COMMENTARY

The Ivermectin Paradox in Oncology: When a Grain of Science Becomes a 'Miracle Cure' Myth

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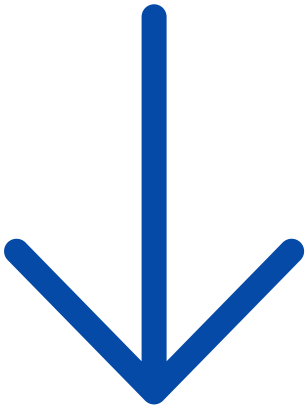


Jump to Key Points

I've started to recognize the cadence of the question before it's fully asked.

A patient or caregiver leans in — often after a long visit about scans, side effects, and the thousand small logistics that cancer demands — and says something like, "Doctor, can I ask you about ivermectin? I heard it's a cancer cure. If it might help, why wouldn't we try it?"

It's a fair question, not because ivermectin is a cancer cure (it isn't), but because the context makes the question inevitable: Oncology is hard, outcomes can be uncertain, and the internet is overflowing with confident claims that require only one click — all while real medicine requires time, nuance, and, too often, patience.

It is completely understandable we want to do everything we *can control* while dealing with cancer, and it is at the crux of why this question is so common. So, let's demystify this without shaming curiosity or turning the conversation into a battle. This is about clarity, safety, and preserving trust, especially when patients are most vulnerable.

Ivermectin, an antiparasitic drug, shows preclinical promise against cancer but lacks clinical efficacy. Despite online claims, its high-dose use poses toxicity risks without proven benefits in human cancer treatment.

Ivermectin Is a Real Drug With a Real Legacy

Ivermectin didn't start as an internet storyline. It is a widely used antiparasitic medication, and its impact on global health is undeniable. The discoveries leading to ivermectin-based therapy for parasitic diseases were recognized with the 2015 Nobel Prize in Physiology or Medicine.

Mechanistically, ivermectin targets glutamate-gated chloride channels in parasites (channels humans don't possess in the same form), which helps explain how it can be effective against parasites while remaining relatively safe at approved doses.

That pedigree is part of what makes the cancer claim so "sticky." A Nobel Prize can become a halo effect — a shortcut that feels like proof. But Nobel-worthy public health impact is not the same as oncology efficacy.

The 'Grain of Truth' Is that Ivermectin Does Things to Cancer Cells (in a Lab)

Ivermectin is not inert in a petri dish.

There is a substantial body of preclinical work showing ivermectin can affect cancer-related pathways and cancer cell viability *in vitro*, and sometimes in animal models. Reviews summarize potential multitarget effects across pathways like Akt/mTOR, Wnt/β-catenin, purinergic receptors, and PAK1.

One area holds particular interest because it speaks the modern language of immuno-oncology: the idea that ivermectin may promote **immunogenic cell death (ICD)**. In preclinical mouse models of breast cancer, researchers observed that ivermectin could potentially turn "cold" tumors "hot" (ie, ICD)

by increasing immune infiltration, theoretically synergizing with checkpoint inhibitors like pembrolizumab.

This is the part that's often repeated online (sometimes accurately, often exaggerated): "It kills cancer cells," "It targets stemness," "It turns cold tumors hot."

But preclinical promise is the beginning of the question, not the end.

Biology In a Dish Is Not Biology in a Human

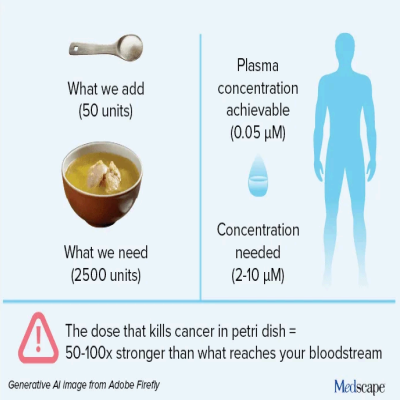
The pharmacologic reality rarely makes it into viral posts.

First, there is the issue of *the concentration gap*. Many *in vitro* cancer effects occur at concentrations that are difficult and/or unsafe to achieve in human tissues. For example, while ivermectin kills cancer cells in a dish at concentrations of 2-10 μM, the maximum achievable concentration in human plasma at standard dosing is roughly 0.05 μM (50 nM). Even at high doses, we struggle to bridge that 50-100x gap.

The Salt in the Soup Analogy: Why Lab Results Don't Translate to Patient Treatment

Imagine you're making a pot of soup (the petri dish). You discover that adding 25 teaspoons of salt per cup eliminates any bacteria completely. Here's the problem: you can't realistically add 25 teaspoons of salt to a gallon of soup without making it undrinkable. Whoever eats the soup will get sick before the bacteria dies. So, you add what's reasonable: 0.25 teaspoons per gallon. The soup reaches your customers' mouths, but the salt concentration is only 1% of what kills bacteria in your test kitchen. The bacteria thrives.

This is exactly the ivermectin problem with cancer.



Second, ivermectin is highly protein-bound in humans (roughly 99%), which further reduces the "free fraction" of the drug capable of diffusing out of blood vessels and into the tumor tissue to do the work. As a result, most of the drug remains bound to protein, leaving only a small portion available to be released and act where it is intended.

Finally, there is the blood–brain barrier. A major safety feature of ivermectin is that it is largely kept out of the central nervous system by the P-glycoprotein pump in an intact blood–brain barrier. This prevents the drug from interacting with human gamma-aminobutyric acid (GABA) receptors in the brain. Altering that protection — be it intentionally or unintentionally via high dosing, interactions with other drugs, or compromised barriers — invites neurotoxicity.

This is the "paradox" in plain language: You can't just scale a petri-dish effect into the human body without paying a toxicity price.

Safety Isn't Binary: 'Safe for parasites' ≠ 'Safe for cancer'

One of the most common rhetorical moves in ivermectin discourse is: "Billions of doses have been given, so it's safe." That's an incomplete argument.

Ivermectin is safe at intermittent, low doses (e.g., 0.2 mg/kg once a year). But the protocols suggested online for cancer often involve daily, high-dose ingestion (up to 1-2 mg/kg daily). The toxicology data show that overdose or accumulation can lead to central nervous system effects such as confusion, ataxia, seizures, and even coma.

This isn't theoretical, either. A 2025 case report in *Pediatric Blood & Cancer* detailed severe ivermectin toxicity in a pediatric oncology patient whose parents administered the drug based on online misinformation. Furthermore, historical data from *Loa loa* treatment shows that when the blood–brain barrier is breached or overwhelmed, ivermectin can cause fatal encephalopathy.

So the safety question is not, "Is ivermectin safe, ever?" but rather "*Is this proposed dose, duration, and combination safe for this patient, right now?*"

What does the human evidence show?

This is where we must be honest and steady. There is no credible clinical evidence that ivermectin is a stand-alone cancer cure. However, there are

legitimate efforts to study it. The viral claims often cite "success" where there is none. We should look at the actual data:

- The Cedars-Sinai Clinical Trial (NCT05318469): This is a phase 2 trial evaluating ivermectin combined with pembrolizumab or balstilimab in metastatic triple-negative breast cancer.
- The results: An abstract presented at the ASCO 2025 annual meeting reported early results. Among 8 evaluable patients, 6 had disease progression (PD), 1 had stable disease, and only 1 had a partial response.

A 12.5% response rate in a combination trial, where the partner drug (a checkpoint inhibitor) is already known to have activity, is far from the "melting tumors" narrative seen on social media — and does not justify treating ivermectin as a proven pillar of oncology outside a clinical trial.

The 'Turbo Cancer' Narrative

You may hear patients use the term "turbo cancer." It is crucial to define this clearly, as it is not a recognized medical diagnosis, but a term born from the anti-vaccine movement.

The term "turbo cancer" emerged sometime in late 2022/early 2023. It was popularized by influencers like Dr William Makis to describe alleged aggressive, rapidly progressing cancers supposedly caused by mRNA COVID-19 vaccines. Proponents claim that the vaccine's spike protein binds to the p53 tumor suppressor gene (the "guardian of the genome") or causes IgG4 class switching, leading to unchecked malignancy.

The reality is that there is no epidemiologic evidence of a post-vaccine "cancer epidemic," and the biological mechanism regarding spike protein and p53 is widely regarded as implausible by the oncologic community. Furthermore, independent reporting on cancer incidence from institutional registries or other organizations has not found any evidence of increased cases. Despite this, ivermectin is often marketed not just as a cancer drug, but as a specific *antidote* to this vaccine injury, sold in "emergency kits" by wellness companies for hundreds of dollars.

When patients bring this up, the goal isn't to humiliate the source, it's to separate fear from evidence. It is the only thing we would want for ourselves or loved ones in such a situation.

Why the Myth Spreads Anyway

The ivermectin-cancer narrative thrives for reasons that have little to do with pharmacology. Instead, it's often driven by complexity fatigue, as modern oncology is sophisticated and personalized, but also overwhelming. This makes the idea of a single drug "answer" feel emotionally relieving and like something within our control.

Anecdotes also tend to outperform statistics, a story of a "stage IV cure" spreads faster than a Kaplan-Meier curve, and celebrity amplification fuels attention no matter the medical reality. Additionally, "layering" creates false attribution, as patients often add ivermectin on top of chemotherapy. If the cancer responds, the add-on becomes the hero of the story, even if chemotherapy did the heavy lifting. If it does not work, then it is easy to blame it on "timing," "wrong dose," or "wrong regimen," (eg, adding mebendazole or fenbendazole), and shift the blame back to the patient or uncontrollable or unverifiable facts.

What I Say in Clinic (A Practical Script)

When a patient asks about ivermectin, I try to keep three promises: I won't dismiss the question, I won't pretend to be certain where we don't have it, and I won't let misinformation quietly increase their risk.

A simple approach:

- Start with alignment: "I understand why you're looking for every possible advantage. It makes total sense to want to leave no stone unturned."
- Clarify the goal: "Are you thinking about replacing treatment, or adding it on top? What did you see that convinced you?"
- Offer the evidence: "It's true that in lab studies, ivermectin affects cancer pathways. That's why scientists are researching it. However, the human trials so far — like the recent breast cancer study — haven't shown it to be effective in shrinking tumors in people, and the doses required to match the lab results can be toxic."
- Make safety a shared value: "If you decide to take anything (eg, prescribed, compounded, or purchased online), I need to know. We need to make sure it doesn't damage your liver or interfere with the chemotherapy that we *know* is working."

The bottom line is that ivermectin is not a suppressed miracle cure. It is a repurposed drug with intriguing preclinical signals that have failed, so far, to translate into clinical benefit for patients with cancer. If someone wants to "take it," that's ultimately their choice. My job, and our shared responsibility as clinicians, is to make sure they're choosing with eyes open, not because a viral story made certainty feel easy. That is why I like how AI is helping us change the narrative, and real repurposed drugs and new molecules can help us reach a world where can cure all cancers.

Arturo Loaiza-Bonilla, MD, MSEd, is the co-founder and chief medical AI officer at Massive Bio, a company connecting patients to clinical trials using artificial intelligence. His research and professional interests focus on precision medicine, clinical trial design, digital health, entrepreneurship, and patient advocacy. Dr Loaiza-Bonilla serves as Systemwide Chief of Hematology and Oncology at St. Luke's University Health Network, where he maintains a connection to patient care by attending to patients 2 days a week.

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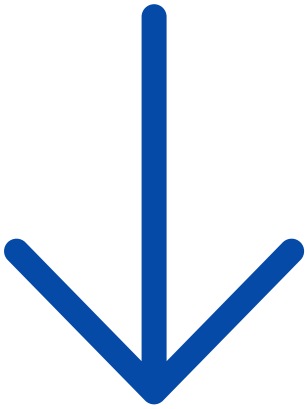
Did Hyperglycemia Affect Inavolisib-Efficacy in Patients With Breast Cancer?

Megan Brooks

May 13, 2026



Jump to Key Points

Efficacy outcomes were numerically higher among patients who experienced inavolisib-associated hyperglycemia, in a post hoc exploratory analysis of the INAVO120 trial. That trial found that patients with *PIK3CA*-mutated, hormone receptor (HR)-positive, HER2-negative advanced breast cancer benefitted from the addition of inavolisib to standard palbociclib and fulvestrant.

The new subgroup analysis also confirmed that the addition of inavolisib to endocrine therapy and cyclin-dependent kinase (CDK)4/6 inhibition improved progression-free survival (PFS) and overall survival (OS) in both patients who did and did not develop hyperglycemia.

Study author Sibylle Loibl, MD, PhD, cautioned that the outcomes by hyperglycemia status are “hypothesis-generating and should not be overinterpreted,” while presenting the new data at ESMO Breast Cancer 2026.

Hyperglycemia is a “well-known on-target toxicity” of PI3K inhibitors, including inavolisib, and these findings raise the possibility that hyperglycemia may reflect “pharmacodynamic target engagement,” explained study discussant Shigehira Saji, MD, PhD, professor and chair, Department of Medical Oncology at Fukushima Medical University in Fukushima, Japan.

Inavolisib improved progression-free and overall survival in breast cancer patients, regardless of hyperglycemia development. Hyperglycemia may indicate effective PI3K pathway targeting, but does not directly enhance efficacy, experts suggest.

The PI3K signaling pathway plays a central role in insulin signaling and glucose regulation. By inhibiting PI3K — particularly the alpha isoform targeted by inavolisib — these drugs can impair glucose uptake and increase blood sugar levels, producing hyperglycemia as a direct pharmacologic effect.

The data do not suggest hyperglycemia itself improves efficacy. Rather, it could be a pharmacodynamic sign that the drug is effectively hitting the PI3K pathway, experts said during the session.

The INAVO120 trial randomized 325 qualifying patients equally to receive either inavolisib 9 mg or placebo orally once daily on a backbone of palbociclib and fulvestrant in 28-day treatment cycles. The main findings from the trial revealed statistically significant improvements in PFS and OS with the addition of inavolisib to endocrine therapy and CDK4/6 inhibition. The FDA approved inavolisib in combination with palbociclib and fulvestrant for endocrine-resistant, *PIK3CA*-mutated, HR-positive, HER2-negative locally advanced or metastatic breast cancer following recurrence on or after adjuvant endocrine therapy, based on the INAVO120 trial.

At baseline, participants had fasting glucose levels below 126 mg/dL with A1c < 6.0%. Hyperglycemia (any grade) was experienced by 63.4% of those in the inavolisib group and 13.5% of those in the placebo group, and adverse events leading to discontinuation occurred in 6.8% and 0.6% of the trial participants in each of the groups, respectively. Baseline characteristics were generally balanced between patients who did and did not develop hyperglycemia, although higher body weight and BMI were associated with a greater likelihood of hyperglycemia.

The subgroup analysis of the trial, which looked at hyperglycemia and its correlation with outcomes among patients in the inavolisib group who developed hyperglycemia, found that 21.6% had a BMI of at least 30 compared with 11.9% among those who did not experience hyperglycemia.

“PFS was consistently improved with the addition of inavolisib, with a small trend of better PFS with hyperglycemia than without,” reported Loibl, who is a gynecologist at Goethe University Frankfurt in Frankfurt, Germany.

Median PFS was 21.0 months in patients with hyperglycemia and 12.8 months in those without hyperglycemia compared with 7.3 months among those in the placebo group. The hazard ratio vs placebo was 0.38 for patients with hyperglycemia and 0.51 for those without hyperglycemia.

Response rates were higher with inavolisib regardless of hyperglycemia status. Objective response rates were 64.7% among patients with hyperglycemia and 59.3% among those without hyperglycemia compared with 28.0% among those in the placebo group.

Median duration of response was 20.3 months in patients with hyperglycemia and 18.2 months in those without hyperglycemia vs 11.1 months among those receiving placebo.

OS findings followed a similar pattern. Median OS was 34.9 months in patients with hyperglycemia and 32.7 months in those without hyperglycemia compared with 27.0 months among those in the placebo group. Hazard ratios vs placebo were 0.66 and 0.72, respectively.

Overall, efficacy was consistently improved with the addition of inavolisib, with “no significant difference between the groups with and without hyperglycemia,” Loibl said.

Standard management strategies for emergent hyperglycemia, which are clearly outlined in prescribing information, allowed for prolonged treatment with inavolisib, she said.

Echoing Loibl, Saji told attendees “proactive management” of elevated glucose is “important” to allow for sustained dosing.

During the Q&A, Loibl was asked about whether hyperglycemia-related dose adjustments might affect outcome.

“We don’t know exactly, and I think it’s important that we don’t panic,” Loibl told attendees. “We should treat patients for hyperglycemia according to

the guidelines, adjust the dose as needed, and keep patients on treatment. They will benefit in the end," she added.

The study was funded by F. Hoffmann-La Roche. Disclosures for study authors are available with the original publication. Saji disclosed having relationships with Eli Lilly, Chugai, AstraZeneca, Daiichi Sankyo, Pfizer, and other pharmaceutical companies.