International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Scholarly Publisher
RS Global Sp. z O.O.
ISNI: 0000 0004 8495 2390

Dolna 17, Warsaw,
Poland 00-773
+48 226 0 227 03
editorial_office@rsglobal.pl

ARTICLE TITLE	FROM LOW-FIBER TO NO-FIBER: A SYSTEMATIC REVIEW COMPARING THE GUT MICROBIOME AND INFLAMMATORY IMPACTS OF KETOGENIC VS. CARNIVORE DIETS
DOI	https://doi.org/10.31435/ijitss.4(48).2025.4235
RECEIVED	22 October 2025
ACCEPTED	23 December 2025
PUBLISHED	30 December 2025
LICENSE	The article is licensed under a **Creative Commons Attribution 4.0 International License.**

FROM LOW-FIBER TO NO-FIBER: A SYSTEMATIC REVIEW COMPARING THE GUT MICROBIOME AND INFLAMMATORY IMPACTS OF KETOGENIC VS. CARNIVORE DIETS

Karol Demel *(Corresponding Author, Email: karoldemel2908@gmail.com)*
University Clinical Hospital in Katowice, Katowice, Poland
ORCID ID: 0009-0005-5485-7987

Filip Kowal
Medical University of Silesia, Katowice, Poland
ORCID ID: 0009-0004-2775-4264

Justyna Talaska
SP ZOZ MSWiA in Katowice, Katowice, Poland
ORCID ID: 0009-0005-4723-9777

Adrian Dyląg
District Railway Hospital in Katowice, Katowice, Poland
ORCID ID: 0009-0000-1599-0383

Jakub Król
Provincial Specialist Hospital No. 4 in Bytom, Bytom, Poland
ORCID ID: 0009-0006-0696-1904

Maciej Łydka
LUX MED St. Elizabeth Hospital, Warsaw, Poland
ORCID ID: 0000-0002-3024-2524

Justyna Lewandowska
Warsaw Southern Hospital, Warsaw, Poland
ORCID ID: 0000-0001-7570-3374

Monika Dziedzic
Complex of Municipal Hospitals in Chorzów, Chorzów, Poland
ORCID ID: 0009-0002-4482-0898

Julia Holzer
Medical University of Silesia, Katowice, Poland
ORCID ID: 0009-0002-9827-1820

Natalia Libudzic
Maritime Hospital of the Polish Red Cross in Gdynia, Gdynia, Poland
ORCID ID: 0009-0007-8712-0839

ABSTRACT

Background: The ketogenic diet (KD) and the emerging carnivore diet (CD) represent two extremes of carbohydrate restriction, with distinct implications for gut microbiome ecology and inflammation. Although both regimens minimize dietary fiber intake, their comparative effects on microbial composition and immune modulation remain unclear.
Objectives: This systematic review aimed to synthesize current evidence on the impact of ketogenic and carnivore diets on gut microbiota structure, microbial metabolites, and inflammatory outcomes in human and animal studies.
Methods: A comprehensive search of PubMed, Scopus, and Web of Science databases was conducted according to PRISMA guidelines, including studies published between 2014 and 2025. Eligible studies evaluated microbiome composition or inflammatory biomarkers following adherence to KD or CD. Data were extracted and compared narratively due to heterogeneity in study designs.
Results: Across reviewed studies, ketogenic diets consistently reduced saccharolytic taxa (e.g., Bifidobacterium, Roseburia) and short-chain fatty acid production, yet in some cases improved systemic inflammation through β-hydroxybutyrate–mediated mechanisms. In contrast, carnivore diets led to further reductions in microbial diversity, enrichment of bile-tolerant taxa, and elevated proteolytic fermentation byproducts linked to intestinal barrier dysfunction. Evidence for long-term adaptation or recovery remains limited.
Conclusions: Both KD and CD reshape the gut ecosystem toward reduced fermentation capacity and altered inflammatory signaling. While ketogenic interventions may confer transient metabolic benefits, the carnivore diet represents an extreme model of fiber deprivation with uncertain safety. Controlled longitudinal trials are urgently needed to define the long-term microbiome and immune consequences of these ultra-low-fiber regimens.

KEYWORDS

Ketogenic Diet, Carnivore Diet, Gut Microbiome; Dietary Fiber, Inflammation, Short-chain Fatty Acids

CITATION

Karol Demel, Filip Kowal, Justyna Talaska, Adrian Dyląg, Jakub Król, Maciej Łydka, Justyna Lewandowska, Monika Dziedzic, Julia Holzer, Natalia Libudzic. (2025). From Low-Fiber to No-Fiber: A Systematic Review Comparing the Gut Microbiome and Inflammatory Impacts of Ketogenic vs. Carnivore Diets. *International Journal of Innovative Technologies in Social Science*. 4(48). doi: 10.31435/ijitss.4(48).2025.4235

Introduction

Low-carbohydrate diets that minimize or eliminate plant-derived foods have gained increasing popularity for weight management and metabolic health. Two such dietary approaches — the ketogenic diet (KD), which restricts carbohydrates while allowing limited plant fiber, and the carnivore diet (CD), which excludes plants entirely — represent distinct ends of a fiber continuum with potentially divergent effects on the gut microbiome and host inflammation [1,2].

Dietary fiber serves as a key substrate for colonic microbial fermentation, producing short-chain fatty acids (SCFAs) such as butyrate that maintain epithelial integrity and exert anti-inflammatory effects [3]. Conversely, low-fiber or fiber-free diets have been associated with decreased microbial diversity, reduced SCFA production, and shifts toward bile-tolerant, proteolytic taxa that may increase intestinal permeability and systemic inflammation [3,4].

Emerging data suggest that the KD modulates the microbiome by reducing saccharolytic taxa while increasing ketone bodies that may mimic some butyrate functions [1,5]. Evidence regarding the CD remains scarce; early observational data highlight its complete lack of fermentable substrates and uncertain long-term consequences for microbial and immune homeostasis [2,6].

Given the central role of dietary fiber in shaping the gut microbiota and regulating inflammation, a systematic synthesis comparing KD and CD is warranted. This review aims to evaluate available evidence on how these contrasting low- and no-fiber dietary patterns alter microbiome composition, microbial metabolites, and inflammatory biomarkers, and to identify research gaps relevant to metabolic and immune health.

Methodology

This systematic review followed the PRISMA 2020 guidelines [7]. A comprehensive search was conducted in PubMed, Scopus, and Web of Science for studies published between 2014 and October 2025 using the terms *"ketogenic diet"*, *"carnivore diet"*, *"gut microbiome"*, *"microbiota"*, and *"inflammation"*. Eligible studies included human or translational animal research assessing ketogenic or carnivore diets with reported outcomes on gut microbiome composition, microbial metabolites, or inflammatory markers. Reviews, abstracts, and studies without defined dietary protocols were excluded. Two reviewers independently screened and extracted data on study design, population, dietary intervention, and outcomes. Risk of bias was assessed using the Joanna Briggs Institute (JBI) tools, and results were synthesized qualitatively due to heterogeneity in study designs and analytical methods.

Results

1. Ketogenic Diet (KD) – Microbiome and Inflammatory Effects

Across the reviewed studies, ketogenic diets consistently demonstrated profound and reproducible modifications in gut microbial composition, metabolic activity, and inflammatory signaling. In most clinical and experimental interventions, the marked restriction of fermentable carbohydrates led to a quantifiable decline in saccharolytic taxa such as *Bifidobacterium*, *Roseburia*, and *Eubacterium rectale*, which are key producers of short-chain fatty acids (SCFAs) — particularly butyrate [1, 2]. This reduction was typically accompanied by lower fecal concentrations of SCFAs, altered acetate-to-butyrate ratios, and an overall decrease in microbial richness and diversity. Concurrently, there was a proportional enrichment of microbial taxa capable of metabolizing amino acids, fatty acids, and ketone intermediates, reflecting a shift from carbohydrate-driven to lipid- and protein-driven microbial metabolism.

Interestingly, this compositional simplification did not always correlate with worsened systemic inflammation. In several controlled studies, participants following a ketogenic diet exhibited reductions in circulating pro-inflammatory cytokines such as TNF-α, IL-6, and CRP, despite a concurrent decline in microbial diversity. This paradox has been attributed to the signaling role of β-hydroxybutyrate (BHB), a principal ketone body with recognized anti-inflammatory properties [8]. BHB can act as a histone deacetylase (HDAC) inhibitor, promoting the expression of anti-inflammatory genes and suppressing the NLRP3 inflammasome — a key mediator of innate immune activation. Such effects may, at least transiently, compensate for the loss of butyrate, which normally fulfills similar immunomodulatory functions within the colon.

Animal and human data further suggest that ketogenic diets may modify intestinal barrier physiology. A reduction in dietary fiber often predisposes to mucosal thinning and diminished mucus secretion, compromising epithelial integrity [4]. However, BHB and other ketone metabolites appear to enhance mitochondrial function and oxidative balance in intestinal epithelial cells, possibly offsetting some of the structural losses associated with low-fiber intake. These compensatory pathways might explain why ketogenic interventions, though microbiologically restrictive, have demonstrated beneficial effects in certain inflammatory and metabolic contexts such as epilepsy, obesity, and insulin resistance. Nonetheless, the long-term sustainability of these adaptations remains uncertain. Extended periods of carbohydrate deprivation may progressively deplete fiber-dependent microbial taxa, limiting the capacity for SCFA production and potentially increasing susceptibility to inflammatory and metabolic dysregulation once dietary diversity is restored.

2. Carnivore Diet (CD) – Evidence and Emerging Insights

The current evidence base regarding strictly carnivorous eating patterns remains limited but increasingly scrutinized. A recent exploratory investigation of individuals consuming exclusively animal-based foods revealed no gross reduction in microbial diversity relative to omnivorous controls; however, metagenomic analysis demonstrated a pronounced underrepresentation of genes associated with carbohydrate metabolism, fiber fermentation, and SCFA biosynthesis [2]. These data suggest a microbiome that is compositionally stable but functionally constrained, relying heavily on proteolytic and bile-acid–mediated energy pathways.

Nutritional assessments of carnivore diets consistently confirm an absence of microbiota-accessible carbohydrates (MACs), minimal intake of dietary fiber, and potential insufficiencies in micronutrients such as folate, magnesium, and vitamin C [9]. From a microbial perspective, this environment selects for bile-tolerant taxa (e.g., *Bilophila wadsworthia*, *Alistipes*) and proteolytic species (*Clostridium*, *Bacteroides*), promoting metabolic end-products such as branched-chain fatty acids, phenols, indoles, and secondary bile acids [10, 11]. Accumulation of these metabolites has been associated with impaired mucosal homeostasis, oxidative stress, and the activation of pro-inflammatory signaling pathways within the intestinal epithelium.

Furthermore, the absence of fermentable substrates restricts the ecological niches available for commensal SCFA-producing bacteria, reducing colonocyte access to butyrate — their primary energy source. Over time, this imbalance could facilitate epithelial atrophy and enhance gut permeability, creating a low-grade inflammatory environment. While anecdotal reports and short-term case studies describe improved subjective well-being and metabolic stability on carnivore diets, the mechanistic basis for such effects is poorly defined and likely context-dependent. Existing studies are constrained by small sample sizes, self-selection bias, and limited follow-up duration, leaving substantial uncertainty regarding long-term outcomes.

Collectively, current evidence suggests that ketogenic diets reshape the gut ecosystem by selectively reducing fermentative activity while maintaining limited anti-inflammatory balance through ketone-mediated pathways. In contrast, the carnivore diet represents a more extreme and potentially maladaptive deprivation of microbiota-accessible nutrients, characterized by metabolic rigidity and increased exposure to proteolytic byproducts. Although both regimens share mechanistic overlap in reducing carbohydrate availability, the degree of microbial simplification and potential inflammatory risk appear significantly greater in the carnivore paradigm. Robust, controlled human trials are urgently needed to determine the long-term implications of such ultra-low-fiber eating patterns on gut microbiome resilience, immune modulation, and systemic health.

Discussion

The synthesis of current evidence suggests that both ketogenic and carnivore diets, despite their shared carbohydrate restriction, induce distinct yet overlapping trajectories of microbial and inflammatory adaptation. The ketogenic diet (KD) fosters a selective ecological niche in which lipid- and amino acid–metabolizing taxa prevail, while saccharolytic species decline in relative abundance [12]. This compositional simplification often leads to diminished short-chain fatty acid (SCFA) output—particularly butyrate and propionate—but may coincide with improved systemic inflammatory profiles through ketone-mediated mechanisms. β-hydroxybutyrate, the principal circulating ketone body, has been shown to inhibit the NLRP3 inflammasome and modulate oxidative stress, providing a biochemical rationale for these paradoxical benefits in inflammatory regulation despite microbial contraction [13].

However, these metabolic advantages appear context-dependent. In neurological or metabolic disorders, where ketone utilization confers clinical benefit, transient reductions in microbial diversity may represent an acceptable trade-off. Yet in otherwise healthy individuals, prolonged suppression of SCFA-producing taxa could impair epithelial energy supply, intestinal barrier integrity, and mucosal immune regulation [14]. Additionally, KD-associated increases in bile acid flux and lipid oxidation intermediates might contribute to oxidative stress and mild endotoxemia over extended durations, suggesting a potential ceiling to its metabolic safety when applied chronically [15].

The carnivore diet (CD), by contrast, eliminates virtually all microbiota-accessible carbohydrates, representing an extreme of ecological deprivation. This forces microbial metabolism toward proteolysis and amino acid fermentation, generating byproducts such as ammonia, indoles, and secondary bile acids that are linked to intestinal barrier dysfunction and low-grade inflammation [16, 17]. Emerging data indicate that such metabolic rerouting diminishes beneficial taxa like *Faecalibacterium prausnitzii* while enriching bile-tolerant organisms such as *Bilophila wadsworthia* and *Alistipes spp.*, taxa often associated with inflammatory phenotypes [18]. Over time, these shifts may predispose individuals to dysbiosis-related pathologies, including increased gut permeability, immune dysregulation, and altered hepatic metabolism.

An important dimension of this discussion concerns the adaptability and resilience of the gut microbiota under extreme dietary constraints. Some findings suggest partial reversibility of microbial diversity following reintroduction of fermentable substrates, highlighting the gut ecosystem's dynamic recovery potential [19]. Nevertheless, repeated or chronic fiber deprivation, as observed in long-term low-carbohydrate adherence, may lead to irreversible depletion of keystone taxa crucial for mucin preservation and immune signaling [20]. Furthermore, inter-individual variability—shaped by genetics, baseline microbiota, and prior diet—likely mediates the extent of both microbial loss and host inflammatory response, complicating generalizations across populations.

Clinically, these findings underscore the need for nuanced application. While the ketogenic diet maintains therapeutic value in epilepsy, insulin resistance, and neurodegenerative disorders, extending carbohydrate restriction to the carnivore extreme lacks robust empirical validation and may incur cumulative harm to gut and systemic homeostasis. Future investigations should employ integrated multi-omics methodologies—including metagenomics, metabolomics, and immunophenotyping—to delineate adaptive versus maladaptive responses to sustained ultra-low-fiber diets. Longitudinal human trials are particularly warranted to determine whether the transient anti-inflammatory effects of ketone signaling can offset the long-term ecological and immunological costs of chronic fiber exclusion.

Conclusions

This systematic review demonstrates that both ketogenic and carnivore diets exert profound but distinct influences on the gut microbiome and host inflammatory status. The ketogenic diet, though low in carbohydrates, appears to preserve a degree of microbial diversity through residual plant components and ketone-driven anti-inflammatory pathways. In contrast, the carnivore diet represents an extreme form of carbohydrate and fiber exclusion, resulting in a microbiota functionally oriented toward protein and bile acid metabolism. Such a shift is consistently associated with the production of potentially harmful metabolites, including ammonia, p-cresol, and secondary bile acids, which may contribute to mucosal inflammation and barrier dysfunction.

Collectively, these findings support the notion that dietary fiber is a non-redundant component of gut and systemic health, and its absence cannot be fully compensated by metabolic adaptations to fat or protein-based energy sources. While short-term adherence to ketogenic diets may yield therapeutic benefits—particularly for neurological and metabolic disorders—sustained fiber deprivation may lead to long-term dysbiosis, impaired immune regulation, and heightened inflammatory tone. The carnivore diet, in its complete exclusion of plant-derived nutrients, lacks robust scientific validation and should therefore be approached with clinical caution.

From an ecological perspective, the gut microbiome appears resilient but not infinitely adaptable; its structural and functional diversity depends on consistent exposure to complex carbohydrates. The progressive loss of microbial species observed in ultra-low-fiber diets could represent a reversible yet clinically relevant form of "diet-induced microbiome extinction." Emerging evidence also suggests that such alterations may influence not only gastrointestinal but also metabolic, cardiovascular, and neuroimmune functions.

Future research should move beyond descriptive microbial analyses toward integrated models combining metagenomics, metabolomics, and immunophenotyping to delineate causality between specific microbial pathways and inflammatory responses. Randomized, long-term human trials comparing ketogenic and carnivore diets are urgently needed to establish safe thresholds of carbohydrate restriction and to identify compensatory strategies—such as periodic fiber reintroduction or postbiotic supplementation—that might mitigate adverse effects.

In conclusion, transitioning from a low-fiber to a no-fiber dietary pattern appears to amplify the disruption of gut microbial ecology and immune balance. Although both ketogenic and carnivore diets challenge traditional nutritional paradigms, only the former currently offers an evidence-based framework for clinical application. The total elimination of fermentable substrates, as observed in carnivore regimens, may ultimately compromise gut health, underscoring the irreplaceable role of dietary fiber in maintaining symbiosis between diet, microbiota, and host physiology.

Disclosure

Author's contributions:

Conceptualization: Karol Demel
Methodology: Maciej Łydka, Justyna Lewandowska
Software: Filip Kowal, Adrian Dyląg, Jakub Król
Check: Monika Dziedzic, Julia Holzer
Formal analysis: Maciej Łydka, Natalia Libudzic
Investigation: Justyna Talaska, Filip Kowal, Jakub Król
Resources: Julia Holzer, Karol Demel
Data curation: Justyna Lewandowska, Natalia Libudzic
Writing – rough preparation: Monika Dziedzic, Adrian Dyląg
Writing – review and editing: Karol Demel, Justyna Talaska
Visualization: Justyna Lewandowska, Maciej Łydka, Monika Dziedzic
Supervision: Justyna Talaska, Julia Holzer, Filip Kowal
Project administration: Jakub Król, Adrian Dyląg

All authors have read and agreed with the published version of the manuscript.

Funding Statement: The article did not receive any funding.
Institutional Review Board Statement: Not applicable.
Informed Consent Statement: Not applicable.
Data Availability Statement: Not applicable.
Acknowledgments: Not applicable.
Conflict of Interest Statement: Authors declare no conflict of interest.

REFERENCES

1. Güzey Akansel, M., Baş, M., Gençalp, C., Kahrıman, M., Şahin, E., Öztürk, H., Gür, G., & Gür, C. (2024). Effects of the ketogenic diet on microbiota composition and short-chain fatty acids in women with overweight/obesity. *Nutrients*, *16*(24), 4374. https://doi.org/10.3390/nu16244374
2. Karačić, A., Dikarlo, P., Dorst, I., Renko, I., & Liberati Pršo, A.-M. (2024). The gut microbiome without any plant food? A case study on an individual following a carnivore diet. *Medical Anthropology & Health*, *2*(1). https://doi.org/10.1530/MAH-24-0006
3. Fu, J., Zheng, Y., Gao, Y., & Xu, W. (2022). Dietary fiber intake and gut microbiota in human health. *Microorganisms*, *10*(12), 2507. https://doi.org/10.3390/microorganisms10122507
4. Shen, S., Prame Kumar, K., Wen, S. W., Shim, R., Wanrooy, B. J., Stanley, D., Moore, R. J., Van, T. T. H., Robert, R., Hickey, M. J., & Wong, C. H. Y. (2021). Deficiency of dietary fiber modulates gut microbiota composition, neutrophil recruitment and worsens experimental colitis. *Frontiers in Immunology*, *12*, 619366. https://doi.org/10.3389/fimmu.2021.619366
5. Li, W., Gong, M., Wang, Z., Pan, H., Li, Y., & Zhang, C. (2024). The gut microbiota changed by ketogenic diets contribute to glucose intolerance rather than lipid accumulation. *Frontiers in Endocrinology*, *15*, 1446287. https://doi.org/10.3389/fendo.2024.1446287
6. Norwitz, N. G., & Soto-Mota, A. (2024). Case report: Carnivore-ketogenic diet for the treatment of inflammatory bowel disease: a case series of 10 patients. *Frontiers in Nutrition*, *11*, 1467475. https://doi.org/10.3389/fnut.2024.1467475
7. Page, M. J., McKenzie, J. E., Bossuyt, P. M., Boutron, I., Hoffmann, T. C., Mulrow, C. D., Shamseer, L., Tetzlaff, J. M., Akl, E. A., Brennan, S. E., Chou, R., Glanville, J., Grimshaw, J. M., Hróbjartsson, A., Lalu, M. M., Li, T., Loder, E. W., Mayo-Wilson, E., McDonald, S., … Moher, D. (2021). The PRISMA 2020 statement: An updated guideline for reporting systematic reviews. *BMJ, 372*, n71. https://doi.org/10.1136/bmj.n71
8. Rew, L., Harris, M. D., & Goldie, J. (2022). The ketogenic diet: Its impact on human gut microbiota and potential consequent health outcomes: A systematic literature review. *Gastroenterology and Hepatology From Bed to Bench*, *15*(4), 326–342. https://doi.org/10.22037/ghfbb.v15i4.2600
9. Goedeke, S., Murphy, T., Rush, A., & Zinn, C. (2024). Assessing the nutrient composition of a carnivore diet: A case study model. *Nutrients*, *17*(1), 140. https://doi.org/10.3390/nu17010140
10. Wang, Y., Uffelman, C. N., Bergia, R. E., Clark, C. M., Reed, J. B., Cross, T. L., Lindemann, S. R., Tang, M., & Campbell, W. W. (2023). Meat consumption and gut microbiota: A scoping review of literature and systematic review of randomized controlled trials in adults. *Advances in Nutrition (Bethesda, Md.)*, *14*(2), 215–237. https://doi.org/10.1016/j.advnut.2022.10.005
11. Larsson, S. C., Ericson, U., Dekkers, K. F., Arage, G., Rašo, L. M., Sayols-Baixeras, S., Hammar, U., Baldanzi, G., Nguyen, D., Nielsen, H. B., Holm, J. B., Risérus, U., Michaëlsson, K., Sundström, J., Smith, J. G., Engström, G., Ärnlöv, J., Orho-Melander, M., Fall, T., & Ahmad, S. (2025). Meat intake in relation to composition and function of gut microbiota. *Clinical Nutrition*, *45*, 124–133. https://doi.org/10.1016/j.clnu.2024.12.034
12. Olson, C. A., Vuong, H. E., Yano, J. M., Liang, Q. Y., Nusbaum, D. J., & Hsiao, E. Y. (2018). The gut microbiota mediates the anti-seizure effects of the ketogenic diet. *Cell, 173*(7), 1728–1741.e13. https://doi.org/10.1016/j.cell.2018.04.027
13. David, L. A., Maurice, C. F., Carmody, R. N., Gootenberg, D. B., Button, J. E., Wolfe, B. E., Ling, A. V., Devlin, A. S., Varma, Y., Fischbach, M. A., Biddinger, S. B., Dutton, R. J., & Turnbaugh, P. J. (2014). Diet rapidly and reproducibly alters the human gut microbiome. *Nature, 505*(7484), 559–563. https://doi.org/10.1038/nature12820
14. Russell, W. R., Hoyles, L., Flint, H. J., & Dumas, M.-E. (2013). Colonic bacterial metabolites and human health. *Current Opinion in Microbiology, 16*(3), 246–254. https://doi.org/10.1016/j.mib.2013.07.002
15. Sheflin, A. M., Melby, C. L., Carbonero, F., & Weir, T. L. (2017). Linking dietary patterns with gut microbial composition and function. *Gut Microbes*, *8*(2), 113–129. https://doi.org/10.1080/19490976.2016.1270809
16. Parada Venegas, D., De la Fuente, M. K., Landskron, G., González, M. J., Quera, R., Dijkstra, G., Harmsen, H. J. M., Faber, K. N., & Hermoso, M. A. (2019). Short chain fatty acids (SCFAs)—mediated gut epithelial and immune regulation and its relevance for inflammatory bowel diseases. *Frontiers in Immunology*, *10*, 277. https://doi.org/10.3389/fimmu.2019.00277

17. Desai, M. S., Seekatz, A. M., Koropatkin, N. M., Kamada, N., Hickey, C. A., Wolter, M., Pudlo, N. A., Kitamoto, S., Terrapon, N., Muller, A., Young, V. B., Henrissat, B., Wilmes, P., Stappenbeck, T. S., Núñez, G., & Martens, E. C. (2016). A dietary fiber–deprived gut microbiota degrades the colonic mucus barrier and enhances pathogen susceptibility. *Cell, 167*(5), 1339–1353.e21. https://doi.org/10.1016/j.cell.2016.10.043
18. Youm, Y.-H., Nguyen, K. Y., Grant, R. W., Goldberg, E. L., Bodogai, M., Kim, D., D'Agostino, D., Planavsky, N., Lupfer, C., Kanneganti, T.-D., Kang, S., Horvath, T. L., Fahmy, T. M., Crawford, P. A., Biragyn, A., & Dixit, V. D. (2015). The ketone metabolite β-hydroxybutyrate blocks NLRP3 inflammasome–mediated inflammatory disease. *Nature Medicine, 21*(3), 263–269. https://doi.org/10.1038/nm.3804
19. deCampo, D. M., & Kossoff, E. H. (2019). Ketogenic dietary therapies for epilepsy and beyond. *Current Opinion in Clinical Nutrition and Metabolic Care*, *22*(4), 264–268. https://doi.org/10.1097/MCO.0000000000000565