

A message from Dr. Seyfried:

PLEASE NOTE: *I am not a physician and cannot treat cancer patients or give medical advice.*

Please reply to my email, acknowledging that the information I provide here or elsewhere is for educational purposes only and not to be misconstrued as medical advice.

It is my deepest desire that this information will guide you or your loved one towards a path of healing and recovery.

AI CAUTIONS: *AI is now being used to create unauthorized and misleading YouTube videos using cloned voices. My only authorized videos are full presentations at scientific conferences or podcasts with back-and-forth exchanges with the interviewer. AI has already created “press-pulse protocols” attributed to me, and diet plans attributed to my colleagues. Again, these are not authorized and contain factual errors. This misrepresentation threatens to undermine the important work we are doing. I am also aware that there are several unauthorized summaries of my book; AI will undoubtedly result in more of these.*

Sincerely,
Thomas N. Seyfried, Ph.D.
Professor of Biology

Cancer Information Kit

What are Metabolic Therapies?

Metabolic therapies are non-toxic strategies that target the fundamental metabolic defect that all major cancer cells share - their dependence on fermentable fuels (glucose and glutamine) for growth. This abnormality is a universal trait of all cancer cells, regardless of the genetic mutations that they might develop or their tissue of origin. This applies to both solid and blood-borne cancers.

Implementation of metabolic therapy needs to be individualized based on the patient's medical and metabolic history, as assessed through a broad range of imaging tests and blood biomarkers.

In my opinion, metabolic therapy can be effective in *managing cancer progression* if done correctly and under the supervision of an appropriately trained and knowledgeable physician, physician's assistant, nurse, or nutritionist. This information package includes links to some of the professionals who are trained or knowledgeable in implementing these ketogenic metabolic therapies (KMT) for cancer management. Unfortunately, most conventional physicians and oncologists are unfamiliar with the concepts and implementation of ketogenic metabolic therapy. This lack of familiarity places much of the burden of research on the patients themselves. Although some of the scientific articles referenced here can be challenging to understand, Google searches and the use of reputable AI summaries (such as Gemini) can help you to understand the scientific words and concepts if necessary.

You can learn more about the science behind this concept here: [Cancer as a Metabolic Disease \(Book by Dr. Seyfried\)](#)

The price of my book is set by the publisher and Amazon. I've attached a 20% discount coupon.

Implementing Ketogenic Metabolic Therapies (KMT)

The foundation of KMT is a well-formulated ketogenic diet. This is a pattern of eating that reduces carbohydrate intake but maintains nutrients to nourish healthy cells, putting you into a state of therapeutic ketosis. Maintaining low and stable blood sugar and low insulin levels support the production of ketone bodies. I recommend that persons considering KMT for cancer management read Miriam Kalamian's book, [Keto for Cancer](#). She includes detail on specific strategies while also addressing caveats and concerns. Miriam works one-to-one with individuals with primary brain cancers (e.g. GBM); her team supports those with all other cancer diagnoses. Learn more about their services for clients here on her website, [Dietary Therapies](#).

The efficacy of a supervised keto diet for cancer is highly dependent on your Glucose Ketone Index (GKI): a calculation made from daily fingerstick tests of glucose and ketones. This is accomplished easily with a high-quality glucose/ketone meter such as the [KETO-MOJO GK+](#) Glucose and Ketone Testing Kit. We believe that the highest potential for therapeutic success requires GKI values of 2.0 or lower, ideally 1.0 or below if undergoing an active treatment (such as chemo or hyperbaric oxygen). [This paper](#) describes the concepts and procedures for measuring the GKI. We have found that it is not necessary to achieve blood glucose levels as low as 55-65 mg/dl *if* ketone bodies are elevated, producing GKI values of 2.0 or below. It is important to understand, though, that certain conditions and situations can interfere with attaining the ideal GKI.

Any diet that can achieve a GKI of 2.0 or below will have therapeutic efficacy. Possibilities include classic keto, modified keto, paleo, carnivore, vegetarian, Mediterranean keto, etc. The basic food lists of a general ketogenic diet, provided by Dr Jocelyn Tan-Shalaby, are provided as an attachment to my email.

Dietary intake of glucose-containing foods is not essential; the body has multiple redundant pathways to make what it needs to maintain healthy cells. We have seen that hypoglycemia (generally viewed as blood glucose below 70mg/dL or 3.9 mmol/L) is not an issue when ketone bodies (beta hydroxybutyrate) become elevated, according to several clinical reports. Both blood glucose and ketones can be measured after an overnight fast and again in the afternoon, or before an early evening meal. In order to learn more about your trends, you might take GKI measurements at other times during the day, especially during the adaptation period (3-4 weeks). Using a continuous glucose monitor (e.g., Freestyle Libre) and continuous ketone monitor (e.g., SiBio CKM) could be helpful during the initial ketogenic adaptation period, although finger-prick monitoring can provide similar information if sampling is frequent.

A high degree of patient motivation and compliance is essential for success. The case report entitled, *Ketogenic Metabolic Therapy, Without Chemo or Radiation, for the Long-Term Management of IDH1-Mutant Glioblastoma: An 80-Month Follow-Up Case Report*, describes how one patient was able to manage his brain cancer using surgery and metabolic therapy, without radiation or chemotherapy.

[Click here](#) for access to the full text of the paper. This patient survived (and thrived) for 10 years, eventually succumbing to a post-operative complication of surgery.

We believe optimal therapeutic potential requires GKI values of 2.0, ideally 1.0 or below. However, this is not always possible. Also, we have found that it is not necessary for patients to sustain blood glucose levels of 55-65 mg/dl, although they may be achieved occasionally during fasting or intermittent calorie restriction. Absolute glucose levels should be as low as physiologically possible within the natural adaptation to the ketogenic diet; different individuals may have different basal levels of gluconeogenesis and blood glucose levels (glycemia). Miriam Kalamian has written a blogpost [here](#) on factors that can negatively impact blood glucose levels and GKI. Even when a low GKI cannot be maintained continuously, we have reason to speculate that achieving a low number during critical treatment periods, using short-term or modified fasting and ketone supplementation, will still enhance metabolic treatment efficacy.

Targeting Glutamine:

Although glucose is generally cancer's preferred fuel, the amino acid glutamine is also used by cancer cells. *However, we do not suggest that you eliminate foods containing glutamine.* Foods containing glutamine will *not* enhance tumor growth due to saturation of glutamine availability at the de novo synthesis threshold (the body's production of glutamine typically exceeds the utilization by tumor tissues).

Dosage and timing of protein intake play a crucial role here, and the management of protein intake should be guided by a knowledgeable practitioner or team.

Glutamine is an essential metabolite, needed to maintain gut health, muscle mass and immune system functions. Glutamine is also a vital part of neurotransmission in the brain. Blood glutamine levels can be lowered by following a water-only fast for 14-21 days, but this is not advisable if muscle mass is already compromised, as it often is in older adults or in those undergoing aggressive treatment. High-intensity exercise coupled with a very low carbohydrate ketogenic diet can also transiently lower glutamine availability.

Furthermore, attempts to restrict blood levels of glutamine through limiting dietary intake can fail due to redundant metabolic pathways that ensure that cells receive a continuous supply of glutamine even if that means degrading healthy tissue. A healthier approach to lowering glutamine levels is through aerobic exercise and resistance/strength training (with adequate nutrition and recovery) to maintain and recover muscle mass. Consult first with a knowledgeable professional that considers your age, condition, and any limiting factors.

Glutamine targeting drugs that inhibit glutamine availability, such as hydroxychloroquine and sodium phenylbutyrate, are approved drugs for other conditions which makes them available off-label but by prescription only. Both have been tested in human clinical trials for cancer but not in the context of ketogenic metabolic therapy. *Caveat: this needs to be done very carefully and with monitoring for side effects by your medical team.*

Regarding the DON drug: As I have mentioned previously in papers and podcasts, 6-Diazo-5-oxo-L-norleucine (DON) is highly effective in targeting the glutamine pathway, but this drug is difficult to obtain and should be administered parenterally (intravenously or subcutaneously) by a trained medical professional to ensure safe and effective use. (While single dosing is generally not toxic, repeated administration of DON can lead to serious side effects by inhibiting glutamine metabolism across normal tissues, including gastrointestinal tissues and immune system). Oral dosing is likely less effective and limited by toxic GI side effects. Anyone considering this drug must obtain it from a company that produces it for research and find a physician willing to administer it intravenously (perhaps using the dosage information in this [Duraj et al paper](#)). Other DON prodrugs or formulations may be approved in the future for specific cancers which should then make it more accessible off-label.

Other Repurposed Drugs

The anti-parasitic drugs, mebendazole (MBZ) or fenbendazole (FBZ), can produce synergistic effects especially when used while in therapeutic ketosis (GKI 2.0 or below). These drugs can target both glucose and glutamine: they are readily available, relatively inexpensive, and can be administered orally. The [Duraj et al paper](#) provides an extensive list of these drugs, along with information on dosages, timing, and scheduling, all of which we hope will be investigated in future clinical trials. Information on mebendazole, a drug I have researched in my lab, can be found in [this paper](#). Dr. Angie Choi's book, *Whole New Me: Healing From Cancer in Body, Mind, and Spirit*, also has some specific info in a reader-friendly format.

Combining Metabolic Therapies with Conventional Care

The definition of cancer is out-of-control cell division or dysregulated cell growth. As a person with a cancer diagnosis, you may want to ask your oncologist how familiar they are with metabolic therapy as a fourth branch of oncology. Be prepared for a negative or neutral response as most are not yet trained or even knowledgeable about this option.

It is important to know that there are two general categories of ketogenic metabolic therapy (KMT): **Dietary metabolic therapy** (GKI 2.0 or below), and **pharmacological metabolic therapy** (using repurposed drugs, e.g. MBZ, FBZ, DON, together with KMT). For cases of advanced cancer, both categories of KMT may be necessary for optimal results. It will be helpful to find a healthcare provider that is either knowledgeable or open to learn about these non-toxic, evidence-based therapeutic strategies. Most times, you will need to look find an integrative oncologist who works outside of institutional settings or conventional clinics.

Before beginning the standard of care, or even alongside standard treatment, cancer patients and their family members have the right to pose the following simple questions to their oncologist. Be aware, though, that some oncologists could be offended by what they perceive as interference with the planned treatment.

1. Will the treatment you are proposing reduce the availability of the two primary fuels (glucose and glutamine) that are driving the dysregulated growth of my cancer?
2. Will the treatment target my tumor cells while preserving the health and vitality of normal cells and tissues?

3. Is the treatment you are proposing based on the somatic mutation theory or on the mitochondrial metabolic theory of cancer? (This issue should only be approached if you receive satisfactory answers to questions 1 and 2.)

The answers received to these three questions could predict your destiny.

Rationale for these questions:

Blood ketone levels of 1.0-3.0 mmol/L can enhance the health and vitality of normal cells, but not tumor cells. Please note that the glucose and glutamine requirements of normal cells can be significantly reduced by switching from carbohydrate-rich foods to fat/protein-rich foods. This shift places metabolic stress on the tumor cells while, at the same time, enhancing the metabolic efficiency of normal cells. Normal cells will gradually transition metabolism from carbohydrates to fatty acids and ketone bodies because they have healthy mitochondria that produce energy from oxygen. Ketones are the preferred energy source for the brain so it makes sense that the brain is especially adaptable to using ketone bodies when glucose becomes limited. *Tumor cells cannot make this transition to ketone bodies because their mitochondria are defective and cannot produce enough energy from oxygen.* Consequently, tumor cells, regardless of cell or tissue origin, are dependent on fermenting glucose and glutamine for their growth.

Nutritional Ketosis is Normal and Natural

The baseline for nutritional ketosis is defined as blood levels of beta-hydroxybutyrate at 0.5 mmol/L. We believe that blood ketone levels of 1.0-3.0 mmol can enhance the health and vitality of normal cells, but not tumor cells, as described in the paragraph above

It's important to draw the distinction between nutritional ketosis and aberrant diabetic ketoacidosis. *Nutritional ketosis is a healthy, evolutionarily conserved, metabolic adaptation to starvation or limited nutrient availability; it is fundamentally different from diabetic ketoacidosis, which is an unhealthy, pathological state associated with poorly-controlled diabetes.* Recognition of the above-mentioned facts is supported by a mountain of peer-reviewed and published scientific evidence.

Supporting Further Research into Metabolic Therapies for Cancer

Support for my research comes largely from philanthropy and private foundations. I update the information in this kit regularly and provide it to you without cost. If you find that this summary and the attached information is helpful, please consider donating to our research:

Boston College Biology Cancer Research:

Donations can be made directly to my cancer research through the Boston College Biology Department mentioned at the bottom right of my department web page (Give to Biology). The details can be found on the link below. In making a special gift to the department in my name, please be in touch with the Director of Development, Schools and Programs, Sally Murray, by email at sally.murray@bc.edu or by phone at 617-552-8517. (<https://www.bc.edu/bc-web/schools/morrissey/departments/biology/people/faculty-directory/thomas-seyfried.html>).

The Foundation for Cancer Metabolic Therapies:

The Foundation is a 501(c)(3) foundation (EIN #46-4127870) dedicated to supporting our studies using metabolic therapy for cancer management. You can find the "donate" tag on the top row of the foundation site. The Foundation address is 3213 West Main Street #256, Rapid City, SD 57702. The Foundation encourages [donations](#) through the PayPal Giving Fund (they provide IRS-applicable receipts and charge no fees): 100% of your donation will go to the foundation and research on cancer metabolism. The PayPal Giving Fund or a check are also easy options for larger donations. Please remember that no donation amount is too small or too large.

Your donation will keep our research moving forward!

Cancer as a Metabolic Disease

I discuss further aspects of cancer as a metabolic disease in the following videos.

<https://youtu.be/wY-JZ6TTNh8> youtu.be/1ebPZP9hBPA
<https://youtu.be/06e-PwhmSq8>
<https://youtu.be/6PJfOFTaYow>
<https://youtu.be/pwhRskOPwVk?si=7keyLVIQ-p1ZM7LT>
<https://youtu.be/MakS2iRkj1Q?si=CXnWBMVArR6PScmc>
https://youtu.be/A5ONwV1FU_c?si=JRLN0zVAbJgqpRGx
<https://youtu.be/X2uSRnQbWMw?si=dyPc9tEfDi5zViRG>
<https://youtu.be/VaVC3PAWqLk?si=3pJu0XY4lrq1B61O>
https://youtu.be/kDbZrYu4V1E?si=V57y1ZEif_kh40Y4
<https://youtu.be/KjdAtauO2cA?si=LpvSoSavFqi-GtQy>

Ron Piana's take on the corrupted cancer industry

<https://www.youtube.com/watch?v=foj4sfQP3ek&t=877s>

Specific Cancers

Brain cancers

Despite its universal acceptance, we have found that radiation therapy for malignant brain cancer can contribute to rapid tumor recurrence and reduced patient survival. Brain tumor radiotherapy increases levels of glucose and glutamine in the tumor environment thus increasing the aggressiveness of the remaining tumor cells and rapid tumor recurrence. Steroids, notably dexamethasone, are used liberally to reduce the side effects of this treatment, further contributing to high blood glucose and a poorer prognosis ([click here](#)). The evidence supporting these statements also appears in our research ([here](#) and [here](#)), and in the [Duraj et. al](#) paper. Please read these papers very carefully so that you are informed of the risks. Unfortunately, many oncologists withhold this information, choosing to focus on algorithm-driven protocols and symptom relief rather than addressing the negative effects.

Keep in mind that the recurrence rate for glioblastoma (GBM) is near 95%, with less than 1% of patients surviving 10 years after diagnosis and treatment with the current standard of care (surgery, if possible, followed by radiotherapy and temozolomide chemotherapy). People diagnosed with GBM, and their loved ones, understandably find it devastating to read these dire statistics. There is hope for a better outcome by layering in metabolic therapy, so I encourage you to carefully review the publications above that provide the evidence supporting this approach. We also present novel preclinical data for managing pediatric high-grade glioma (like adult GBM) in [this publication](#)).

Dr. Jethro Hu is also treating GBM patients with ketogenic diet and other metabolic therapy at Cedars-Sinai Hospital in Los Angeles, CA (Jethro.Hu@cshs.org).

Dr. Kris Smith also treats brain cancer patients with surgery and metabolic therapy at Barrow Neurological Institute, Phoenix, AZ USA (Kris A. Smith, neuropub.smith@barrowneuro.org).

Preliminary evidence suggests that mebendazole could be more effective than fenbendazole for managing glioblastoma. The following additional videos and papers may also be useful. (youtu.be/cNvt8L3SINI) (youtu.be/KusaU2taxow)

Links to additional publications on the metabolic management of malignant brain cancer

1. [Role of ketogenic metabolic therapy in malignant glioma: A systematic review](#)
2. [Ketogenic Metabolic Therapy for Glioma](#)
- 3.. [Antiparasitic mebendazole shows survival benefit in 2 preclinical models of glioblastoma multiforme](#)

Prostate cancer:

I suggest contacting Mr. Guy Tenenbaum, who successfully managed his advanced metastatic prostate cancer using metabolic therapy (guytenenbaum@gmail.com). It is likely that most of the therapeutic benefit could be attributed to his strict calorie-restricted ketogenic diet and his extended (18-20 day) water-only fasting that produced GKI values of 1.0 and below. One caveat: he has stated in videos and papers that he believes the SCOT enzyme fuels cancer progression. However, it is our opinion that the SCOT enzyme does *not* play a significant role here so it is not necessary to use any drug or supplement to inhibit this enzyme. That said, we do not rule out the possibility that SCOT inhibitors could have anti-cancer effects independent of SCOT inhibition. The following additional video and papers may also be useful.

- [Guy Tenenbaum's story](#)

- Also see the Martin Friedel treatment plan for prostate cancer (attached to the email). This plan may also be effective for managing other cancers.

- Please also read [Seyfried, Prostate Cancer, Nature](#) (attached to the email)

Breast cancers:

I can recommend Susan Wadia-Ells' book, "Busting Breast Cancer: Five Simple Steps to Keep Breast Cancer Out of Your Body." For breast cancers, we believe that the use of fenbendazole is preferable to the use mebendazole. Here are some links to additional publications on the metabolic management of breast cancers.

1. Metabolically Supported Chemotherapy for Managing End-Stage Breast Cancer: A Complete and Durable Response (<https://pmc.ncbi.nlm.nih.gov/articles/PMC8072186/>)
2. Consideration of Ketogenic Metabolic Therapy as a Complementary or Alternative Approach for Managing Breast Cancer (presents additional information on breast cancer management including the biopsy-induced spread of cancer cells.) (<https://doi.org/10.3389/fnut.2020.00021>)

Lung Cancer & Other Cancers

There is a growing body of evidence to support the use of KMT to manage a wide range of cancers, including colon, bladder, uterine, bone, kidney, liver, pancreatic, and various blood cancers. Here are links just a few:

1. Press-Pulse Therapeutic strategy (<https://t.co/mvcKo4wXKE>).
2. Modified Atkins diet in advanced malignancies (<https://nutritionandmetabolism.biomedcentral.com/articles/10.1186/s12986-016-0113-y>).
3. Managing Metastatic Thymoma With Metabolic and Medical Therapy: A Case Report. (<https://t.co/uOL74ZsTCJ>).
4. Feasibility study of metabolically supported chemotherapy with weekly carboplatin/paclitaxel combined with ketogenic diet, hyperthermia and hyperbaric oxygen therapy in metastatic non- small cell lung cancer. (<https://www.tandfonline.com/loi/ihyt20>).
5. Promising Effect of a New Ketogenic Diet Regimen in Patients with Advanced Cancers (<https://pmc.ncbi.nlm.nih.gov/articles/PMC7284721/>).
6. Restricted Ketogenic Diet Therapy for Primary Lung Cancer With Metastasis to the Brain: A Case Report (<https://pmc.ncbi.nlm.nih.gov/articles/PMC9435310/>)

This information is dedicated to the millions of people who have suffered and died from toxic cancer therapies.