Cancer: Asking the Hard Questions Without Losing Sight of Effective Care

Human Health Strategies® — patient and family education

Educational guide. Not independently reviewed by an oncology clinician.

Evidence review: September 2026. Sources cited: 65. This is a general educational guide for patients and families, not a treatment recommendation. Cancer type, stage, biomarkers, prior treatment, overall health, and personal priorities can change the right decision. Do not start, stop, delay, or replace cancer treatment—or begin fasting, a restrictive diet, or an unapproved drug—on the basis of this guide. Children need decisions led by a pediatric oncology team.

Introduction

A cancer diagnosis can leave a family trying to make life-changing decisions while learning an unfamiliar language. “Response,” “survival benefit,” “standard of care,” and “promising research” can sound more definite than they are. Patients deserve understandable explanations of what a treatment is trying to achieve, how much benefit is likely, what the burdens are, and what remains uncertain. Asking for that explanation is compatible with respecting clinical expertise. The National Cancer Institute explicitly encourages questions about treatment goals, benefits, risks, research, and second opinions. [1]

Reasonable questions deserve careful answers, while hopeful explanations should not be presented as proven treatments. The practical aim is a better conversation with the oncology team that evaluates each option on its evidence, relevance, benefits, and risks.

Established care, emerging research, and widely shared personal accounts carry different levels of evidence. Mentioning an individual, idea, or story is not an endorsement or a claim that all approaches have equal support. Patients and families can bring these questions to their clinical team: What is known? What remains uncertain? Does this apply to my situation? What are the potential benefits, risks, and interactions? Is there a relevant clinical trial? Use the examples to start a discussion with the professionals responsible for your care, not to make treatment changes on your own.

1. The scale of cancer and childhood incidence trends

The American Cancer Society projects 2,114,850 new invasive cancer cases and 626,140 cancer deaths in the United States in 2026. Both figures are modeled forecasts, not completed 2026 counts. Separately, NCI estimates that 18.64 million people were living with a history of cancer in 2023; that prevalence figure is not the number newly diagnosed that year and does not mean everyone had active cancer. [2] [3]

Some of the most commonly diagnosed cancers illustrate the breadth of the problem:

Cancer type | Projected new U.S. cases in 2026

Prostate | 333,830

Female breast | 321,910

Lung and bronchus | 229,410

Colon and rectum | 158,850

Melanoma of the skin | 112,000

Leukemia, all ages | 67,790

Source: ACS 2026 estimates. These are new diagnoses, not prevalence or individual risk. Common basal-cell and squamous-cell skin cancers are not included in these cancer totals; most in-situ cancers are also excluded. [2]

Childhood cancer incidence

For 2026, ACS projects 9,680 diagnoses in children ages 0–14 and 5,660 in adolescents ages 15–19—15,340 across those two age groups. Leukemias, brain tumors, and lymphomas are major pediatric groups, but their frequency and biology differ by age. [2]

A national study published in 2026 examined ages 0–19 through 2022: overall age-standardized incidence increased about 0.94% annually during 2001–2016, then decreased about 0.96% annually during 2016–2022. Lymphoma incidence increased over the longer study period. A separate national report for ages 0–14 also found a recent overall decline, alongside increases in some subtypes. These findings do not establish a broad recent increase in aggressive pediatric cancers, while subtype-specific trends and families' concerns still warrant attention. [4] [5]

“Aggressive” is not a single surveillance category. Evaluating aggressiveness requires a defined cancer subtype, age range, location, time period, and measure—such as stage, grade, relapse, or mortality. Diagnosis counts alone cannot establish greater biological aggressiveness. The available pediatric incidence studies end in 2021 or 2022 and cannot establish a nationwide trend in 2024–2026. Changes in annual projected counts also should not be mistaken for observed changes in incidence rates. [4] [5] [2]

2. Start with the goal: what is this treatment trying to do?

Cancer is not one disease, and “cancer treatment” does not describe one level of benefit. A treatment may aim to cure, reduce the risk of recurrence after surgery, produce long-term control, slow progression, relieve symptoms, or preserve function. Some treatments offer substantial or durable benefit; in other settings, the average benefit can be modest and the burden substantial. It is misleading either to promise that treatment will work or to suggest cancer treatments generally buy only a few weeks. The question is what the evidence shows for this particular situation. [1] [6]

Ask the team to complete this sentence:

“For a person with my diagnosis and circumstances, this treatment is intended to ___, and the best evidence suggests ___.”

Follow with questions that are direct without being adversarial:

• Is the goal cure, reducing recurrence, longer life, symptom relief, or a combination?

• What is likely to happen with this option, with another reasonable option, and with supportive care alone where appropriate?

• What is the best realistic outcome, the typical outcome, and the worst realistic outcome?

• What burdens should I expect: fatigue, pain, neuropathy, infection risk, hospital time, cognitive effects, travel, and cost?

• How will we know whether it is helping, and when will we reassess?

• If it is not helping—or the burdens become unacceptable—what happens next?

These are conversation prompts, not a reason to refuse care. The right comparison must be selected with the treating team; an untreated comparison is not ethically or clinically appropriate for every curable or urgent cancer. NCI recommends discussing both likely benefit and harms in the context of a person's goals. [1] [6]

Quality of life is an outcome, not an afterthought

Someone may accept significant side effects for a realistic chance of cure. Someone else may prioritize being alert, staying at home, or attending a family event when the expected benefit is small. Neither priority should be assumed. Ask for measured quality-of-life results, serious adverse effects, treatment discontinuation, and likely time in clinics or hospitals—not just tumor measurements. Palliative care can support symptoms, coping, and decision-making alongside cancer-directed treatment; it is not synonymous with hospice or giving up. [6] [7]

3. Translate “benefit” into numbers you can actually use

Illustrative example—not a forecast for any cancer: Suppose a study finds that, at a specified time, 40 of 100 comparable patients die with treatment A and 30 of 100 with treatment B. The absolute reduction is 10 percentage points: 10 fewer deaths per 100 patients over that period. The relative reduction in the risk of death over that specified period is 25%, because 10 is one-quarter of 40. Both describe the same hypothetical result. “A 25% improvement” without the baseline and time period leaves out information needed to judge its meaning.

Ask: “Out of 100 people reasonably similar to me, how many have the outcome with each option, and over what period?” Also ask how precise the estimate is, whether it comes from a randomized trial, and how closely the study population resembles you. Group results cannot identify in advance exactly which individual will benefit. NCI encourages individualized discussion, and statistical guidance cautions against translating a single relative measure into an individual's chance of survival. [1] [8]

Three common misunderstandings deserve attention:

A hazard ratio is not an absolute probability. It summarizes a comparison of event hazards over follow-up. A hazard ratio of 0.75 does not mean “25 more people out of 100 survive,” nor does it automatically mean each person's probability of dying falls by 25%. Ask to see absolute survival at meaningful time points and the survival curves. [8]

Median survival is not a countdown. It is the time at which the study's estimated survival probability reaches 50%. If one arm has median survival of 12 months and another 15 months, it is not correct to promise each patient three extra months. Some may gain little; others may have a durable response. The distribution, uncertainty, subsequent treatments, and toxicity matter. [8]

Progression-free survival is not the same as overall survival or feeling better. Delaying progression can be valuable, but it does not automatically establish longer life or improved quality of life. In a 2018 analysis of 38 randomized trials, differences between treatment arms in median progression-free survival were not significantly associated at the trial level with differences in the global, physical, or emotional quality-of-life measures analyzed. The review had important follow-up and statistical-power limitations. Ask whether overall survival, symptoms, and quality of life were measured directly and what they showed. [7]

4. Biopsies: acknowledge the real risks without losing the diagnostic benefit

A biopsy can establish whether a lesion is cancer, identify its type, and provide information that changes treatment selection. Tissue can also support biomarker testing. Treating without adequate diagnostic information can mean treating the wrong disease or missing a useful option. The value of a biopsy therefore depends on what it can clarify and change—not simply on whether it is customary. [9] [10]

The broad claim that biopsies routinely cause cancer to spread is not supported. NCI says surgery-caused spread is extremely unlikely and notes that clinicians use precautions during both biopsy and surgery. Needle-tract seeding—tumor-cell implantation along a sampling route—has nevertheless been reported as a rare, context-specific complication. These points are not contradictory. [11] [10] [12]

The risk varies with tumor type, anatomy, biopsy route, technique, and subsequent treatment. In suspected sarcoma, for example, biopsy planning with the specialist team can matter for later surgery. Reports involving pancreatic sampling illustrate why route and tumor location matter. A figure from one organ or older technique should not be presented as the risk for every needle biopsy; finding microscopic cells is also not the same outcome as a clinically significant recurrence. [10] [12]

Other risks, such as bleeding, infection, pain, or organ-specific complications, need procedure-specific discussion. The fair comparison is the risks of this biopsy versus the risks of not obtaining the information, not “biopsy versus no risk.” [10] [1]

Questions worth asking:

• How might this test change treatment choices in my case?

• What would a positive, negative, or inconclusive result change?

• If immediate treatment would be unchanged, would it still help with diagnosis, prognosis, staging, future options, or trial eligibility?

• Is existing tissue sufficient? Would another sampling method answer the same question?

• Is needle-tract seeding a recognized concern for this particular tumor and route, and how do you reduce that risk?

• Should the team responsible for definitive treatment plan the biopsy?

• How urgent is the test, and would a second opinion create an unsafe delay?

These questions help clarify a recommendation. They do not establish that a biopsy should be skipped. Whether imaging, prior tissue, a blood-based test, or a new tissue sample can answer the question depends on the clinical setting. [9] [10]

5. Warburg, Seyfried, and the legitimate study of cancer metabolism

What is established?

Many cancer cells take up large amounts of glucose and produce lactate even when oxygen is available—a pattern called the Warburg effect, or aerobic glycolysis. This is an important biological observation. It is not, by itself, proof that every cancer is caused primarily by damaged mitochondria or that removing sugar from food will eliminate it. Cancer cells vary in mitochondrial function and reliance on oxidative phosphorylation—the process that couples respiration to production of the energy-carrying molecule ATP. Mitochondria can retain some functions without settling how effectively they produce ATP; their presence or oxygen consumption alone does not resolve that question. Cancer metabolism must be evaluated in the specific biological setting. [13] [14] [15]

Otto Warburg was a major scientist and a Nobel laureate. A useful historical distinction: his 1931 Nobel Prize recognized his discovery of the nature and mode of action of the respiratory enzyme, not proof of a cancer diet or validation of all his later cancer hypotheses. [16]

What does Thomas N. Seyfried argue?

Boston College biology professor Thomas N. Seyfried has argued that impaired mitochondrial respiration is central to cancer and that many genetic changes are downstream consequences. His research and writings explore metabolic approaches, including dietary strategies and metabolic targeting. This is a legitimate hypothesis to examine, but describing his group as having conclusively validated a universal cause of cancer would overstate the evidence. His hypothesis paper relies heavily on experimental models; it is not a controlled demonstration of improved survival across human cancers. [17]

His 2025 review develops this argument specifically around impaired ATP-producing oxidative phosphorylation and compensatory fermentation. It is a mechanistic interpretation of research, not a new clinical trial. In a 2019 experimental study, a calorie-restricted ketogenic diet combined with the glutamine antagonist DON reduced tumor burden and improved survival in two mouse glioblastoma models. That combination is substantive preclinical evidence worth studying, but it does not establish benefit or safety in patients. A metabolic intervention could prove useful without establishing a universal mitochondrial cause of cancer; the causal hypothesis and the treatment question require separate tests. [15] [18]

In the Boston College video “Supporting evidence,” uploaded in May 2009, Seyfried describes collaborative experimental research using animal tumor models at Boston College and biochemical analysis at Washington University in St. Louis. He interprets mitochondrial lipid abnormalities as support for Warburg's theory that impaired respiration is central to cancer. This is a scientific argument grounded in experimental work, not simply a dietary opinion. The separate questions are how broadly that explanation applies and which interventions improve human outcomes. [19]

In a second interview, “Rethinking Cancer: The Metabolic Theory,” uploaded April 11, 2026, Seyfried continues to argue that mitochondrial dysfunction is primary and that many genetic changes are downstream. The upload date does not establish the recording date. The interview explains his position; it is not itself a controlled treatment study. [20]

Genetics and metabolism interact

Patients with the same broad diagnosis, such as breast cancer, can have similar or very different molecular findings, and testing may find none of the commonly tested or currently actionable alterations. This variation—called tumor heterogeneity—is well recognized. “No marker found” means no relevant finding on the tests performed, not proof that the cancer has no genetic alterations. Receptor markers are also not interchangeable with gene mutations: for example, triple-negative breast cancer lacks estrogen and progesterone receptors and HER2 overexpression, but is not thereby a cancer without genetic changes. [9] [21]

Seyfried interprets the diversity of genetic findings, including the lack of a single shared mutation across cancers, as part of the argument that many genetic abnormalities are downstream consequences of mitochondrial metabolic dysfunction rather than the primary initiating cause. This is an important part of his position and deserves to be represented clearly. However, heterogeneity alone does not establish the direction of causation: different genetic changes can affect overlapping pathways, and experimental evidence supports causal roles for some driver alterations. The observation of variation is established; the broader conclusion that cancer-associated genetic changes are generally downstream remains disputed. [15] [22] [23]

Modern cancer biology includes genetics, epigenetics, metabolism, immunity, and the tumor environment. Genetic changes can alter metabolism; metabolic conditions can influence cell behavior. Calling cancer a genetic disease does not mean every case is inherited: mutations can arise during life, including through environmental exposures and errors in cell division. Research has identified and experimentally supported numerous cancer driver genes. [23] [22] [13]

Metabolism is also not excluded from conventional treatment. Some established cancer medicines exploit metabolic dependencies, and newer treatments target selected metabolic abnormalities. Their success in particular settings supports studying metabolism; it does not establish a universal dietary treatment or invalidate genetically targeted therapies. [14]

The useful question is:

“Is there a clinically supported or trial-based way to target a metabolic vulnerability in my particular cancer, alongside appropriate care?”

6. Ketogenic diets, fasting, and metabolic inhibitors: what has actually been shown in people?

Different interventions, different questions

These approaches should not be treated as interchangeable:

• Ketogenic diets substantially restrict carbohydrate intake to promote ketone production; they need not restrict total calories.

• Short-term fasting around treatment temporarily restricts food intake near a treatment cycle.

• Fasting-mimicking diets provide a formulated, energy-restricted diet intended to reproduce some fasting-related changes while allowing food.

• Time-restricted eating changes the daily eating window, without necessarily prescribing calorie restriction or a ketogenic diet.

• Prolonged water-only fasting involves a longer period without food and requires particular attention to screening, monitoring, medication management, and refeeding.

Differences in food composition, duration, energy intake, cancer type, and concurrent therapy can change both outcomes and risks. Evidence for one intervention is not evidence for all five. [24] [25] [26]

A paper presenting the argument for ketogenic research

“Ketogenic Diets and Cancer: Emerging Evidence,” by Jocelyn Tan-Shalaby, MD, published in Federal Practitioner in February 2017, presents the rationale for studying ketogenic diets as an addition to cancer care. This is a narrative review, not a randomized trial and not a paper authored by Seyfried. It discusses glucose dependence, insulin-related growth signaling, mitochondrial function, animal experiments, case reports, and small human studies. It is a useful source for understanding the argument in favor of metabolic approaches. [27]

The review describes encouraging observations, including stable disease, changes in tumor glucose uptake, and responses in some reports. These observations justify further investigation, but the human studies were generally small, nonrandomized, and focused on safety or feasibility. Concurrent cancer treatment, differences among patients, and difficulty sustaining the diet limit conclusions about what caused an outcome. A PET glucose-uptake change is not equivalent to a demonstrated survival benefit. The review also includes weight loss and adherence difficulties; its favorable safety conclusion should not be translated into “safe for every cancer patient.” [27]

The author's conclusion is about potentially combining the diet with chemotherapy or radiotherapy and conducting more research, not replacing standard treatment. Its broad statements that ketones do not benefit cancer cells and that ketogenic diets selectively starve tumors should be read as the review's mechanistic framing, not universal clinical findings. Research documents ketone utilization in some cancer models; this challenges a universal claim without proving that ketogenic diets help or harm every patient. A 2017 review also cannot settle the evidence available today. [27] [28]

What subsequent clinical evidence can—and cannot—tell us

Laboratory findings can justify a clinical trial. They cannot tell a patient whether an intervention will improve survival, preserve muscle, interact safely with treatment, or be practical to sustain. A change in glucose or ketones is not the same outcome as tumor control or longer life. The evidence reviewed spans small trials, case reports, reviews, and larger studies still underway; their conclusions must not be treated as interchangeable. [29] [30] [31]

Evidence | What it contributes | What it does not establish

Human ketogenic-diet systematic review, 2021: 39 studies, 770 patients | Documents feasibility, adherence problems, weight loss, and a heterogeneous, generally weak evidence base | Conclusive antitumor or overall-survival benefit. [29]

ERGO2 randomized trial, 2020: 50 patients with recurrent malignant glioma | Tested a short calorie-restricted ketogenic/fasting intervention alongside reirradiation | No statistically significant improvement in its primary six-month progression-free endpoint or overall survival; not a definitive test of every possible metabolic strategy. [32]

Controlled-study meta-analysis, 2025 | Examines metabolic, body-composition, and other outcomes in newer controlled reports | Changes in such measures should not be presented as established survival benefit. [30]

DIRECT randomized phase II trial, 2020: 131 patients with HER2-negative breast cancer | A fasting-mimicking diet with neoadjuvant chemotherapy produced an encouraging radiological-response signal | No significant improvement in pathological complete response or severe toxicity; only 20% adhered through all chemotherapy cycles. Dexamethasone use also differed between groups. [33]

Randomized phase II pancreatic cancer trial, 2026: 36 randomized, 32 evaluable patients | With chemotherapy in both arms, a medically supervised ketogenic diet produced median progression-free survival of 8.5 versus 6.2 months and overall survival of 13.7 versus 10.2 months | A preliminary signal, not confirmed survival benefit: the small screening trial used a one-sided significance threshold of 0.20, and both hazard-ratio confidence intervals included no effect. [34]

Ovarian cancer randomized pilot, ASCO 2026 conference abstract: 36 patients completing three chemotherapy cycles | Met its primary insulin endpoint; reported a favorable pathological-response score in 10/17 versus 3/17 surgical patients and median progression-free survival of 38 versus 24 months | Not a full published trial report. With median follow-up of 18 months and no hazard ratio, confidence intervals, or event counts provided in the abstract, the survival estimates require caution. Progression-free does not mean cancer-free. [35]

DIET2TREAT registry, NCT05708352: planned enrollment 170 | Randomizes newly diagnosed glioblastoma patients to ketogenic versus usual diet, with standard treatment in both groups; primary endpoint is overall survival | The August 5, 2026 registry update listed recruiting, estimated primary completion September 2028, and no posted results. Registration is not evidence of benefit. [36]

Human studies include encouraging response and survival signals in selected settings, alongside negative or inconclusive findings. Larger, well-controlled trials are needed to determine which patients benefit, by how much, and with what risks. The evidence does not establish that ketogenic diets or fasting cure cancer or reliably improve survival across cancers. A small negative trial does not answer every future question, but an encouraging pilot or case report is not definitive evidence either. [29] [32] [34] [35]

In the pancreatic trial, the progression-free-survival hazard ratio was 0.53 (95% confidence interval 0.21–1.37), and the overall-survival hazard ratio was 0.58 (0.25–1.37). These wide intervals include benefit as well as no benefit or harm. The trial's permissive statistical threshold was intended to identify an intervention worth testing in a larger study, not to provide confirmatory evidence. Quality-of-life scores did not significantly decline in the ketogenic arm, but improved more in the comparison arm; the study should not be described as demonstrating superior quality of life with the diet. [34]

What the newer experimental research contributes

The biological rationale is more specific than simply “removing sugar.” Researchers test how dietary changes interact with drugs, tumor adaptation, and immunity:

• Pancreatic cancer: A 2024 study combined a ketogenic diet with the experimental drug tomivosertib (eFT508) to restrain tumor growth in mice. A separate mouse study found that ketogenic diet alone did not slow pancreatic tumor growth, while its combination with chemotherapy improved tumor control and survival. These are combination-treatment findings, not evidence that diet alone works in patients. [37] [38]

• Prostate cancer: A 2024 mouse study found that a cyclic ketogenic diet or a ketone-promoting intervention could improve responses to immune checkpoint blockade in selected models. Immune and metabolic effects—not merely depriving tumors of fuel—were implicated. [39]

• Immune-cell function: A 2024 study showed fasting-related changes in natural killer cells that improved antitumor activity in mice. Its human component involved healthy fasting donors, not a cancer-patient treatment trial. It supports a mechanism to investigate, not a demonstrated clinical cancer benefit. [40]

The 2023 review by Blaževitš, Di Tano, and Longo discusses protection of normal cells, sensitization of cancer cells, immune effects, and metabolic adaptations that drugs might target. The source was available through its abstract, highlights, and section excerpts and synthesizes earlier work rather than adding another clinical trial. Longo disclosed equity in L-Nutra, a medical-food company; that interest should be considered without treating it as a reason to disregard the findings. [24]

Symptoms, treatment tolerance, and quality of life

Cancer control is not the only meaningful outcome. Better function, fewer symptoms, or easier treatment could matter even without longer survival. These benefits must be measured rather than assumed. A 2023 systematic review and meta-analysis did not find a significant reduction in chemotherapy adverse effects or neutropenia with fasting; heterogeneous regimens and a small evidence base limit certainty. [41]

Cedars-Sinai's 2024 article describes symptom improvements reported by some patients and the rationale for DIET2TREAT and other dietary trials. Those observations warrant investigation but do not establish that diet caused the improvement, shrank tumors, or extended survival. Ohio State's July 2026 article describes research on fasting around chemotherapy in gynecologic cancers, including quality of life. It reports no completed trial results, sample sizes, or quantitative benefits. Its suggestion about reduced digestive energy demands is a hypothesis, not an established mechanism of improved chemotherapy tolerance. Institutional articles about research should not be counted as additional positive trials. [25] [42]

TrueNorth: clinical experience and preliminary cancer reports

TrueNorth Health Center, founded in 1984, combines medically supervised water-only fasting, supervised refeeding, and whole-plant-food nutrition without added salt, oil, or sugar (SOS-free). Founder Alan Goldhamer, DC, has decades of supervised-fasting, research, and clinician-training experience. His official biography reports supervision of more than 25,000 patients' fasts; that is an attributed experience claim, not a controlled measure of effectiveness. [43] [44]

A 2024 case series reported reductions in lymph-node size and metabolic activity in three patients with low-grade follicular lymphoma after fasting and dietary intervention. The observations deserve investigation, but the absence of a comparison group, patient selection, concurrent diet, and variable disease course prevent attributing outcomes to fasting or generalizing to other cancers. Goldhamer disclosed ownership of the center. [45]

TrueNorth's blood-pressure findings concern metabolic and cardiovascular health, not demonstrated cancer efficacy or longer human lifespan. Its retrospective safety review covered 768 supervised visits: most recorded adverse events were mild, but severe events occurred and two events required hospitalization. These findings concern screened patients in a supervised setting, not home fasting. Patients with cancer need oncology coordination and attention to nutrition, muscle preservation, medications, electrolyte changes, and refeeding risks. [46] [26] [47]

Nutrition and safety remain part of the treatment question

A caution from Goldhamer himself: In an interview excerpt documented by transcript screenshots spanning approximately 34:34–35:48, he emphasizes contraindications, prescriber involvement, screening, appropriate laboratory monitoring, and coordination with a local doctor even for selected remotely supervised patients. He also describes restrictions on work, driving, and exercise during prolonged fasting and raises concern about lean-tissue loss. These are attributed comments about his fasting approach, not universal instructions for every dietary intervention or independent proof of safety. The full interview and its publication date were not available for verification. [48]

Do not stop, taper, skip, or change prescribed medication to make fasting possible. Goldhamer's comments about medication withdrawal must not be read as advice to discontinue necessary treatment. Any medication decision belongs to the prescribing clinician, with the oncology team involved; fasting may be inappropriate when essential treatment or nutritional needs cannot safely be accommodated. A modified fasting regimen is not automatically safe either. This guide does not reproduce the interview's calorie prescription or offer a home-fasting protocol. Rest and hydration alone do not establish safety. [48] [47] [26]

There are important practical limits. Cancer and its treatment can cause malnutrition and muscle loss. Restrictive diets or fasting can work against nutrition goals, particularly when someone is losing weight or struggling to eat. ASCO found insufficient evidence to recommend specific restrictive diets or fasting for cancer-control benefits during treatment; ESPEN emphasizes nutrition adequacy and cautions against fasting without established benefit. These guidelines are older than some recent studies, so their evidence cutoffs must be considered alongside newer trial results. [49] [47] [50]

“Starving cancer” is an appealing shorthand, but it can hide two problems: tumors differ in fuel use, and a patient can become nutritionally depleted without achieving useful tumor control. Do not pursue a target glucose or ketone number as a substitute for clinical outcomes. Discuss experimental metabolic drugs through an oncology team and a legitimate trial—not through a self-designed combination. [13] [14] [47]

Ask: Is there a trial for my tumor type and treatment stage? Does it add to standard care or replace something effective? What is the primary endpoint? Who monitors nutrition, muscle, adverse effects, and drug interactions? What would cause the study team to stop the intervention?

7. Joe Tippens, Keytruda, and fenbendazole: what his experience can and cannot show

Joe Tippens' widely shared account describes surviving extensively metastatic small-cell lung cancer while using a fenbendazole-containing regimen and participating in an immunotherapy clinical trial. Public reporting identifies the trial treatment as pembrolizumab (Keytruda). This concurrent treatment is central to understanding his story, not a minor detail. In a February 2022 update, Tippens reported reaching five years with no evidence of disease (NED). These are accounts of his treatment and outcome, not independently audited medical records; NED means no cancer was detected on the assessments performed, not proof that it can never return. [51] [52]

Tippens' interpretation: he recounts being told that his response was exceptional among comparable trial participants and interprets that apparent difference as evidence that his fenbendazole-containing regimen was responsible. However, the account of other participants' outcomes is his recollection, not a published, independently verified comparison of the same patients, treatments, and follow-up. His exact trial identifier and the claim that no other participant achieved a durable complete response have not been independently verified. [51]

A remarkable, long-lasting response deserves attention, but being an exceptional responder does not identify which treatment caused it. Responses to the same cancer treatment can differ substantially. Because he received multiple interventions, his experience cannot establish “fenbendazole cured him,” exclude a contribution from Keytruda, or establish that immunotherapy alone caused his remission. The American Cancer Society likewise highlights the simultaneous immunotherapy trial when discussing this story. His explanation is a hypothesis to investigate, not a demonstrated treatment effect. [51] [52] [53]

Fenbendazole is a veterinary antiparasitic; it is not FDA-approved for human use or cancer treatment. Experimental findings do not establish human effectiveness, dose, interactions, or safety. At the evidence-review cutoff, no controlled human oncology trial establishing benefit or human fenbendazole oncology intervention in ClinicalTrials.gov had been identified. This does not establish the status of every registry worldwide. Studies of related drugs such as mebendazole or oxfendazole are not interchangeable with fenbendazole studies. [53] [54]

There are published reports of drug-induced liver injury associated with self-administered fenbendazole, including severe cases. Case reports cannot tell us how often harm occurs, but they are a meaningful warning—not evidence that the drug is harmless because it is used in animals. Liver injury can also complicate interpretation of cancer-treatment toxicity. [55] [56] [57]

An additional issue matters when evaluating internet citations: a 2025 three-patient fenbendazole cancer case series was retracted in January 2026. The publisher cited an undeclared conflict of interest involving services related to the subject and concerns about interpretation and recommendation. The notice did not establish that the clinical observations were fabricated; nevertheless, the retracted paper should not be offered as dependable evidence of efficacy. [58]

Mebendazole: a related human medicine with actual human cancer trials

Mebendazole is the correct spelling. It is a distinct drug in the same broad antiparasitic family as fenbendazole, used to treat parasitic infections in people—not simply a human formulation of fenbendazole. Its cancer use remains investigational. Published human trials exist, so it would be inaccurate to describe all research on this drug family as laboratory or animal work. These studies do not establish that fenbendazole works or that the two drugs are interchangeable. [59] [60]

• Metastatic colorectal cancer, 2022: a randomized, double-blind study of 40 patients added mebendazole or placebo to bevacizumab plus FOLFOX4 chemotherapy. It reported an objective response rate of 65% versus 10% and median progression-free survival of 9.25 versus 3 months in favor of mebendazole. This is a positive clinical signal worth discussing, but the trial was small, retrospectively registered, and tested an addition to established treatment—not a stand-alone cure. Independent replication is needed. [59]

• Advanced gastrointestinal cancers, 2021: a small, uncontrolled phase 2a study enrolled 11 patients, of whom 10 started treatment. All eight patients evaluable at eight weeks had progressive disease. Difficulty reaching the intended blood concentration complicates interpretation, but this study did not show an anticancer benefit. [60]

• Recurrent glioblastoma, 2022: a randomized phase II study of 88 adults compared two chemotherapy combinations, both containing mebendazole. Neither met the prespecified nine-month survival benchmark. Because there was no mebendazole-free comparison group, it could not isolate mebendazole's contribution. [61]

Bottom line: human mebendazole research is real but limited and mixed. It supports further investigation, not a proven cancer cure or a reason to replace established treatment. The retracted fenbendazole case series discussed above is a different publication from these mebendazole trials. Ask the oncology team whether a relevant, properly monitored trial is available; approval for treating parasites does not establish safety or effectiveness for cancer treatment. [59] [60] [61] [58]

The ethical response is not ridicule. It is transparent discussion: “I encountered this story. What evidence would show whether the approach works, what harms are known, and is there a properly monitored study relevant to me?” Do not self-administer veterinary products. Tell the oncology team and pharmacist about all products already being used; withholding that information makes safe care harder. [53] [57]

8. Sugar, chemotherapy snacks, and the difference between nutrition and a cancer treatment

Seeing sweet drinks, desserts, or highly processed snacks in a chemotherapy unit can reasonably prompt questions. But a snack tray does not establish the institution's motives or show that dietary sugar restriction would treat the cancer. The NCI states that although cancer cells consume more glucose, evidence does not show that simply stopping dietary sugar makes a tumor shrink or disappear. That is different from saying dietary quality, glucose management, and long-term health do not matter. [11] [62]

During treatment, a person may have nausea, mouth sores, altered taste, difficulty swallowing, or very little appetite. A tolerable food can sometimes be more useful than a nutritionally ideal food that the person cannot eat. NCI guidance recognizes that some patients need extra protein and calories to maintain strength and reduce malnutrition. That can explain why calorie-dense foods are offered; it cannot tell us why a specific hospital chose a particular menu or whether that choice suited every patient. [63]

At the same time, patients should be able to request alternatives and individualized advice. Someone maintaining weight may have different needs from someone losing muscle, and a person with diabetes or steroid-related high glucose may need coordinated nutrition and glucose management. Cancer cachexia is more than not eating enough: inflammation and metabolic changes contribute, and food alone may not reverse it. Restricting calories without recognizing this context can be harmful. [50] [64] [47]

A constructive question is:

“What nutrition goal are we trying to meet right now—maintaining weight, preserving muscle, controlling glucose, managing side effects, or something else? Could an oncology registered dietitian help us choose foods that fit?”

Ask for options with more protein or less added sugar when appropriate, without assuming every carbohydrate is harmful. This guide does not endorse a junk-food diet, nor does it endorse fasting or a restrictive diet as cancer treatment. It supports an individualized plan developed with the oncology team. [63] [62] [47]

9. How to bring emerging research into the appointment

Bring a paper, not just a headline. Ask whether the claim comes from cells, animals, a case report, an uncontrolled patient series, or a randomized comparison. Check the cancer type, stage, biomarkers, concurrent treatments, comparison group, and outcomes. Look for harms, missing data, withdrawals, conflicts of interest, and retractions. A mechanism can be real while the proposed treatment fails; a person's improvement can be real while the explanation remains uncertain. The metabolic and fenbendazole examples above show why those distinctions matter. [32] [29] [53] [58]

Then turn the discussion toward action:

• “Does this finding apply to my exact cancer and treatment stage?”

• “Is there biomarker testing or a pathology review that could change our options?”

• “Is this treatment supported, uncertain, not applicable, or known to be unsafe—and why?”

• “Is there a legitimate clinical trial, and can you help me contact the study team?”

• “Would a second opinion at a center with expertise in this cancer be useful?”

• “How much time can we safely take to investigate?”

• “What supportive and palliative care can help me now, regardless of the treatment choice?”

NCI encourages questions about trials, specialists, treatment effects, and second opinions. An appropriate answer may be “not for this tumor,” “not yet proven,” or “worth evaluating in a trial.” Ask for an explanation rather than a promise. [1] [9] [6]

Peer discussion and evaluating community resources

Keto for Cancer community — https://www.skool.com/ketoforcancer

For patients and caregivers interested in discussing information and arguments supporting Seyfried's position, the video promotes this group as a place to connect and share information. A discussion resource is not an endorsement of the group's claims, services, or treatment advice, and the video does not establish that Seyfried or Boston College operates or medically supervises it. The group's current membership terms, fees, moderation, and privacy practices have not been independently verified. [65]

Peer discussion can help people formulate questions and find papers to bring to their clinicians. Personal accounts are not controlled evidence of treatment benefit. Before posting, review who can see the information and avoid sharing identifying medical records. Discuss proposed diet, supplement, or treatment changes with the oncology team and an oncology dietitian rather than relying on group advice alone.

10. A one-page conversation checklist

Before the visit: Bring the diagnosis and pathology report, stage and biomarker information if available, a current medication/supplement list, and your three highest-priority concerns.

During the visit, ask:

1. What are we treating, and how certain are we of the diagnosis?
2. What is the goal of each option?
3. What is the absolute benefit, over what time period, compared with what?
4. Do the studies show longer life, better symptoms, better quality of life, or mainly a test/scan change?
5. What burdens or serious harms are most relevant to me?
6. How might this proposed test or biopsy change treatment choices or other important care decisions?
7. Are pathology review, biomarkers, a second opinion, or a clinical trial relevant?
8. What nutrition plan will protect strength and fit my treatment and health conditions?
9. How can we evaluate the emerging research I brought without delaying effective care?
10. When will we reassess, and what is the plan if the treatment is not helping?

Before leaving: Repeat the plan in your own words. Confirm who to call about symptoms, test results, side effects, and new questions. These prompts adapt the patient-centered approach encouraged by NCI; they are not a protocol for any particular cancer. [1]

The central message

Hope and scrutiny belong in the same conversation. A patient can value established treatment and still ask difficult questions about benefit, burden, diagnosis, nutrition, and research. Taking ownership means understanding the decisions, making personal priorities explicit, and asking for qualified help—not being expected to become an oncologist or feeling responsible for controlling every outcome.

The standard for a new approach should be neither its popularity nor its distance from the mainstream. It should be the quality of the evidence, its relevance to the particular cancer, its risks, and whether it helps people live longer or better.

Evidence limitations and review status

This is a researched educational guide, not an oncology-reviewed clinical guideline or an exhaustive systematic review. Population statistics do not predict an individual's outcome. Annual forecasts, observed incidence, prevalence, and mortality use different years and methods; the pediatric data cannot settle claims about periods not yet observed.

Foundational sources from 2009–2021 remain useful for history, methods, and individual trials, but are not presented as current comprehensive evidence on their own. They are considered alongside newer reviews and studies. Several sources were available as abstracts or registry summaries rather than full patient-level data; uncertain details are not treated as established facts.

The Boston College video and newer interview are represented through their available descriptions and transcripts, with screenshots providing additional context. Neither interview is clinical efficacy evidence. The community listing comes from the video's promotion, not private group content. Tippens' personal records and exact trial protocol were not available; no causal conclusion can be made. No single biopsy-risk percentage applies across cancers. Drug doses, veterinary protocols, fasting schedules, and glucose/ketone targets require qualified clinical guidance and are not provided here.

Editorial evidence review: September 20, 2026. Independent review by an oncology clinician and an oncology registered dietitian has not been completed. Readers should discuss treatment and nutrition decisions with their own qualified care team. Emerging-treatment findings and trial status can change; the dated evidence presented here is not a substitute for current individualized medical advice.

Sources

Each source is labeled by its publication or study type, not a numerical credibility tier. A case report, animal study, randomized trial, guideline, and institutional news article answer different questions and should not be treated as equivalent evidence. A registry describes a study; it does not establish benefit. Publication dates and access or data dates are shown where available. Labels also identify conference abstracts and sources reviewed only through excerpts or summaries.

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