Review Article

Metabolic Diets and Fasting in Oncology: Integrating Guideline-Based Nutrition, Nutrition Assessments and Biomarker-Driven Clinical Pathology

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Popular narratives often promote “special diets” as strategies to cure cancer, frequently invoking autophagy and Nobel-related discoveries. In contrast, international oncology nutrition guidelines state that no diet has been proven to reproducibly cure cancer. This review integrates guideline-based oncology nutrition, experimental metabolic diets and a biomarker-driven clinical pathology perspective within a translational framework.

This narrative review synthesizes international and national oncology nutrition guidelines and clinical evidence on fasting, fasting-mimicking diets, time-restricted eating, intermittent fasting, Ramadan fasting and ketogenic diets in cancer. Priority was given to randomized and prospective trials, systematic reviews and selected mechanistic studies, with attention to nutritional risk, toxicity, metabolic and inflammatory markers, body composition, cachexia staging and pathological response. Current guidelines discourage restrictive diets outside evidence-based care and recommend early nutritional screening and stepwise nutrition support. Clinical studies suggest that fasting-related regimens and ketogenic diets may improve selected metabolic, hematological, quality-of-life and pathological endpoints in carefully selected patients, but a reproducible survival benefit has not been established. Cachexia-oriented models indicate that cancer-related nutritional decline is multidimensional and partly integrates biochemical, inflammatory, functional and patient-reported domains. Mechanistic studies support plausible effects on autophagy, nutrient-sensing pathways, ketone metabolism, inflammation and the tumor microenvironment.

The evidence supports a clear distinction between guideline-based oncology nutrition, which is fundamental supportive care, and metabolic diets or fasting regimens, which remain experimental adjuncts rather than curative treatments. The translational opportunity lies in embedding these

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interventions within malnutrition-, sarcopenia- and cachexia-aware frameworks and using biomarkers to improve risk stratification, patient selection and mechanistic interpretation.

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Highlights

- Oncology nutrition guidance does not support diets as cancer cures.
- GLIM, EWGSOP2 and cachexia frameworks improve clinical phenotyping.
- Biomarkers may complement clinical and imaging assessment in oncology.
- Fasting and ketogenic diets remain feasible but unproven for survival benefit.
- The review integrates malnutrition, sarcopenia and cachexia with clinical pathology perspectives.

Introduction

Claims that specific diets can “starve” cancer cells, “activate autophagy” or otherwise cure malignancy have become highly visible in patient communities and social media, sometimes encouraging patients to delay or abandon evidence-based treatment in favor of restrictive regimens often supported by selective interpretations of preclinical findings and misconceptions surrounding Nobel Prize-winning research on autophagy [1]. Fasting regimens, ketogenic diets (KD), alkaline diets, and other nutritional strategies are frequently promoted as stand-alone anticancer therapies, despite clinical evidence remaining limited.

This narrative stands in direct contrast to contemporary oncology nutrition guidance. The European Society for Clinical Nutrition and Metabolism (ESPEN), the European Society for Medical Oncology (ESMO), Sociedad Española de Oncología Médica (SEOM), American Society of Clinical Oncology (ASCO), National Comprehensive Cancer Network (NCCN) and the National Cancer Institute (NCI)-related materials converge on a common message: no specific diet has been shown to cure cancer or reliably prevent recurrence, and dietary care in oncology should be individualized, evidence-based and integrated with multidisciplinary cancer treatment. ASCO’s guideline on exercise, diet and weight management during active treatment is especially relevant in this context because it supports healthy dietary patterns and individualized counseling, yet concludes that evidence remains insufficient to endorse specific diets during treatment [2][3][4][5][6][7][8][9][10].

At the same time, oncology nutrition has evolved from a largely intake- and weight-focused discipline into one that increasingly differentiates malnutrition, sarcopenia and cachexia as related but distinct clinical constructs. The Global Leadership Initiative on Malnutrition (GLIM) has formalized malnutrition diagnosis through phenotypic and etiologic criteria, the revised European Working Group on Sarcopenia in Older People (EWGSOP2) has refined the definition of sarcopenia around strength and muscle quantity/quality, and cachexia frameworks have increasingly incorporated symptom burden, inflammation, function and prognosis. This shift is highly relevant when considering metabolic diets, because any intervention that restricts intake or changes substrate availability must be interpreted against baseline nutritional vulnerability, inflammatory burden, body composition and treatment intent [3][4][11][12][13][14][15][16][17][18][19][20].

In parallel, a growing clinical literature is evaluating fasting-mimicking diets (FMDs), time-restricted eating (TRE), Ramadan-patterned fasting and KDs as adjuncts to chemotherapy, endocrine therapy, radiotherapy and immunotherapy. These studies have generated data not only on feasibility and safety but also on endocrine, metabolic, inflammatory, body-composition and pathological endpoints. This makes the field particularly relevant to clinical pathology and laboratory medicine, where the central question is not simply whether a diet is feasible, but whether measurable biomarkers can help identify who is likely to benefit, who is vulnerable to harm, and whether systemic metabolic shifts correspond to clinically meaningful biological effects within tumors [6][20][21][22][23][24][25][26][27][28][29].

The purpose of this review is not to endorse ‘cancer diets,’ but to place metabolic interventions within the broader framework of oncology nutrition and cachexia care. The manuscript examines how major guidelines address nutritional support and unproven diets, reviews the principal dietary regimens studied in cancer, discusses the biological rationale underlying these approaches, and considers how biomarker-informed assessment may complement current frameworks for malnutrition, sarcopenia, and cachexia. Its novelty lies in integrating guideline-based oncology nutrition, experimental metabolic diets, and clinical pathology perspectives into a single translational review, while distinguishing among related but non-identical nutritional syndromes [2][4][6][7][11][12][13][14][20][21][22][30][31].

Methods

This study is a narrative, non-systematic review integrating oncology nutrition and cachexia guidelines, peri-operative and survivorship frameworks, clinical studies of metabolic diets and fasting, and selected mechanistic and translational literature. Searches were based on PubMed/MEDLINE, Web of Science, and

Scopus from approximately 2011 to March 2026 and were supplemented by hand-searching reference lists and organizational websites [3][4][8][13][32][33][34][35].

Search concepts combined terms related to cancer and oncology with nutrition, malnutrition, sarcopenia, cachexia, body composition, fasting, fasting-mimicking diet, intermittent fasting, time-restricted eating, Ramadan fasting, ketogenic diet, biomarkers, autophagy, IGF-1, insulin, ketone bodies, and inflammation. Priority was given to international and national guidelines, randomized trials, prospective cohorts, systematic reviews, and meta-analyses, together with selected high-impact mechanistic studies relevant to tumor metabolism, autophagy, immune regulation, morphogenesis, and cachexia assessment [6][11][12][14][18][19][20][21][22][31][36].

Because the evidence base is heterogeneous with respect to tumor type, intervention design, timing, endpoints, and comparator groups, no quantitative synthesis was attempted. Instead, the literature was examined comparatively in order to identify recurring themes, clinically relevant tensions, and the most appropriate points at which biomarker-informed assessment can be integrated into existing nutritional frameworks without overstating current evidence.

Guideline-based oncology nutrition and the position on cancer diets

ESPEN, ESMO, SEOM, and ASCO

ESPEN's cancer nutrition guideline and practical guideline are explicit that no diet is known to reproducibly cure cancer or prevent recurrence and that restrictive "anticancer diets" without evidence should be discouraged. These documents recommend systematic screening for nutritional risk at diagnosis and throughout treatment, early nutrition counseling, escalation from oral strategies to enteral or parenteral nutrition when appropriate, and close integration of nutrition with exercise and symptom management [3][4].

ESMO places similar emphasis on structured supportive care. Its clinical practice guideline on cancer cachexia defines cachexia as a multifactorial syndrome characterized by ongoing skeletal muscle loss, with or without fat loss, that cannot be fully reversed by conventional nutritional support and leads to functional impairment. ESMO recommends systematic assessment of weight loss, Body Mass Index (BMI), appetite, inflammatory burden, and functional status, GLIM-based malnutrition diagnosis, together with multimodal management including dietary counseling, physical activity, symptom control, psychosocial support, and selected pharmacological interventions where appropriate [32].

SEOM guidance reinforces the need for early nutrition assessment, multidisciplinary nutrition care and attention to body composition, including sarcopenia and sarcopenic obesity, within routine oncology pathways. ASCO fits coherently within this same framework. Its guideline on exercise, diet and weight management during active treatment endorses individualized dietary support and healthy eating patterns while acknowledging that evidence is insufficient to support specific therapeutic diets during active treatment. ASCO's cachexia guidance and 2023 rapid recommendation update similarly frame cachexia as a multidimensional syndrome requiring clinical, nutritional and symptom-based assessment, with dietary counseling remaining a core component of management [2][8][13].

Taken together, these guidelines do not support the notion of a curative "anticancer diet." Rather, they define nutrition as evidence-based supportive care whose goals are to preserve body mass and function, improve treatment tolerance, reduce complications and protect quality of life.

GLIM, EWGSOP2 and the distinction between malnutrition and sarcopenia

GLIM offers a consensus approach to diagnosing malnutrition through a two-step process: patients are first screened for nutrition risk and are then diagnosed with malnutrition if they meet at least one phenotypic criterion and one etiologic criterion. The phenotypic domain includes non-volitional weight loss, low BMI and reduced muscle mass, while the etiologic domain includes reduced intake or assimilation and inflammation or disease burden. In oncology, GLIM-defined malnutrition is associated with greater treatment toxicity, poorer quality of life and shorter survival [7][18][37].

EWGSOP2 defines sarcopenia primarily through low muscle strength, confirmed by reduced muscle quantity or quality, with impaired physical performance indicating severe sarcopenia. The framework relies on measures such as handgrip strength, chair-stand performance, gait speed and appendicular lean mass, together with imaging- or device-based assessment of muscle quantity and quality. This is especially important in oncology because muscle depletion may coexist with normal or elevated BMI, thereby making weight alone an unreliable guide to nutritional risk [7][11][38].

These frameworks are clinical models that combine anthropometry, function, body composition and simple etiologic assessment, including inflammatory burden in the case of GLIM. Their value in the present context is that they help separate patients who may safely participate in metabolic-diet studies from those in whom additional caloric restriction could worsen malnutrition, accelerate muscle loss or undermine cancer treatment [6][7][30][37][38][39].

Cachexia frameworks and staging models

GLIM and EWGSOP2, however, do not exhaust the problem of cancer-related nutritional decline because cachexia is not synonymous with malnutrition or sarcopenia. Cachexia is a syndrome-level construct that integrates progressive weight and muscle loss with systemic inflammation, anorexia, functional decline and adverse prognosis. For this reason, cachexia requires complementary frameworks that capture features not fully encompassed by GLIM or EWGSOP2 alone [12][13][14][32].

The traditional stage-based cachexia continuum of pre-cachexia, cachexia and refractory cachexia remains clinically useful because it links assessment to treatment intent and prognosis. More multidimensional models have also emerged. The Cachexia Score (CASCO) was developed as a staging tool that includes body weight and lean body mass loss, anorexia, inflammatory, immunological and metabolic disturbances, physical performance and quality of life, thereby explicitly integrating laboratory and patient-centered variables. More recent indices such as the Cancer Cachexia Staging Index incorporate weight-loss patterns, body composition, appetite, physical status and inflammatory markers such as C-reactive protein (CRP), prealbumin (PA) and neutrophil-to-lymphocyte ratio (NLR), and have shown prognostic value for complications, recurrence and survival. Simpler cachexia staging scores have likewise combined weight loss, functional measures, appetite and abnormal biochemistry to discriminate pre-cachexia, cachexia and refractory cachexia with prognostic relevance [14][15][16][17][36].

These frameworks show that biomarker-enriched nutritional assessment is not a purely hypothetical proposal. In the cachexia field, multidimensional models already exist that combine clinical, functional and biochemical domains. The unresolved question is how such models should interface with GLIM- and EWGSOP2-based assessment and whether metabolic-diet trials should routinely adopt these broader cachexia-aware classifications in order to improve patient protection and biological interpretability [12][13][14][16].

North American, peri-operative and pediatric extensions

NCCN educational materials and the NCI PDQ “Nutrition in Cancer Care” monograph similarly emphasize early nutrition screening, recognition of intake problems and weight loss, referral to dietitians, and individualized supportive strategies rather than unproven diets. In North American practice, these resources are highly compatible with ASCO guidance and provide further support for integrating nutrition into multidisciplinary cancer care [2][7][33][37].

Enhanced Recovery After Surgery (ERAS) pathways extend these principles to surgical oncology. Their emphasis on preoperative nutritional optimization, minimization of unnecessary fasting, carbohydrate loading, early postoperative oral or enteral feeding, and avoidance of unnecessary parenteral nutrition highlights how structured nutrition care can influence infection rates, wound healing, recovery time, and other outcomes central to treatment tolerance. This is especially relevant when discussing fasting-based interventions, because perioperative fasting in ERAS is managed with a very different logic from elective metabolic restriction in experimental oncology nutrition [34].

Pediatric frameworks and conceptual models

Pediatric oncology frameworks, including International Society of Pediatric Oncology / International Initiative for Pediatrics and Nutrition (SIOP/IIPAN) recommendations and national groups (e.g., Italian Association of Pediatric Hematology and Oncology/Alleanza Contro Cancro (AIEOP/ACC), Indian Association for Parenteral and Enteral Nutrition (IAPEN)), also recognize nutrition as a core component of supportive care and stress regular screening, anthropometry, body-composition follow-up, and dedicated pathways for high-risk contexts such as Central Nervous System (CNS) tumors and transplantation. Although the present review is mainly adult-focused, pediatric guidance reinforces the same fundamental principle: in oncology, nutritional vulnerability must be identified early and protected, not challenged with restrictive regimens in the absence of robust evidence [6][7][40].

Metabolic diets studied in oncology

Fasting and fasting-mimicking diets

Among the metabolic interventions studied in cancer, FMDs have attracted particular interest because of their proposed effects on treatment tolerance, cellular stress resistance, and growth factor signaling. Reviews and prospective studies suggest that short-term fasting around chemotherapy is generally feasible in carefully selected, well-nourished adults and may reduce selected toxicities and improve quality of life, although a survival benefit has not been shown [6][21][23][25][41][42][43].

The multicenter DIRECT trial remains a key study. In women with human epidermal growth factor receptor 2 (HER2)-negative stage II–III breast cancer receiving neoadjuvant chemotherapy, a fasting-mimicking diet reduced insulin-like growth factor (IGF)-1 and glucose, increased ketone levels, and was associated with signals in radiological and pathological response, but the trial was not powered to

demonstrate a survival benefit. More recent studies have reported similar metabolic and hematological effects, including reduced vomiting, neutropenia, body weight, and visceral fat, together with preservation of muscle mass in selected breast cancer cohorts. A systematic review of intermittent fasting during chemotherapy found that these regimens were generally safe and feasible, with some suggestion of reduced toxicities and improved fatigue, but insufficient evidence for oncological endpoints [6][21][23][41][44].

From a clinical pathology perspective, these studies repeatedly measured IGF-1, insulin, glucose, ketones, lipid profiles, complete blood counts, and inflammatory markers, suggesting candidate biomarkers for future standardization. However, the evidence remains limited by small sample sizes, heterogeneous schedules, and the frequent exclusion of nutritionally vulnerable patients [23][24][41][45].

Time-restricted eating and related intermittent fasting models

TRE has a broader lifestyle and survivorship profile than classical fasting regimens and may therefore seem more clinically accessible. A 2025 systematic review found that TRE interventions in cancer were generally well tolerated, with adherence rates of 67% and 98% and signals for improvements in quality of life, fatigue, and selected metabolic markers, but the evidence base remained small and heterogeneous across tumor types, eating windows, and treatment contexts[28][46][47].

Observational work in early breast cancer suggested that shorter overnight fasting may be associated with a higher recurrence risk, but this finding should be interpreted as hypothesis-generating rather than causal evidence. From a laboratory perspective, TRE studies have mainly assessed fasting glucose, insulin, hemoglobin A1c (HbA1c), lipid profile, CRP, and selected cancer-related markers, providing a basis for future biomarker standardization rather than routine clinical application[46][47][48][49][50].

ASCO guidance supports healthy dietary patterns during active treatment but does not endorse specific therapeutic diets. Accordingly, TRE should currently be framed as a promising behavioral intervention, not a validated antitumor strategy [2][19].

Ramadan fasting

Ramadan fasting, involving abstention from food and drink from dawn to sunset for approximately one lunar month, provides a naturalistic model of daily intermittent fasting for patients who fast for religious reasons. Reviews suggest that stable outpatients with preserved performance status may often fast safely

under supervision, and some studies describe psychological or spiritual benefits; however, there is no evidence that Ramadan fasting leads to tumor regression or improved survival[29][51].

Observational studies in colorectal, gastric, and mixed tumor cohorts have examined biochemical parameters, including renal and liver function, electrolytes, and tumor markers, and generally found no major laboratory derangements when fasting was supervised, although patient selection bias and small sample sizes limit interpretation. In colorectal cancer patients receiving chemotherapy, Ramadan fasting was reported to be feasible and well tolerated, with no significant adverse effect on renal function or major laboratory variables. From a clinical pathology perspective, these studies show how laboratory monitoring, hydration assessment, symptom tracking, and individualized counseling can be applied when patients choose to fast outside a formal trial[29][52][53][54].

This supports the broader principle that oncology nutrition must remain patient-centered and safety-oriented, even when dietary behavior is shaped by religious practice rather than experimental intervention [29].

Ketogenic diets

KDs have been studied most intensively in neuro-oncology, especially glioma and glioblastoma, because of longstanding interest in tumor glucose dependence and metabolic flexibility. Systematic reviews and meta-analyses suggest that KDs are generally feasible in cancer patients and can induce ketosis with favorable changes in glucose, IGF-1, body weight, and fat mass, although a survival benefit has not been demonstrated reliably [55][56][57][58][59][60][61][62][63][64].

Clinical studies commonly monitor ketone levels, glucose, lipid profiles, liver and renal function, and, in some neuro-oncology protocols, imaging or histopathological findings. This makes KDs particularly relevant to a pathology-oriented review because they are among the few dietary interventions in oncology linked not only to body composition and patient-reported outcomes but also to tissue and imaging correlates [55][56][57][60][61][62][63][65].

Outside neuro-oncology, ketogenic approaches have also been explored in gastrointestinal malignancies and mixed advanced-cancer populations, but the evidence remains more suggestive of feasibility and metabolic modulation than of reliable tumor control. Careful attention is therefore needed to the risk of unintended weight loss and dietary burden in nutritionally vulnerable patients [19][40][66][67].

Biological rationale and mechanistic limits

Mechanistic studies have identified several biological pathways through which metabolic diets and fasting may influence cancer biology and treatment tolerance. From a clinical pathology perspective, these pathways generate testable hypotheses and candidate biomarkers.

The interest in metabolic diets rests in part on their capacity to alter nutrient sensing, growth signaling and stress-response pathways. Fasting and FMDs can reduce circulating IGF-1, insulin and glucose while increasing ketone production, activating adenosine monophosphate-activated protein kinase (AMPK), inhibiting mechanistic target of rapamycin complex 1 (mTORC1) and promoting autophagy, thereby shifting cellular metabolism from anabolic to catabolic states. These adaptations may help normal tissues tolerate treatment-related stress while exposing metabolically constrained tumor cells to additional pressure, an idea often described as differential stress resistance [21][22][39][68][69].

From a pathology perspective, these mechanisms are potentially measurable through immunohistochemical markers of autophagy such as microtubule-associated protein 1 light chain 3 (LC3) and sequestosome 1 (p62/SQSTM1), ultrastructural assessment of autophagosomes and gene expression signatures. However, autophagy remains highly context-dependent and may be tumor-suppressive in one setting and tumor-supportive in another, while heterogeneous autophagic activity across human tumors currently limits its value as a predictive biomarker for selecting patients for fasting-based interventions. Likewise, not all cancers are equally restricted by carbohydrate limitation or equally unable to adapt to ketone-rich or fasting-like conditions [22][31][39][68][69][70][71][72].

Recent metabolic-flux studies in glioblastoma have identified distinct metabolic phenotypes under ketogenic conditions, suggesting that tumor-level metabolic heterogeneity may account for some of the variability observed across preclinical and clinical studies. Beyond nutrient sensing, metabolic interventions may also influence endocrine, inflammatory and immune pathways that are accessible to clinical pathology. Reductions in circulating IGF-1, insulin and glucose, together with increased ketone body production, have been observed in clinical studies of fasting and FMDs and may influence tumor proliferation, mitochondrial metabolism, oxidative stress and host immune responses. These systemic adaptations can be monitored using routine laboratory measurements, including glucose, insulin, IGF-1, ketone bodies and lipid profiles, and may ultimately be integrated with inflammatory markers and body composition into composite metabolic-response biomarkers[60][61][62][68][69][72].

At the same time, fasting and ketogenic interventions may modify the tumor microenvironment by altering cytokine signaling, myeloid-derived suppressor cell activity (MDSC), T-cell exhaustion (TEX) and immune checkpoint expression (ICP). Although preliminary human studies have reported reductions in inflammatory markers such as CRP following fasting interventions, evidence remains limited, highlighting the need for translational studies incorporating cytokine profiling, flow cytometry and molecular immune signatures[68][69][73].

Mechanistic interest also extends beyond metabolism alone. Studies in glioblastoma models have linked ketogenic or calorie-restricted interventions to reduced microvascular density, altered proliferation markers and changes in inflammatory signaling. More recently, advanced three-dimensional (3D) spatial transcriptomic analyses have suggested that metastatic outgrowth in breast cancer follows a highly organized morphogenetic blueprint, with metastases forming fractal epithelial cords orchestrated by embryonic-like transcriptional programs and growth factor signaling. These structured architectures may depend on metabolic support from IGF-1, fibroblast growth factor (FGF) and glucose-driven pathways, raising the hypothesis that systemic metabolic interventions capable of suppressing these signals, such as FMDs, could disrupt morphogenetic programs that sustain metastatic progression. Although such findings remain far from direct clinical proof, they support the rationale for carefully designed translational oncology-nutrition studies integrating tissue-based biomarkers, spatial transcriptomics, multiplex immunohistochemistry and metabolomic imaging in paired pre- and post-intervention tumor samples [20][60][61][62][68][69][72][74][75].

These complementary mechanisms help explain why endocrine, inflammatory and tissue biomarkers recur across clinical trials and why future studies will likely need to integrate systemic metabolic measurements with spatially resolved tissue endpoints if they are to move beyond feasibility and establish biologically interpretable, pathology-informed oncology research[68][69].

Nutritional risk, patient selection and biomarker-informed assessment

A central lesson from both guidelines and clinical trials is that the potential meaning of a metabolic intervention depends heavily on baseline nutritional status. Malnutrition, sarcopenia, and cachexia are not background variables; they are major determinants of toxicity, functional decline, treatment interruption, and survival. This is why studies of FMDs, KDs, and related regimens almost always exclude

low-BMI patients, those with significant recent weight loss, poor performance status, or uncontrolled comorbidities [7][12][13][18][19][30][37][38].

In this context, GLIM and EWGSOP2 are the backbone of assessment because they provide clinically grounded ways to identify malnutrition and muscle failure before any restrictive intervention is considered. At the same time, cachexia-specific staging models remind clinicians that inflammation, anorexia, function, symptom burden, and prognosis cannot always be inferred from malnutrition or sarcopenia criteria alone. This is particularly important in advanced cancer, where a patient may satisfy elements of malnutrition or low muscle mass yet also display a broader catabolic syndrome with different implications for treatment and supportive care [7][11][12][13][14][16][17][30][32].

Trials of FMD, KD, and TRE have typically excluded patients with low BMI, significant recent weight loss, poor performance status, or uncontrolled comorbidities and have mandated baseline and on-treatment laboratory monitoring: complete blood count, electrolytes, liver and renal function, glucose, insulin and IGF-1, ketones, and lipid profile. Clinical pathologists play a crucial role in interpreting these data, identifying abnormalities that require intervention, and contributing to composite risk–benefit assessments[76][77].

If fasting-related regimens are partly mediated through reduced IGF-1, insulin, and glucose and increased ketogenesis, then endocrine and metabolic markers such as IGF-1, insulin, fasting glucose, β-hydroxybutyrate, and lipid profile become logical companion measures in interventional trials. If malnutrition and cachexia are driven in part by inflammatory and catabolic processes, then CRP, albumin, interleukin (IL)-6, tumor necrosis factor (TNF)-α, PA, and related indices may help clarify when metabolic interventions are biologically inappropriate or potentially harmful [6][7][12][22][37][38][78].

Body-composition and functional measures remain equally important because biochemical data alone cannot capture the clinical consequences of nutritional stress. Computed tomography-derived skeletal muscle index (CT-SMI), visceral adiposity, dual-energy X-ray absorptiometry (DXA)- or bioimpedance-derived lean mass, phase angle (PhA), handgrip strength, and chair-stand performance are especially relevant where a patient may appear weight-stable but is in fact losing muscle or function. Finally, in translational settings, tumor and microenvironmental markers such as light chain 3 (LC3), p62, Ki-67, cleaved caspase-3, cluster of differentiation 31 (CD31), immune-infiltration patterns, and spatial or metabolomic signatures may help link systemic metabolic shifts to tissue biology [11][20][22][38][74][78].

By integrating biomarkers, guideline-based nutritional assessment identifies who is vulnerable, while laboratory, imaging, and tissue measures may help explain what a metabolic intervention is doing, for whom, and at what biological cost. Cachexia frameworks strengthen this interpretation by showing that multidimensional assessment already exists in oncology and can serve as a bridge between supportive care nutrition and translational biomarker research [7][12][13][14][30].

Implications for future research and clinical interpretation

The current evidence does not justify recommending fasting regimens, KDs or TRE as anticancer treatments in routine practice. What it does justify is a more disciplined research agenda. Future studies should be embedded within formal nutritional pathways rather than designed as isolated lifestyle experiments, because the clinical meaning of any metabolic intervention depends on whether patients are well nourished, sarcopenic, inflamed, actively losing muscle, or already on a cachexia trajectory [2][3][4][6][7][12][13][30][37][38].

This has several practical consequences for study design. Eligibility should be anchored in standardized malnutrition and sarcopenia assessment, ideally using GLIM and EWGSOP2-compatible definitions, while advanced-disease cohorts should also consider cachexia-aware staging where prognosis, appetite loss, inflammation and functional decline are central to risk interpretation. Laboratory monitoring should go beyond routine safety chemistry to include the metabolic markers most consistently affected by these interventions, particularly IGF-1, insulin, glucose, ketones and inflammatory indices, because these variables may distinguish true biological response from simple adherence or caloric deprivation [6][7][11][12][13][16][21][22][78].

Where tumor tissue is available, translational substudies should be integrated prospectively rather than appended later. This would allow routine histopathology to be aligned with proliferation, apoptosis, vascular, immune and spatial endpoints, creating a more coherent bridge between malnutrition, sarcopenia, cachexia biology and tumor behavior. Likewise, microbiome and metabolomic analyses may prove useful when the objective is to understand why some patients tolerate or respond to metabolic interventions while others deteriorate nutritionally or derive no apparent benefit [11][20][21][22][74].

From a clinical standpoint, the most important implication is restraint. International guidance already provides a strong framework for cancer nutrition care, and the task is not to replace it with restrictive diet culture but to determine whether selected metabolic interventions can be studied safely and

meaningfully inside it. That question can only be answered through trials that are nutritionally literate, malnutrition-, sarcopenia-, and cachexia-aware, biomarker-informed and explicit about patient protection [2][3][4][6][7][12][13].

Conclusions

International oncology nutrition guidance from ESPEN, ESMO, ASCO, SEOM, NCCN and related bodies is remarkably consistent in its central message: no special diet has been shown to cure cancer, and nutrition in oncology should be approached as structured supportive care rather than as an alternative to evidence-based treatment. Within this framework, GLIM and EWGSOP2 provide clinically useful ways to identify malnutrition and sarcopenia, while cachexia-oriented guidance and staging systems remind clinicians that cancer-related nutritional decline also includes syndrome-level processes involving inflammation, anorexia, function, prognosis and quality of life [2][7][11][12][13][14][16][30][37][79].

The human literature on FMDs, KDs, TRE and Ramadan fasting suggests feasibility and biological activity in selected, well-nourished patients, with recurring signals in endocrine, metabolic, inflammatory, body-composition and occasionally pathological endpoints, but without convincing evidence of reproducible survival benefit. The appropriate scientific response is therefore neither dismissal of all metabolic interventions nor acceptance of anticancer diet claims. Rather, it is to study these interventions inside robust nutritional, malnutrition-, sarcopenia- and cachexia-aware frameworks and to use biomarkers not as substitutes for clinical assessment, but as tools that may refine interpretation of tolerance, mechanism and patient selection [6][7][11][12][13][18][19][21][22][38][78].

In this sense, the future of oncology nutrition is unlikely to lie in a single “cancer diet.” It is more likely to lie in a tighter integration of guideline-based supportive care, rigorous nutritional phenotyping, malnutrition and sarcopenia assessments, cachexia staging and translational biomarker strategies that can distinguish physiological adaptation from nutritional harm and biological promise from therapeutic overstatement [7][11][12][14][21][22].

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Potential Competing Interests

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Author Contributions

The author contributed fully to the conceptualization and methodology of the study, prepared the original draft, and completed the editing and final revisions.

References

1. ^*Levine B, Klionsky DJ (2017). "Autophagy Wins the 2016 Nobel Prize in Physiology or Medicine: Breakthroughs in Baker's Yeast Fuel Advances in Biomedical Research." Proc Natl Acad Sci U S A. **114**:201–205. doi:10.1073/pnas.1619876114.*
2. a, b, c, d, e, f, g, h*Ligibel JA, Bohlke K, May AM, Clinton SK, Demark-Wahnefried W, Gilchrist SC, Irwin ML, Late M, Mansfield S, Marshall TF, Meyerhardt JA, Thomson CA, Wood WA, Alfano CM (2022). "Exercise, Diet, and Weight Management During Cancer Treatment: ASCO Guideline." J Clin Oncol. **40**:2491–2507. doi:10.1200/JCO.22.00687.*
3. a, b, c, d, e, f*Arends J, Bachmann P, Baracos V, Barthelemy N, Bertz H, Bozzetti F, Fearon K, Hütterer E, Isenring E, Kaasa S, Krznaric Z, Laird B, Larsson M, Laviano A, Mühlebach S, Muscaritoli M, Oldervoll L, Ravasco P, Solheim T, Strasser F, de van der Schueren M, Preiser JC (2017). "ESPEN Guidelines on Nutrition in Cancer Patients." Clin Nutr. **36**:11–48. doi:10.1016/j.clnu.2016.07.015.*
4. a, b, c, d, e, f, g*Muscaritoli M, Arends J, Bachmann P, Baracos V, Barthelemy N, Bertz H, Bozzetti F, Hütterer E, Isenring E, Kaasa S, Krznaric Z, Laird B, Larsson M, Laviano A, Mühlebach S, Oldervoll L, Ravasco P, Solheim TS, Strasser F, de van der Schueren M, Preiser JC, Bischoff SC (2021). "ESPEN Practical Guideline: Clinical Nutrition in Cancer." Clin Nutr. **40**:2898–2913. doi:10.1016/j.clnu.2021.02.005.*
5. ^*Liposits G, Orrevall Y, Kaasa S, Österlund P, Cederholm T (2021). "Nutrition in Cancer Care: A Brief, Practical Guide With a Focus on Clinical Practice." JCO Oncol Pract. **17**:e992–e998. doi:10.1200/OP.20.00704.*
6. a, b, c, d, e, f, g, h, i, j, k, l, m*Rauh S, Antonuzzo A, Bossi P, Eckert R, Fallon M, Fröbe A, Gonella S, Giusti R, Lakatos G, Santini D, Villarini A (2018). "Nutrition in Patients With Cancer: A New Area for Medical Oncologists? A Practising Oncologist's Interdisciplinary Position Paper." ESMO Open. **3**:e000345. doi:10.1136/esmoopen-2018-000345.*

7. a, b, c, d, e, f, g, h, i, j, k, l, m, n, o, p, qSulosaari V, Beurskens J, Laviano A, Erickson N (2025). "Malnutrition Diagnosed via Global Leadership Initiative on Malnutrition (GLIM) Criteria - Association With Clinical Outcomes and Predictive Value: A Systematic Review of Systematic Reviews." Semin Oncol Nurs. **41**:151798. doi:10.1016/j.soncn.2024.151798.

8. a, b, cde Las Peñas R, Majem M, Perez-Altozano J, Virizuela JA, Cancer E, Diz P, Donnay O, Hurtado A, Jimenez-Fonseca P, Ocon MJ (2019). "SEOM Clinical Guidelines on Nutrition in Cancer Patients (2018)." Clin Transl Oncol. **21**:87–93. doi:10.1007/s12094-018-02009-3.

9. ^Anonymous (2025). "American Cancer Society Guideline for Diet and Physical Activity for Cancer Prevention." American Cancer Society-ASCO. https://www.cancer.org/cancer/risk-prevention/diet-physical-activity/acs-guidelines-nutrition-physical-activity-cancer-prevention.html.

10. ^Gill A, Givi B, Moore MG (2019). "AHNS Series - Do You Know Your Guidelines?: Assessment and Management of Malnutrition in Patients With Head and Neck Cancer: Review of the NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines)." Head Neck. **41**:577–583. doi:10.1002/hed.24866.

11. a, b, c, d, e, f, g, h, i, j, kCruz-Jentoft AJ, Bahat G, Bauer J, Boirie Y, Bruyère O, Cederholm T, Cooper C, Landi F, Rolland Y, Sayer AA, Schneider SM, Sieber CC, Topinkova E, Vandewoude M, Visser M, Zamboni M (2019). "Sarcopenia: Revised European Consensus on Definition and Diagnosis." Age Ageing. **48**:16–31. doi:10.1093/ageing/afy169.

12. a, b, c, d, e, f, g, h, i, j, k, l, m, n, oWang J, Tan S, Xu J, Li S, Yan M, Yang F, Huang Q, Zhang Z, Zhang Y, Han J, Liu H, Zhuang Q, Xi Q, Meng Q, Wu G (2023). "Development and Application of the Cancer Cachexia Staging Index for the Diagnosis and Staging of Cancer Cachexia." Nutrition. **114**:112114. doi:10.1016/j.nut.2023.112114.

13. a, b, c, d, e, f, g, h, i, j, k, l, m, nRoeland EJ, Bohlke K, Baracos VE, Smith TJ, Loprinzi CL, null n, Bruera E, Fabbro Ed, Dixon S, Fallon M, Herrstedt J, Lau H, Platek M, Rugo HS, Tan W (2023). "Cancer Cachexia: ASCO Guideline Rapid Recommendation Update." J Clin Oncol. **41**:4178–4179. doi:10.1200/JCO.23.01280.

14. a, b, c, d, e, f, g, h, i, jArgilés JM, López-Soriano FJ, Toledo M, Betancourt A, Serpe R, Busquets S (2011). "The Cachexia Score (CASCO): A New Tool for Staging Cachectic Cancer Patients." J Cachexia Sarcopenia Muscle. **2**:87–93. doi:10.1007/s13539-011-0027-5.

15. a, bArgilés JM, Betancourt A, Guàrdia-Olmos J, Peró-Cebollero M, López-Soriano FJ, Madeddu C, Serpe R, Busquets S (2017). "Validation of the CAchexia SCOre (CASCO). Staging Cancer Patients: The Use of miniCASCO as a Simplified Tool." Front Physiol. **8**.

16. a, b, c, d, e, fZhou T, Wang B, Liu H, Yang K, Thapa S, Zhang H, Li L, Yu S (2018). "Development and Validation of a Clinically Applicable Score to Classify Cachexia Stages in Advanced Cancer Patients." J Cachexia Sarcop

enia Muscle. ***9****:306–314. doi:10.1002/jcsm.12275.*

17. a, b, c*Ueshima J, Maeda K, Shimizu A, Nagano A, Ishida Y, Takeuchi T, Nonogaki T, Matsuyama R, Yamanaka Y, Murotani K, Mori N (2023). "Cachexia Staging Score Predicts Survival in Patients With Cancer Who Recei ve Palliative Care." Nutrition.* ***106****:111880. doi:10.1016/j.nut.2022.111880.*
18. a, b, c, d, e*Zhou J, Yang S, Liu T, Sun Y, Li S (2025). "Diagnostic Performance of GLIM and PG-SGA for Malnutri tion Assessment in Adult Cancer Patients: A Systematic Review and Meta-Analysis." BMC Cancer.* ***25****:765. do i:10.1186/s12885-025-13809-6.*
19. a, b, c, d, e, f*Orell HK, Pohju AK, Osterlund P, Schwab US, Ravasco P, Mäkitie A (2022). "GLIM in Diagnosing M alnutrition and Predicting Outcome in Ambulatory Patients With Head and Neck Cancer." Front Nutr.* ***9****:103 0619. doi:10.3389/fnut.2022.1030619.*
20. a, b, c, d, e, f, g*Mukherjee P, Augur ZM, Li M, Hill C, Greenwood B, Domin MA, Kondakci G, Narain NR, Kiebish MA, Bronson RT, Arismendi-Morillo G, Chinopoulos C, Seyfried TN (2019). "Therapeutic Benefit of Combini ng Calorie-Restricted Ketogenic Diet and Glutamine Targeting in Late-Stage Experimental Glioblastoma." Commun Biol.* ***2****:200. doi:10.1038/s42003-019-0455-x.*
21. a, b, c, d, e, f, g, h, i, j*Brandhorst S, Levine ME, Wei M, Shelehchi M, Morgan TE, Nayak KS, Dorff T, Hong K, Cri mmins EM, Cohen P, Longo VD (2024). "Fasting-Mimicking Diet Causes Hepatic and Blood Markers Chang es Indicating Reduced Biological Age and Disease Risk." Nat Commun.* ***15****:1309. doi:10.1038/s41467-024-452 60-9.*
22. a, b, c, d, e, f, g, h, i, j, k*Espinoza SE, Park S, Connolly G, Qi W, Zhang N, Semwal M, Li Y, Lauzon M, Salmon AB, Hsu W, Wei M, Musi N (2025). "Effect of Fasting-Mimicking Diet on Markers of Autophagy and Metabolic H ealth in Human Subjects." Geroscience. doi:10.1007/s11357-025-02035-4.*
23. a, b, c, d*de Groot S, Lugtenberg RT, Cohen D, Welters MJP, Ehsan I, Vreeswijk MPG, Smit VTHBM, de Graaf H, Heijns JB, Portielje JEA, van de Wouw AJ, Imholz ALT, Kessels LW, Vrijaldenhoven S, Baars A, Kranenbarg EM -K, Carpentier MD-d, Putter H, van der Hoeven JJM, Nortier JWR, Longo VD, Pijl H, Kroep JR, de Graaf H, Heij ns JB, Portielje JEA, van de Wouw AJ, Imholz ALT, Kessels LW, Vrijaldenhoven S, Baars A, Göker E, Pas AJM, H onkoop AH, van Leeuwen-Stok AE, Kroep JR, Dutch Breast Cancer Research G (2020). "Fasting Mimicking D iet as an Adjunct to Neoadjuvant Chemotherapy for Breast Cancer in the Multicentre Randomized Phase 2 DIRECT Trial." Nat Commun.* ***11****:3083. doi:10.1038/s41467-020-16138-3.*
24. a, b*Bahrami A, Haghighi S, Moghani MM, Khodakarim N, Hejazi E (2024). "Fasting Mimicking Diet During Neo-Adjuvant Chemotherapy in Breast Cancer Patients: A Randomized Controlled Trial Study." Front Nutr.* ***11****.*

25. ^a ^b Xue M, Zhang N, Dong X, Tian Q, Shen K, Wu B (2025). "The Feasibility and Safety of Fasting-Mimicking Diet in Breast Cancer Patients With Chemotherapy in China." Breast Cancer Res Treat. **213**:313–323. doi:10.1007/s10549-025-07756-w.

26. ^ Fatima G, Mehdi AA, Fedacko J, Hadi N, Magomedova A, Mehdi A (2025). "Fasting as Cancer Treatment: Myth or Breakthrough in Oncology." Cureus. **17**:e81395. doi:10.7759/cureus.81395.

27. ^ Maes J, Durieux V, Liebmann M, Salmon M, Preiser JC (2025). "Impact of Intermittent Fasting on Patients With Cancer Undergoing Chemotherapy and/or Targeted Therapies: A Systematic Review of the Literature." Support Care Cancer. **33**:863. doi:10.1007/s00520-025-09907-7.

28. ^a ^b Stringer EJ, Cloke RWG, Van der Meer L, Murphy RA, Macpherson NA, Lum JJ (2025). "The Clinical Impact of Time-Restricted Eating on Cancer: A Systematic Review." Nutr Rev. **83**:e1660–e1676. doi:10.1093/nutrit/nuae105.

29. ^a ^b ^c ^d Bragazzi NL, Briki W, Khabbache H, Rammouz I, Chamari K, Demaj T, Re TS, Zouhir M (2016). "Ramadan Fasting and Patients With Cancer: State-of-the-Art and Future Prospects." Front Oncol. **6**:27. doi:10.3389/fonc.2016.00027.

30. ^a ^b ^c ^d ^e ^f ^g Cederholm T, Jensen GL, Correia M, Gonzalez MC, Fukushima R, Higashiguchi T, Baptista G, Barazzoni R, Blaauw R, Coats AJS, Crivelli AN, Evans DC, Gramlich L, Fuchs-Tarlovsky V, Keller H, Llido L, Malone A, Mogensen KM, Morley JE, Muscaritoli M, Nyulasi I, Pirlich M, Pisprasert V, de van der Schueren MAE, Siltharm S, Singer P, Tappenden K, Velasco N, Waitzberg D, Yamwong P, Yu J, Van Gossum A, Compher C (2019). "GLIM Criteria for the Diagnosis of Malnutrition - A Consensus Report From the Global Clinical Nutrition Community." J Cachexia Sarcopenia Muscle. **10**:207–217. doi:10.1002/jcsm.12383.

31. ^a ^b ^c Rushin A, Shaikh A, Hardin C, Deleyrolle LP, Merritt ME (2025). "Metabolic Flux Analysis of Glioblastoma Neural Stem Cells Reveals Distinctive Metabolic Phenotypes in Ketogenic Conditions." Sci Rep. **15**:18736. doi:10.1038/s41598-025-02124-6.

32. ^a ^b ^c ^d Arends J, Strasser F, Gonella S, Solheim TS, Madeddu C, Ravasco P, Buonaccorso L, de van der Schueren MAE, Baldwin C, Chasen M, Ripamonti CI (2021). "Cancer Cachexia in Adult Patients: ESMO Clinical Practice Guidelines (☆)." ESMO Open. **6**:100092. doi:10.1016/j.esmoop.2021.100092.

33. ^a ^b Sanft T, Day AT, Ansbaugh SM, Ariza-Heredia EJ, Armenian S, Baker KS, Ballinger TJ, Cathcart-Rake EJ, Cohen SH, Evgeniou E, Fairman NP, Feliciano J, Flores TF, Friedman DL, Gabel NM, Goldman ME, Hill-Kayser CE, Hock K, Kline-Quiroz C, Koura D, Lee NK, McDonough AL, Mooney K, Moore HCF, Moryl N, Neuman H, Overholser L, Patel CG, Peterson LL, Pirl WF, Porpiglia A, Rogers LQ, Saidain A, Schapira L, Smith SK, Tenne

*L, Tevaarwerk A, Von Ah D, Yang EH, Zee P, McMillian N, Freedman-Cass D (2025). "NCCN Guidelines® Insights: Survivorship, Version 2.2025." J Natl Compr Canc Netw. **23**:208–217. doi:10.6004/jnccn.2025.0028.*

34. a, b *Bertazzoni G, Testa G, Tomasoni M, Mattavelli D, Del Bon F, Montalto N, Ferrari M, Andreoli M, Morello R, Sbalzer N, Vecchiati D, Piazza C, Nicolai P, Deganello A (2022). "The Enhanced Recovery After Surgery (ERAS) Protocol in Head and Neck Cancer: A Matched-Pair Analysis." Acta Otorhinolaryngol Ital. **42**:325–333. doi:10.14639/0392-100x-n2072.*

35. ^ *Viani K, Alves J, Damasco-Avila E, Murra MS, Schoeman J, Walters M, Ladas EJ (2025). "Consensus Recommendations for the Nutritional Management of Children With Cancer in Limited Resource Settings: A Report From the International Initiative for Pediatrics and Nutrition." Front Nutr. **12**:1605632. doi:10.3389/fnut.2025.1605632.*

36. a, b *Wiegert EVM, de Oliveira LC, Calixto-Lima L, Chaves GV, Silva Lopes MS, Peres WAF (2021). "New Cancer Cachexia Staging System for Use in Clinical Practice." Nutrition. **90**:111271. doi:10.1016/j.nut.2021.111271.*

37. a, b, c, d, e, f, g *Gascón-Ruiz M, Casas-Deza D, Marti-Pi M, Torres-Ramón I, Zapata-García M, Sesma A, Lambea J, Álvarez-Alejandro M, Quilez E, Isla D, Arbonés-Mainar JM (2022). "Diagnosis of Malnutrition According to GLIM Criteria Predicts Complications and 6-Month Survival in Cancer Outpatients." Biomedicines. **10**. doi:10.3390/biomedicines10092201.*

38. a, b, c, d, e, f, g *Murnane LC, Forsyth AK, Koukounaras J, Shaw K, King S, Brown WA, Mourtzakis M, Tierney AC, Burton PR (2023). "Malnutrition Defined by GLIM Criteria Identifies a Higher Incidence of Malnutrition and Is Associated With Pulmonary Complications After Oesophagogastric Cancer Surgery, Compared to ICD-10-Defined Malnutrition." J Surg Oncol. **128**:769–780. doi:10.1002/jso.27366.*

39. a, b, c *Ozorio GA, Ribeiro LMK, Santos BC, Bruzaca WFS, Rocha G, Marchi L, Santos FM, Alves de Almeida MMF, Kulcsar MAV, Junior UR, Correia M, Waitzberg DL (2023). "Exploring the Use of the GLIM Criteria to Diagnose Malnutrition in Cancer Inpatients." Nutrition. **116**:112195. doi:10.1016/j.nut.2023.112195.*

40. a, b *Colombo E, Righini M, Balendra V, Rustamli K, Garrone O, Ratti M, Ghidini M (2025). "Impact of Ketogenic and Fast-Mimicking Diet in Gastrointestinal Cancer Treatment." Front Oncol. **15**:1677509. doi:10.3389/fonc.2025.1677509.*

41. a, b, c *Lugtenberg RT, de Groot S, Kaptein AA, Fischer MJ, Kranenbarg EM-K, Carpentier MD-d, Cohen D, de Graaf H, Heijns JB, Portielje JEA, van de Wouw AJ, Imholz ALT, Kessels LW, Vrijaldenhoven S, Baars A, Fiocco M, van der Hoeven JJM, Gelderblom H, Longo VD, Pijl H, Kroep JR, on behalf of the Dutch Breast Cancer Research G (2021). "Quality of Life and Illness Perceptions in Patients With Breast Cancer Using a Fasting Mimic*

king Diet as an Adjunct to Neoadjuvant Chemotherapy in the Phase 2 DIRECT (BOOG 2013–14) Trial." Breast Cancer Res Treat. ***185****:741–758. doi:10.1007/s10549-020-05991-x.*

42. ^*Ligorio F, Fucà G, Provenzano L, Lobefaro R, Zanenga L, Vingiani A, Belfiore A, Lorenzoni A, Alessi A, Pruneri G, de Braud F, Vernieri C (2022). "Exceptional Tumour Responses to Fasting-Mimicking Diet Combined With Standard Anticancer Therapies: A Sub-Analysis of the NCT03340935 Trial." Eur J Cancer.* ***172****:300–310. doi:10.1016/j.ejca.2022.05.046.*
43. ^*Vernieri C, Ligorio F, Zattarin E, Rivoltini L, de Braud F (2020). "Fasting-Mimicking Diet Plus Chemotherapy in Breast Cancer Treatment." Nat Commun.* ***11****:4274. doi:10.1038/s41467-020-18194-1.*
44. ^*Bahrami A, Haghighi S, Moghani MM, Khodakarim N, Hejazi E (2024). "Fasting Mimicking Diet During Neo-Adjuvant Chemotherapy in Breast Cancer Patients: A Randomized Controlled Trial Study." Front Nutr.* ***11****: 1483707. doi:10.3389/fnut.2024.1483707.*
45. ^*Ligorio F, Vingiani A, Torelli T, Sposetti C, Drufuca L, Iannelli F, Zanenga L, Depretto C, Folli S, Scaperrotta G, Capri G, Bianchi GV, Ferraris C, Martelli G, Maugeri I, Provenzano L, Nichetti F, Agnelli L, Lobefaro R, Fucà G, Fotia G, Mariani L, Morelli D, Ladisa V, De Santis MC, Lozza L, Trecate G, Belfiore A, Brich S, Bertolotti A, Lorenzini D, Ficchì A, Martinetti A, Sottotetti E, Arata A, Corsetto P, Sorrentino L, Rediti M, Salvadori G, Minucci S, Foiani M, Apolone G, Pagani M, Pruneri G, de Braud F, Vernieri C (2025). "Early Downmodulation of Tumor Glycolysis Predicts Response to Fasting-Mimicking Diet in Triple-Negative Breast Cancer Patients." Cell Metab.* ***37****:330–344.e7. doi:10.1016/j.cmet.2024.11.004.*
46. a, b*Shams-White MM, Goldbaum AA, Agurs-Collins T, Czajkowski S, Herrick KA, Nebeling L, Reedy J, Riscuta G, Ross S, Sauter ER (2025). "Time-Restricted Eating and Cancer: Lessons Learned and Considerations for a Path Forward." J Natl Cancer Inst.* ***117****:1311–1315. doi:10.1093/jnci/djae331.*
47. a, b*Wen KY, Freedman J, Tafese A, Kelly W, Simone N (2026). "Fasting-Based Dietary Interventions in Cancer Patients and Survivors: A Scoping Review." Nutrients.* ***18****. doi:10.3390/nu18071035.*
48. ^*O'Donnell E, Shapiro Y, Comander A, Isakoff S, Moy B, Spring L, Wander S, Kuter I, Shin J, Specht M, Kournioti C, Hu B, Sullivan C, Winters L, Horick N, Peppercorn J (2022). "Pilot Study to Assess Prolonged Overnight Fasting in Breast Cancer Survivors (longfast)." Breast Cancer Res Treat.* ***193****:579–587. doi:10.1007/s10549-022-06594-4.*
49. ^*Stringer EJ, Cloke RWG, Van der Meer L, Murphy RA, Macpherson NA, Lum JJ (2025). "The Clinical Impact of Time-Restricted Eating on Cancer: A Systematic Review." Nutr Rev.* ***83****:e1660–e1676. doi:10.1093/nutrit/nuae105.*

50. ^Marinac CR, Nelson SH, Breen CI, Hartman SJ, Natarajan L, Pierce JP, Flatt SW, Sears DD, Patterson RE (2016). "Prolonged Nightly Fasting and Breast Cancer Prognosis." JAMA Oncol. 2:1049–1055. doi:10.1001/jamaoncol.2016.0164.

51. ^Tiwari S, Sapkota N, Han Z (2022). "Effect of Fasting on Cancer: A Narrative Review of Scientific Evidence." Cancer Sci. 113:3291–3302. doi:10.1111/cas.15492.

52. ^Alshammari K, Alhaidal HA, Alharbi R, Alrubaiaan A, Abdel-Razaq W, Alyousif G, Alkaiyat M (2023). "The Impact of Fasting the Holy Month of Ramadan on Colorectal Cancer Patients and Two Tumor Biomarkers: A Tertiary-Care Hospital Experience." Cureus. 15:e33920. doi:10.7759/cureus.33920.

53. ^Zeeneldin AA, Taha FM (2012). "Fasting Among Muslim Cancer Patients During the Holy Month of Ramadan." Ann Saudi Med. 32:243–249. doi:10.5144/0256-4947.2012.243.

54. ^Daşıran MF, AA, Göğüş F, et al (2021). "The Evaluation of Ramadan Fasting Experience of Patients With Gastric Cancer Who Underwent Curative Resection." J Contemp Med. 11:639–373. doi:10.16899/jcm.896651.

55. a, bDuraj T, Kalamian M, Zuccoli G, Maroon JC, D'Agostino DP, Scheck AC, Poff A, Winter SF, Hu J, Klement RJ, Hickson A, Lee DC, Cooper I, Kofler B, Schwartz KA, Phillips MCL, Champ CE, Zupec-Kania B, Tan-Shalaby J, Serfaty FM, Omene E, Arismendi-Morillo G, Kiebish M, Cheng R, El-Sakka AM, Pflueger A, Mathews EH, Worden D, Shi H, Cincione RI, Spinosa JP, Slocum AK, Iyikesici MS, Yanagisawa A, Pilkington GJ, Chaffee A, Abdel-Hadi W, Elsamman AK, Klein P, Hagihara K, Clemens Z, Yu GW, Evangeliou AE, Nathan JK, Smith K, Fortin D, Dietrich J, Mukherjee P, Seyfried TN (2024). "Clinical Research Framework Proposal for Ketogenic Metabolic Therapy in Glioblastoma." BMC Med. 22:578. doi:10.1186/s12916-024-03775-4.

56. a, bValerio J, Borro M, Proietti E, Pisciotta L, Olarinde IO, Fernandez Gomez M, Alvarez Pinzon AM (2024). "Systematic Review and Clinical Insights: The Role of the Ketogenic Diet in Managing Glioblastoma in Cancer Neuroscience." J Pers Med. 14. doi:10.3390/jpm14090929.

57. a, bDal Bello S, Valdemarin F, Martinuzzi D, Filippi F, Gigli GL, Valente M (2022). "Ketogenic Diet in the Treatment of Gliomas and Glioblastomas." Nutrients. 14. doi:10.3390/nu14183851.

58. ^Firdous J, Asif AE, Haris HM, Iqbal U, Khalid M, Zahid HF, Mubarika M, Abufatima IO, Amin MHJ, Hafeez S, Jabeen F, Shahzaib M, Fatima N, Kumar N, Sen S, Hassan A (2026). "Efficacy and Safety of Ketogenic Diet in Glioblastoma: An Updated Systematic Review and Meta-Analysis." Neurol Sci. 47. doi:10.1007/s10072-026-09035-y.

59. ^Zhao H, Jin H, Xian J, Zhang Z, Shi J, Bai X (2022). "Effect of Ketogenic Diets on Body Composition and Metabolic Parameters of Cancer Patients: A Systematic Review and Meta-Analysis." Nutrients. 14. doi:10.3390/nu14194192.

60. ^ a, b, c, d Champ CE, Palmer JD, Volek JS, Werner-Wasik M, Andrews DW, Evans JJ, Glass J, Kim L, Shi W (2014). "Targeting Metabolism With a Ketogenic Diet During the Treatment of Glioblastoma Multiforme." J Neuroo ncol. **117**:125–131. doi:10.1007/s11060-014-1362-0.

61. ^ a, b, c, d Amaral LJ, Gresham G, Kim S, Tighiouart M, Nelson TA, Welborn A, Lockshon L, Noorvash B, Rudnick JD, Irwin SA, Freedland SJ, Hu J (2025). "A Phase 1 Safety and Feasibility Trial of a Ketogenic Diet Plus Stand ard of Care for Patients With Recently Diagnosed Glioblastoma." Sci Rep. **15**:21064. doi:10.1038/s41598-025-06675-6.

62. ^ a, b, c, d Klein P, Tyrlikova I, Zuccoli G, Tyrlik A, Maroon JC (2020). "Treatment of Glioblastoma Multiforme W ith "Classic" 4:1 Ketogenic Diet Total Meal Replacement." Cancer Metab. **8**:24. doi:10.1186/s40170-020-00230-9.

63. ^ a, b Amanollahi A, Khazdouz M, Malekahmadi M, Klement RJ, Lee D, Khodabakhshi A (2023). "Effect of Keto genic Diets on Cardio-Metabolic Outcomes in Cancer Patients: A Systematic Review and Meta-Analysis of C ontrolled Clinical Trials." Nutr Cancer. **75**:95–111. doi:10.1080/01635581.2022.2117388.

64. ^ Salido-Bueno B, Gil-Hernandez E, Rueda-Ruzafa L, Gomez-Chica P, Roman P, Cardona D (2024). "Effects o f Ketogenic Diets on Cancer-Related Variables: A Systematic Review and Meta-Analysis of Randomised Con trolled Trials." Nutr Bull. **49**:264–277. doi:10.1111/nbu.12693.

65. ^ Kesarwani P, Kant S, Zhao Y, Miller CR, Chinnaiyan P (2022). "The Influence of the Ketogenic Diet on the I mmune Tolerant Microenvironment in Glioblastoma." Cancers (Basel). **14**. doi:10.3390/cancers14225550.

66. ^ Römer M, Dörfler J, Huebner J (2021). "The Use of Ketogenic Diets in Cancer Patients: A Systematic Revie w." Clin Exp Med. **21**:501–536. doi:10.1007/s10238-021-00710-2.

67. ^ Sremanakova J, Sowerbutts AM, Burden S (2018). "A Systematic Review of the Use of Ketogenic Diets in Ad ult Patients With Cancer." J Hum Nutr Diet. **31**:793–802. doi:10.1111/jhn.12587.

68. ^ a, b, c, d, e, f Nencioni A, Caffa I, Cortellino S, Longo VD (2018). "Fasting and Cancer: Molecular Mechanisms a nd Clinical Application." Nat Rev Cancer. **18**:707–719. doi:10.1038/s41568-018-0061-0.

69. ^ a, b, c, d, e, f Abdalla MMI, Bhatnagar P, Zulkaflee MHB, Mohammed Irfan AMS, Eid N (2026). "Intermittent F asting Enhances Cancer Therapy via Autophagy-Dependent and Independent Mechanisms: Evidence and I mplications." World J Clin Oncol. **17**:115289. doi:10.5306/wjco.v17.i2.115289.

70. ^ Chung HY, Park YK (2017). "Rationale, Feasibility and Acceptability of Ketogenic Diet for Cancer Treatmen t." J Cancer Prev. **22**:127–134. doi:10.15430/jcp.2017.22.3.127.

71. ^ Tan-Shalaby J (2017). "Ketogenic Diets and Cancer: Emerging Evidence." Fed Pract. **34**:37s–42s.

72. ^ a b c *Su Z, Liu Y, Xia Z, Rustgi AK, Gu W (2024). "An Unexpected Role for the Ketogenic Diet in Triggering Tumor Metastasis by Modulating BACH1-Mediated Transcription." Sci Adv.* ***10****:eadm9481. doi:10.1126/sciadv.adm9481.*

73. ^ *de Gruil N, Pijl H, van der Burg SH, Kroep JR (2022). "Short-Term Fasting Synergizes with Solid Cancer Therapy by Boosting Antitumor Immunity." Cancers (Basel).* ***14****. doi:10.3390/cancers14061390.*

74. ^ a b c *Maeyama M, Tanaka K, Nishihara M, Irino Y, Shinohara M, Nagashima H, Tanaka H, Nakamizo S, Hashiguchi M, Fujita Y, Kohta M, Kohmura E, Sasayama T (2021). "Metabolic Changes and Anti-Tumor Effects of a Ketogenic Diet Combined with Anti-Angiogenic Therapy in a Glioblastoma Mouse Model." Sci Rep.* ***11****:79. doi:10.1038/s41598-020-79465-x.*

75. ^ *Caire R, Bordo R, Zanconato F, Panciera T, Audoux E, Contessotto P, Fakiola M, Bason R, Romano O, Suli A, Battilana G, Marchionni M, Forcato M, Donzelli S, Dieci MV, Griguolo G, Carosi M, Fassan M, Guzzardo V, Dei Tos AP, Marsoni S, Wu P-H, Wirtz D, He S, Casali C, Volpin F, Blandino G, Tripodo C, Bicciato S, Guarneri V, Pagani M, Cordenonsi M, Piccolo S (2026). "A 3D Morphogenetic Blueprint for Metastatic Outgrowth in Breast Cancer." Cell. doi:10.1016/j.cell.2026.03.009.*

76. ^ *Matias-Guiu X, Raspollini MR, Kulka J, Ryska A, Al Dieri R, Schirmacher P (2025). "The Role of the Pathologist in the Design and Conducting of Biomarker-Driven Clinical Trials in Cancer: Position Paper of the European Society of Pathology." Virchows Arch.* ***486****:207–214. doi:10.1007/s00428-024-04005-1.*

77. ^ *Nagtegaal ID, West NP, van Krieken JH, Quirke P (2014). "Pathology is a Necessary and Informative Tool in Oncology Clinical Trials." J Pathol.* ***232****:185–189. doi:10.1002/path.4261.*

78. ^ a b c d *Cederholm T, Jensen GL, Correia M, Gonzalez MC, Fukushima R, Higashiguchi T, Baptista G, Barazzoni R, Blaauw R, Coats A, Crivelli A, Evans DC, Gramlich L, Fuchs-Tarlovsky V, Keller H, Llido L, Malone A, Mogensen KM, Morley JE, Muscaritoli M, Nyulasi I, Pirlich M, Pisprasert V, de van der Schueren MAE, Siltharm S, Singer P, Tappenden K, Velasco N, Waitzberg D, Yamwong P, Yu J, Van Gossum A, Compher C (2019). "GLIM Criteria for the Diagnosis of Malnutrition - A Consensus Report From the Global Clinical Nutrition Community." Clin Nutr.* ***38****:1–9. doi:10.1016/j.clnu.2018.08.002.*

79. ^ *Virizuela JA, Camblor-Álvarez M, Luengo-Pérez LM, Grande E, Álvarez-Hernández J, Sendrós-Madroño MJ, Jiménez-Fonseca P, Cervera-Peris M, Ocón-Bretón MJ (2018). "Nutritional Support and Parenteral Nutrition in Cancer Patients: An Expert Consensus Report." Clin Transl Oncol.* ***20****:619–629. doi:10.1007/s12094-017-1757-4.*

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