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Study Finds Linoleic Acid May Directly Influence Cancer Growth

Analysis by Dr. Joseph Mercola | May 15, 2026

STORY AT-A-GLANCE

- Linoleic acid (LA), a ubiquitous omega-6 fat in Western diets, may directly influence aggressive cancer growth by activating specific cellular pathways, according to recent animal research
- High levels of LA, found predominantly in ultraprocessed foods, vegetable oils, and many packaged snacks, may contribute to cancer risk
- The research shows that triple-negative breast cancer (TNBC) cells in animal models thrive on LA because it appears to trigger mTOR signaling, a pathway linked to rapid tumor growth
- To support cellular health, consider reducing your daily LA intake by less than 5 grams per day by limiting ultraprocessed foods, nuts, seeds, and conventionally raised meats
- Swapping high-LA foods for more stable, healthier fats and targeted carbohydrates may support energy production and may help lower the inflammatory load that research associates with cancer progression

Linoleic acid (LA) is an omega-6 polyunsaturated fat (PUF) abundantly found in vegetable oils like safflower, soybean, and sunflower oil, as well as most ultraprocessed foods. Despite mainstream medical advice dictating that LA is essential, I believe that it is one of the worst ingredients in the food system today because excessive intake may compromise your cellular and mitochondrial function.

The usual intake of LA is approximately 29 grams per day, which is a far cry from our ancestors' intake back in 1865 — just 2 grams daily! Given this drastic shift, some researchers have proposed a link between elevated LA intake and rising rates of chronic disease, including cancer.

Aggressive Cancer Cells Thrive on LA

A study published in Science journal investigated how LA may influence cancer growth in animal and cellular models.[1] The researchers focused on aggressive breast cancer cells, known as triple-negative breast cancer (TNBC), using an animal model to understand the process behind these tumors. This form of breast cancer is especially difficult to treat because it does not respond to common therapies.

- **A high-LA diet substantially accelerated tumor growth —** Mice fed with LA, similar to diets high in ultraprocessed foods and vegetable oils, developed larger tumors than those consuming lower levels of this PUF.

  What is notable is how LA appeared to drive such rapid tumor growth. The researchers found that LA didn't merely provide fuel — it also activated a cellular growth pathway, mTORC1 (mechanistic target of rapamycin complex 1), which acts like a switch for your cells, telling them when to multiply. When this switch is activated by LA in these models, cancer cells multiply more rapidly.

- **The link between mTORC1 and FABP5 —** The activation of mTORC1 was evident in cancer cells containing high amounts of fatty acid binding protein 5 (FABP5), which can be thought of as a delivery vehicle that picks up LA from your diet and transports it toward cancer cells. Once inside, FABP5 binds to and helps activate mTORC1. When this occurs, cells multiply more rapidly, contributing to tumor growth.

  The researchers observed this effect in the test models they ran. Within just a few weeks of starting a high-LA diet, tumors in the high-LA mice subjects grew larger and faster compared to groups fed diets with lower LA content. This effect was

consistent and measurable, suggesting a relationship between dietary LA and accelerated tumor progression in these mice.

- **Exploring the role of mTORC1 —** The researchers observed that the effect was especially pronounced in triple-negative breast cancer tumors, which are among the hardest forms of cancer to treat. The mechanism behind this accelerated growth involves the protein complex mTORC1.

  Normally, mTORC1 helps your body regulate growth, responding to nutrients and ensuring your cells multiply when they have sufficient resources. However, when continuously activated by LA through FABP5 in these models, mTORC1 pushes cells toward a sustained state of growth. For cancer cells, this can translate into continuous multiplication.

- **The mechanism behind FABP5 —** When the LA from your diet binds to FABP5, it is transported toward the cellular area where mTORC1 resides. FABP5 then helps LA interact with the components of mTORC1, pushing the switch to turn "on." Without FABP5, LA does not efficiently activate mTORC1, and tumor growth is reduced.

  By binding to FABP5, LA forms a complex that activates mTORC1, which drives tumor cell growth, the researchers noted. "Feeding mice that model triple-negative breast cancer a high-linoleic-acid diet increased FABP5 levels, mTORC1 activation and tumor growth."[2]

- **Cancer tissues show elevated FABP5 —** The researchers found higher FABP5 levels in cancer tissues compared to normal tissues, suggesting that aggressive cancers may upregulate FABP5 to increase fatty acid uptake. These findings add to the evidence base supporting the value of reducing LA consumption, particularly for individuals concerned about cancer risk.

Co-author John Blenis, Ph.D., summarizes the study, saying that "This discovery helps clarify the relationship between dietary fats and cancer, and sheds light on how to define which patients might benefit the most from specific nutritional recommendations in a personalized manner."[3]

Expanding on these findings, co-author Nikos Koundouros, Ph.D., says, "There may be a broader role for FABP5-mTORC1 signaling in other cancer types and even in common chronic diseases such as obesity and diabetes."[4]

How FABP5 Is Implicated in Cancer Spread and Growth

Going deeper into FABP5, a 2023 review published in Drug Discovery Today explored its role in various cancers and why this protein is considered a priority research target in oncology.[5]

- **FABP5 is a "chaperone" protein —** This means FABP5 helps transport fat into cancer cells. Examining patient-derived tumor samples, the review reported that higher FABP5 levels were observed more frequently in aggressive cancers such as liver, prostate, breast, and brain tumors.
- **Elevated FABP5 was associated with poorer outcomes —** In the patient cohorts analyzed, higher FABP5 levels in tumors correlated with lower survival. In one cohort of prostate cancer patients, approximately 35% survived beyond 40 months compared with approximately 80% among those with lower FABP5 levels.
- **FABP5 expression was associated with worse breast cancer outcomes —** In aggressive TNBC cases reviewed, high FABP5 levels corresponded to lower survival over several years compared to patients whose tumors expressed less FABP5. These findings suggest FABP5 may be more than a biomarker — it may play an active role in cancer progression.
- **Reducing FABP5 activity slowed cancer growth in lab studies —** The review highlighted improvements in tumor control when FABP5 activity was reduced. In laboratory tests, inhibiting FABP5 slowed cancer cell growth, decreasing cell proliferation by more than half in several aggressive cancers, including prostate and breast cancer cell lines.

In the cited preclinical studies, treatment with FABP5 inhibitors substantially reduced tumor size within weeks and reduced metastasis, supporting FABP5 as a potential therapeutic target.

- **Reducing FABP5 produced rapid changes in tumor models —** Within days, prostate cancer tumors in these animal models began to shrink; over approximately one month, tumor volumes decreased by roughly twofold compared to untreated animals.

  Overall, the review suggests that aggressive cancers such as TNBC, prostate cancer, and liver cancer may show the greatest response to FABP5 reduction strategies in preclinical models. Because these aggressive cancers currently lack effective targeted treatments, FABP5 inhibition could be a promising avenue for further research.

- **FABP5 activity correlates with cancer aggressiveness —** When the researchers compared FABP5 to other growth-related proteins, higher FABP5 levels showed a stronger correlation with worse patient outcomes and faster progression than many other common cancer markers.

  FABP5 is thought to support cancer growth in part by enhancing the activity of several key growth pathways within cells. One of the most prominent is NF-κB, an inflammatory signaling route that works like an accelerator pedal inside cancer cells — when FABP5 activates it in these models, cancer cells rapidly multiply, evade the body's natural defenses, and become more invasive. Inhibiting FABP5, as demonstrated in these studies, effectively lifted the foot off that pedal, slowing cancer growth.

- **FABP5 interacts with peroxisome proliferator-activated receptors (PPARs) —** These proteins regulate how cells handle fats and growth signals. FABP5 appears to deliver fat to these receptors, activating them and pushing cells to multiply more rapidly. Without FABP5's assistance, this cycle is interrupted, and cancer cells in these models are less able to sustain rapid growth.

- **Hypoxia-inducible factor 1-alpha (HIF-1α) expression appears to be boosted by FABP5 —** HIF-1α is a growth pathway that cancer cells use to survive low-oxygen environments typically found within rapidly growing tumors. In other words, when cancer cells multiply quickly, they often outgrow their blood supply, creating areas low in oxygen. HIF-1α allows cancer cells to adapt and survive these conditions, promoting tumor survival.

  Interrupting FABP5 appears to weaken this defense, as shown in preclinical studies, making cancer cells potentially more susceptible to treatment.

These findings are from laboratory or animal research and may not directly apply to human health.

Practical Strategies to Lower Your LA Intake

Taken together, the featured research supports minimizing LA intake as an important strategy that may help reduce cellular dysfunction associated with cancer. The operative word here is "minimize," as it's practically impossible to avoid LA in the food system today. Your body needs LA, but only in small amounts — below 5 grams per day. Here are additional recommendations that may reduce LA exposure to support healthier cellular metabolism:

- **Ditch ultraprocessed foods —** These products, which include packaged foods, normally contain high amounts of LA. Even items you wouldn't expect, like trail mixes, granola bars, or salad dressings, hide this harmful fat. If your food comes with a label, review it thoroughly before eating it.

- **Choose animal meats wisely —** Conventionally raised chicken, pork, and farm-raised salmon are higher in LA because of the industrial feed they're given. Instead, opt for grass fed beef or wild-caught fish. These options have much lower omega-6 levels, reducing your exposure to fats that research associates with cellular inflammation.

- **Lower your overall fat intake —** To support cellular health, I recommend keeping your total daily fat intake below 30% of your calories. This helps prevent the Randle Cycle, a metabolic switch that shifts fuel use toward fat. For a more in-depth look at this topic, read "**Understanding the Randle Cycle**."

- **Cut back on nuts and seeds —** While nuts and seeds are widely promoted as healthy snacks, I don't recommend them anymore because they're loaded with LA. For your safety, choose healthier fats like grass fed butter, tallow, coconut oil, or ghee. These support cellular health without the inflammatory load associated with high-LA foods.

- **Eat more healthy carbs —** To replace the space left by reducing high-LA foods, consume approximately 250 grams of carbohydrates daily, ideally from nutritious sources such as whole fruits, root vegetables, and properly prepared starches like white rice.

If you have gut health concerns, introduce carbs slowly, starting with white rice and whole fruits. This provides your body with its preferred fuel source, supporting your cellular energy without adding to the inflammatory load linked to high-LA foods.

The Pax health platform, which is coming very soon, will include Food Buddy and the Seed Oil Sleuth. This is a special feature designed to help identify hidden sources of LA in your diet as well as estimate the total daily intake. If you're interested in reducing exposure, I invite you to sign up once it becomes available, as you may find it useful as a practical tracking aid.

Frequently Asked Questions (FAQs) About the Link Between LA and Cancer

Q: What is LA and why is it considered concerning?

A: LA is an omega-6 PUF commonly found in vegetable oils and ultraprocessed foods. Research suggests excessive intake may disrupt normal cellular function. Recent animal studies have linked high LA intake to the growth of aggressive cancers such as TNBC.

Q: How does LA contribute to aggressive cancer growth?

A: Animal research suggests LA can bind to a protein called FABP5, which activates the mTORC1 growth pathway. In these models, activated cancer cells multiply more rapidly, forming larger tumors. Whether the same relationship holds in human physiology remains an area of active research.

Q: Which high-LA foods should I consider avoiding?

A: Ultraprocessed foods, vegetable oils, conventionally raised meats like chicken and pork, farmed salmon, and nuts and seeds are major sources of LA. Reducing your intake of these foods in your diet may help lower LA exposure, which research associates with cancer risk.

Q: What does published research say about lowering LA intake and cancer progression?

A: In animal and cellular studies, reducing dietary LA has been associated with lower FABP5 and mTORC1 activation and slower tumor growth. Human clinical validation is still needed before any claim about LA reduction as a cancer treatment can be made.

Q: What dietary changes may support cellular health in relation to LA?

A: Focus on whole, unprocessed foods; choose grass fed beef or wild-caught fish; keep total fat intake below 30% of daily calories; limit nuts and seeds; and consume healthy carbohydrates such as white rice, fruits, and properly prepared starches to fuel your cells.

This article is for informational purposes only and does not constitute medical advice. Consult a qualified health care provider before making changes to your health regimen.

Sources and References

- [1] Science Volume 387, Issue 6739, doi: 10.1126/science.adm9805
- [2, 3, 4] Weill Cornell Medicine, April 1, 2025
- [5] Drug Discovery Today Volume 28, Issue 7, July 2023, 103628