflammatory bowel disease.

CRediT

Author contributions: **Conceptualization**; **Data curation**; **Formal Analysis**; **Investigation; Methodology**; **Project administration; Supervision**; **Writing - original draft**; **Writing-review & editing**- Letícia Catini Trombeta and Kassielly Melissa Ribeiro Rodrigues.

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All referenced sources are accessible through the respective journals or public repositories.

Conflict of Interest

The authors declare no conflict of interest.

Similarity Check

It was applied by Ithenticate®.

Application of Artificial Intelligence (AI)

Not applicable.

Peer Review Process

It was performed.

References

1. Gu JM, Zhao M, Zhu J, Tao HW, Shao XP, Qin LQ, Ge YY, Chen GC. Dietary inflammatory potential, genetic predisposition, and incidence of Crohn's disease and ulcerative colitis. Nutr Metab (Lond). 2025 May 1;22(1):35. doi:

10.1186/s12986-025-00934-z. .

2. Chen J, Dan L, Yuan S, Fu T, Sun J, Wolk A, Ludvigsson JF, Li X, Wang X, Larsson SC. Dietary Antioxidant Capacity, Genetic Susceptibility and Polymorphism, and Inflammatory Bowel Disease Risk in a Prospective Cohort. Clin Gastroenterol Hepatol. 2025 Aug;23(9):1623-1632.e16. doi: 10.1016/j.cgh.2024.09.033.
3. Barberio B, Bertin L, Facchin S, Bonazzi E, Cusano S, Romanelli G, Pesenti FF, Cazzaniga E, Palestini P, Zingone F, Savarino EV. Dietary Interventions and Oral Nutritional Supplementation in Inflammatory Bowel Disease: Current Evidence and Future Directions. Nutrients. 2025 May 30;17(11):1879. doi: 10.3390/nu17111879.
4. Scaldaferri F, Correale C, Gasbarrini A, Danese S. Mucosal biomarkers in inflammatory bowel disease: Key pathogenic players or disease predictors? World J Gastroenterol. 2010 June 7; 16(21): 2616–2625.
5. Côté-Daigneault J, Bouin M, Lahaie R, Colombel JF, Poitras P. Biologics in inflammatory bowel disease: what are the data? United European Gastroenterol J. 2015;3(5):419-28.
6. Roda G, Chien Ng S, Kotze PG, et al. Crohn's disease [published correction appears in Nat Rev Dis Primers. 2020 Apr 6;6(1):26] [published correction appears in Nat Rev Dis Primers. 2020 May 20;6(1):42] [published correction appears in Nat Rev Dis Primers. 2020 Jun 19;6(1):51]. Nat Rev Dis Primers. 2020;6(1):22. Published 2020 Apr 2. doi:10.1038/s41572-020-0156-2
7. Khanna S, Raffals LE. The Microbiome in Crohn's Disease: Role in Pathogenesis and Role of Microbiome Replacement Therapies. Gastroenterol Clin North Am. 2017 Sep;46(3):481-492. doi: 10.1016/j.gtc.2017.05.004. Epub 2017 Jul 19.
8. He Q, Gao Y, Jie Z, Yu X, Laursen JM, Xiao L, Li Y, Li L, Zhang F, Feng Q, Li X, Yu J, Liu C, Lan P, Yan T, Liu X, Xu X, Yang H, Wang J, Madsen L, Brix S, Wang J, Kristiansen K, Jia H. Two distinct metacommunities characterize the gut microbiota in Crohn's disease patients. Gigascience. 2017 Jul 1;6(7):1-11. doi: 10.1093/gigascience/gix050.
9. Green N, Miller T, Suskind D, Lee D. A Review of Dietary Therapy for IBD and a Vision for the Future. Nutrients. 2019 Apr 26;11(5). pii: E947. doi: 10.3390/nu11050947.
10. Benjamin JL, Hedin CR, Koutsoumpas A, Ng SC, McCarthy NE, Hart AL, et al. Randomised, double-blind, placebo-controlled trial of fructo-oligosaccharides in active Crohn's disease. Gut 2011;60:923-9.
11. Bernstein CN, Loftus EV Jr, Ng SC, Lakatos PL, Moum B; Epidemiology and Natural History Task Force of the International Organization for the Study of Inflammatory Bowel Disease (IOIBD). Hospitalisations and surgery in Crohn's disease. Gut 2012;61:622-9.
12. Hanauer SB, Sandborn W; Practice Parameters Committee of the American College of Gastroenterology. Management of Crohn's disease in adults. Am J Gastroenterol 2001;96:635-43.
13. Hedin C, Whelan K, Lindsay JO. Evidence for the use of probiotics and prebiotics in inflammatory bowel disease: a review of clinical trials. Proc Nutr Soc 2007;66:307-15.
14. Kirschner BS. Differences in the management of inflammatory bowel disease in children and adolescents compared to adults. Neth J Med 1998;53:S13-8.
15. Landy J, Al-Hassi HO, McLaughlin SD, et al. Review article: faecal transplantation therapy for gastrointestinal disease. Aliment Pharmacol Ther 2011;34:409-15.
16. Langholz E, Munkholm P, Krasilnikoff PA, Binder V. Inflammatory bowel diseases with onset in childhood: clinical features, morbidity, and mortality in a regional cohort. Scand J Gastroenterol 1997;32:139-47.
17. Meijer BJ, Dieleman LA. Probiotics in the treatment of human inflammatory bowel diseases: update 2011. J Clin Gastroenterol 2011;45:S139-44.
18. Shapira SN, Christofk HR. Metabolic Regulation of Tissue Stem Cells. Trends Cell Biol. 2020 Jul;30(7):566-576. doi: 10.1016/j.tcb.2020.04.004. Epub 2020 Apr 28. PMID: 32359707).
19. Basson A. Vitamin D. Crohn's disease in the adult patient: a review. J Parenter Enteral Nutr. 2014;38:438–58.
20. Roth MP, Petersen GM, McElree C, Vadheim CM, Panish JF, Rotter JI. Familial empiric risk estimates of inflammatory bowel disease in Ashkenazi Jews. Gastroenterology. 1989;96(4):1016-20.
21. Lee YK, Menezes JS, Umesaki Y, Mazmanian SK. Proinflammatory T-cell responses to gut microbiota promote experimental autoimmune encephalomyelitis. Proc Natl Acad Sci USA. 2011;108(suppl 1):4615-4622.
22. Xu XR, Liu CQ, Feng BS. Dysregulation of mucosal immune response in pathogenesis of

inflammatory bowel disease. World J Gastroenterol. 2014;20:3255–64.

23. Adamiak T, Walkiewicz-Jedrzejczak D, Fish D, Brown C, Tung J, Khan K, et al. Incidence, clinical characteristics, and natural history of pediatric IBD in Wisconsin: a population-based epidemiological study. Inflamm Bowel Dis. 2013;19(6):1218-23.
24. Den Besten G, Bleeker A, Gerding A, et al. Short-chain fatty acids protect against high-fat diet-induced obesity via a PPARγ-dependent switch from lipogenesis to fat oxidation. Diabetes. 2015;64(7):2398-2408.
25. Cox SR, Lindsay JO, Fromentin S, Stagg AJ, McCarthy NE, Galleron N, Ibraim SB, Roume H, Levenez F, Pons N, Maziers N, Lomer MC, Ehrlich SD, Irving PM, Whelan K. Effects of Low FODMAP Diet on Symptoms, Fecal Microbiome, and Markers of Inflammation in Patients With Quiescent Inflammatory Bowel Disease in a Randomized Trial. Gastroenterology. 2020 Jan;158(1):176-188.e7. doi: 10.1053/j.gastro.2019.09.024. Epub 2019 Oct 2. PMID: 31586453.
26. Cox SR, Prince AC, Myers CE, Irving PM, Lindsay JO, Lomer MC, Whelan K. Fermentable Carbohydrates [FODMAPs] Exacerbate Functional Gastrointestinal Symptoms in Patients With Inflammatory Bowel Disease: A Randomised, Double-blind, Placebo-controlled, Cross-over, Re-challenge Trial. J Crohns Colitis. 2017 Dec 4;11(12):1420-1429. doi: 10.1093/ecco-jcc/jjx073. PMID: 28525543.
27. Pedersen N, Ankersen DV, Felding M, Wachmann H, Végh Z, Molzen L, Burisch J, Andersen JR, Munkholm P. Low-FODMAP diet reduces irritable bowel symptoms in patients with inflammatory bowel disease. World J Gastroenterol. 2017 May 14;23(18):3356-3366. doi: 10.3748/wjg.v23.i18.3356. PMID: 28566897; PMCID: PMC5434443.
28. Bodini G, Zanella C, Crespi M, Lo Pumo S, Demarzo MG, Savarino E, Savarino V, Giannini EG. A randomized, 6-wk trial of a low FODMAP diet in patients with inflammatory bowel disease. Nutrition. 2019 Nov-Dec;67-68:110542. doi: 10.1016/j.nut.2019.06.023. Epub 2019 Jul 1. PMID: 31470260.
29. Papada E, Amerikanou C, Torović L, Kalogeropoulos N, Tzavara C, Forbes A, Kaliora AC. Plasma free amino acid profile in quiescent Inflammatory Bowel Disease patients orally administered with Mastiha (Pistacia lentiscus); a randomised clinical trial. Phytomedicine. 2019 Mar 15;56:40-47. doi: 10.1016/j.phymed.2018.08.008. Epub 2018 Aug 13. PMID: 30668352.
30. Jian L, Anqi H, Gang L, Litian W, Yanyan X, Mengdi W, Tong L. Food Exclusion Based on IgG Antibodies Alleviates Symptoms in Ulcerative Colitis: A Prospective Study. Inflamm Bowel Dis. 2018 Aug 16;24(9):1918-1925. doi: 10.1093/ibd/izy110. PMID: 29788288.
31. Albenberg L, Brensinger CM, Wu Q, Gilroy E, Kappelman MD, Sandler RS, Lewis JD. A Diet Low in Red and Processed Meat Does Not Reduce Rate of Crohn's Disease Flares. Gastroenterology. 2019 Jul;157(1):128-136.e5. doi: 10.1053/j.gastro.2019.03.015. Epub 2019 Mar 11. PMID: 30872105; PMCID: PMC6726378.
32. Svolos V, Hansen R, Nichols B, Quince C, Ijaz UZ, Papadopoulou RT, Edwards CA, Watson D, Alghamdi A, Brejnrod A, Ansalone C, Duncan H, Gervais L, Tayler R, Salmond J, Bolognini D, Klopfleisch R, Gaya DR, Milling S, Russell RK, Gerasimidis K. Treatment of Active Crohn's Disease With an Ordinary Food-based Diet That Replicates Exclusive Enteral Nutrition. Gastroenterology. 2019 Apr;156(5):1354-1367.e6. doi: 10.1053/j.gastro.2018.12.002. Epub 2018 Dec 11. PMID: 30550821.
33. Levine A, Wine E, Assa A, Sigall Boneh R, Shaoul R, Kori M, Cohen S, Peleg S, Shamaly H, On A, Millman P, Abramas L, Ziv-Baran T, Grant S, Abitbol G, Dunn KA, Bielawski JP, Van Limbergen J. Crohn's Disease Exclusion Diet Plus Partial Enteral Nutrition Induces Sustained Remission in a Randomized Controlled Trial. Gastroenterology. 2019 Aug;157(2):440-450.e8. doi: 10.1053/j.gastro.2019.04.021. Epub 2019 Jun 4. PMID: 31170412.
34. Racine A, Carbonnel F, Chan SS, Hart AR, Bueno-de-Mesquita HB, Oldenburg B, van Schaik FD, Tjønneland A, Olsen A, Dahm CC, Key T, Luben R, Khaw KT, Riboli E, Grip O, Lindgren S, Hallmans G, Karling P, Clavel-Chapelon F, Bergman MM, Boeing H, Kaaks R, Katzke VA, Palli D, Masala G, Jantchou P, Boutron-Ruault MC. Dietary Patterns and Risk of Inflammatory Bowel Disease in Europe: Results from the EPIC Study. Inflamm Bowel Dis. 2016 Feb;22(2):345-54. doi: 10.1097/MIB.0000000000000638. PMID: 26717318.
35. Braly K, Williamson N, Shaffer ML, Lee D, Wahbeh G, Klein J, Giefer M, Suskind DL. Nutritional Adequacy of the Specific Carbohydrate Diet in Pediatric Inflammatory Bowel Disease. J Pediatr Gastroenterol Nutr.

2017 Nov;65(5):533-538. doi: 10.1097/MPG.0000000000001613. PMID: 28825603; PMCID: PMC5653423.

36. Machado JF, Oya V, Coy CS, Morcillo AM, Severino SD, Wu C, Sgarbieri VC, Vilela MM. Whey and soy protein supplements changes body composition in patients with Crohn's disease undergoing azathioprine and anti-TNF-alpha therapy. Nutr Hosp. 2015 Apr 1;31(4):1603-10. doi: 10.3305/nh.2015.31.4.8362. PMID: 25795947.

37. Brotherton CS, Taylor AG, Bourguignon C, Anderson JG. A high-fiber diet may improve bowel function and health-related quality of life in patients with Crohn disease. Gastroenterol Nurs. 2014 May-Jun;37(3):206-16. doi: 10.1097/SGA.0000000000000047. PMID: 24871666; PMCID: PMC4260718.

38. Sökülmez P, Demirbağ AE, Arslan P, Dişibeyaz S. Effects of enteral nutritional support on malnourished patients with inflammatory bowel disease by subjective global assessment. Turk J Gastroenterol. 2014 Oct;25(5):493-507. doi: 10.5152/tjg.2014.4955. PMID: 25417609.

39. Kyaw MH, Moshkovska T, Mayberry J. A prospective, randomized, controlled, exploratory study of comprehensive dietary advice in ulcerative colitis: impact on disease activity and quality of life. Eur J Gastroenterol Hepatol. 2014 Aug;26(8):910-7. doi: 10.1097/MEG.0000000000000127. PMID: 24942954.

40. Hanai H, Iida T, Takeuchi K, Arai H, Arai O, Abe J, Tanaka T, Maruyama Y, Ikeya K, Sugimoto K, Nakamura T, Nakamura K, Watanabe F. Nutritional therapy versus 6-mercaptopurine as maintenance therapy in patients with Crohn's disease. Dig Liver Dis. 2012 Aug;44(8):649-54. doi: 10.1016/j.dld.2012.03.007.

41. Kang Y, Kim S, Kim SY, Koh H. Effect of short-term partial enteral nutrition on the treatment of younger patients with severe Crohn's disease. Gut Liver. 2015 Jan;9(1):87-93. doi: 10.5009/gnl13345.

42. Green N, Miller T, Suskind D, Lee D. A Review of Dietary Therapy for IBD and a Vision for the Future. Nutrients. 2019 Apr 26;11(5):947. doi: 10.3390/nu11050947.