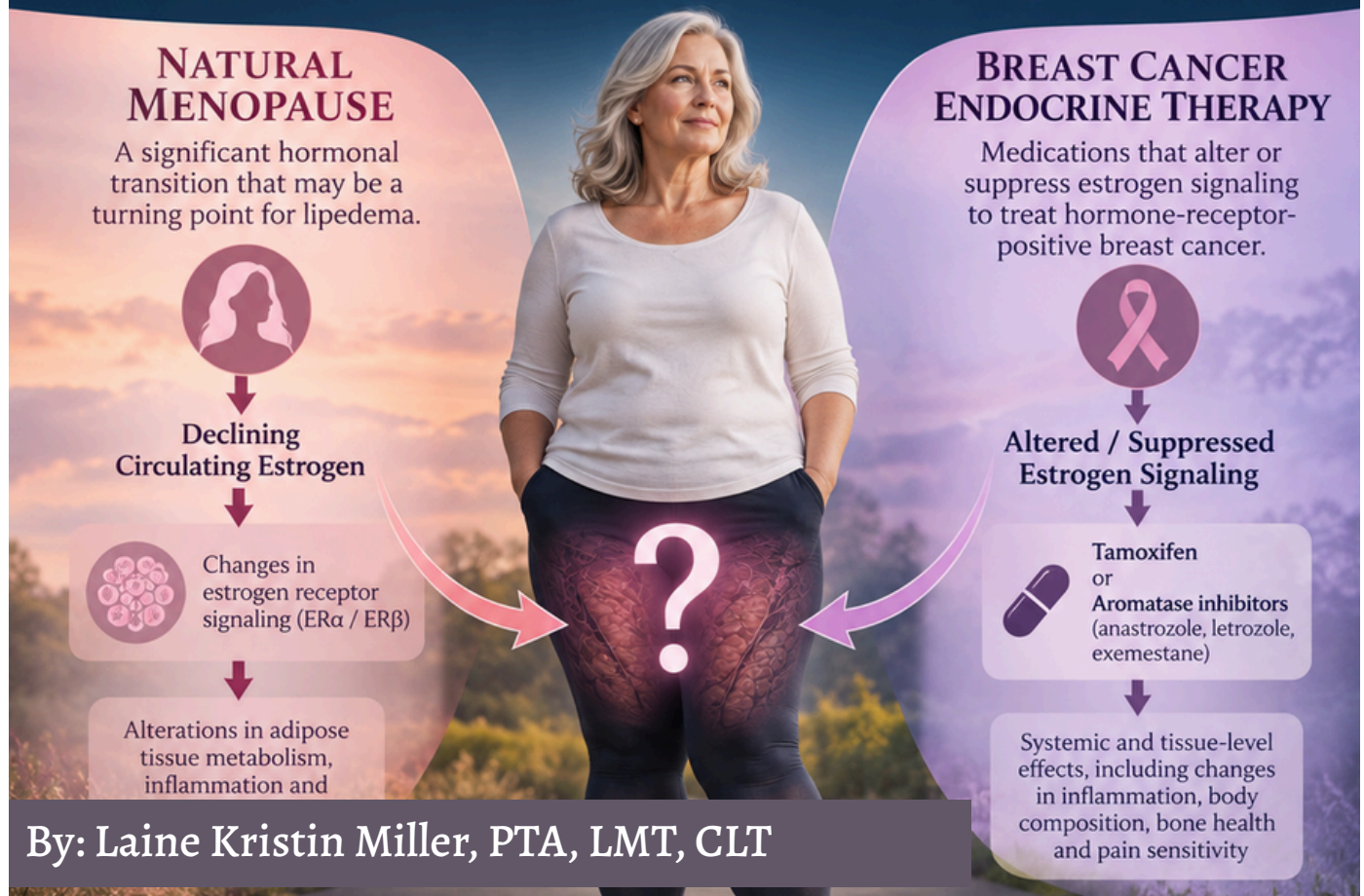


LIPDEMA, MENOPAUSE & HORMONE-BLOCKING THERAPY

What happens when estrogen signaling changes?

EMERGING SCIENCE. REAL WOMEN. IMPORTANT QUESTIONS.



Why Are We Asking This Question?

Lipedema occurs almost exclusively in women and has long been associated with periods of significant hormonal change. Many women report the onset or progression of symptoms around puberty, pregnancy, perimenopause, or menopause.

For years, these observations led researchers and clinicians to suspect that female sex hormones—particularly estrogen—may influence the development and progression of lipedema.

Today, research is beginning to explore why.

Recent studies have shifted the conversation beyond simply asking whether estrogen is “high” or “low.” Researchers are investigating estrogen-receptor signaling, local estrogen metabolism within adipose tissue, inflammation, connective-tissue remodeling, and changes in adipose biology as possible pieces of the lipedema puzzle.

A 2026 systematic review examining hormones and lipedema identified only 15 publications that met the authors' inclusion criteria. While the available evidence supports continued investigation of hormonal mechanisms, the small number of qualifying studies demonstrates how much remains unknown.

Menopause Adds Another Piece to the Puzzle

Menopause is particularly interesting.

Circulating estrogen levels decline substantially during menopause, yet many women report that their lipedema becomes more noticeable or progresses during this same period.

If estrogen contributes to lipedema, why wouldn't declining estrogen improve the condition?

The answer may lie in an important distinction:

The amount of estrogen circulating in the bloodstream is not necessarily the same as estrogen activity occurring within adipose tissue.

Researchers are investigating whether changes in estrogen receptors—particularly the relationship between ER α and ER β —and the local production and metabolism of estrogen within adipose tissue could influence how lipedema behaves during and after menopause.

This suggests that the relationship between estrogen and lipedema may be considerably more complex than simply “more estrogen equals more lipedema.”

And that raises another question.

What Happens When Estrogen Signaling Is Intentionally Changed

Millions of women are treated for hormone-receptor-positive breast cancer with medications designed to alter estrogen signaling.

Some receive tamoxifen, which acts on estrogen receptors. Others receive aromatase inhibitors, including anastrozole, letrozole, and exemestane, which dramatically reduce estrogen production. These treatments save lives and remain an essential part of breast cancer care.

But what happens when a woman receiving these medications also has lipedema?

Could years of medically altering estrogen signaling influence lipedema pain, adipose tissue, inflammation, fibrosis, body composition, or lymphatic function?

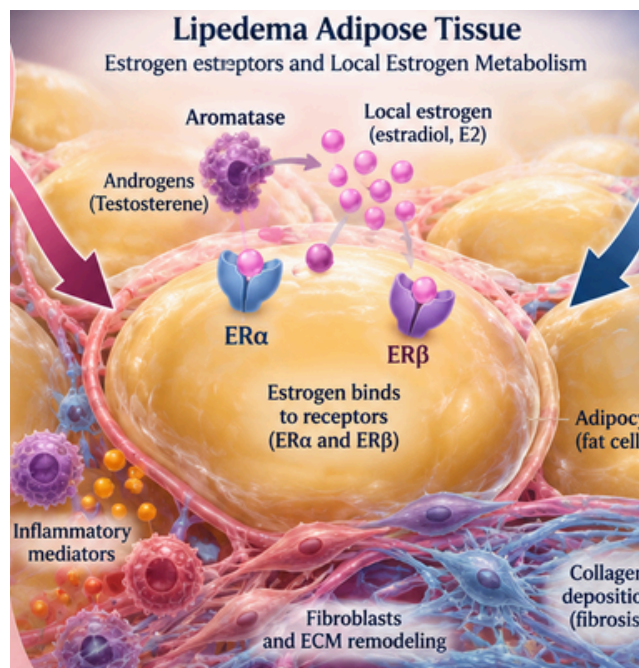
Could it make certain symptoms worse?

Could it potentially improve others?

Or could it have no meaningful effect at all?

We don't know.

That is precisely why this question deserves to be studied.



Important: There is currently no evidence that women with lipedema should stop, avoid, or change medically prescribed breast-cancer endocrine therapy. Decisions regarding cancer treatment should always be made with the patient's oncology team. This article explores an emerging scientific question—not a recommendation to alter cancer treatment.

Reference

Lüchinger JE, Pavicic E, Giachino CL, et al. Impact of hormones on lipedema development: a systematic literature review. *Archives of Gynecology and Obstetrics*. 2026;313:60.

The logic is strong: lipedema appears sensitive to hormonal transitions; emerging research implicates estrogen-receptor signaling and local estrogen metabolism; menopause is frequently associated with progression; and endocrine therapy deliberately changes estrogen signaling for prolonged periods.

Together, these factors make hormonal transition a scientifically important context rather than a background detail.

Changes in estrogen exposure may help researchers examine whether shifts in receptor activity or local hormone handling coincide with changes in lipedema symptoms or tissue behavior.

Estrogen Signaling: It Is More Than How Much Estrogen We Have

When discussing hormones and lipedema, it is tempting to think only about whether estrogen levels are high or low. Emerging research suggests the relationship may be considerably more complex.

Estrogen does not simply circulate through the bloodstream. It communicates with cells through estrogen receptors, particularly estrogen receptor alpha (ER α) and estrogen receptor beta (ER β). Adipose tissue is also hormonally active and contains enzymes involved in the local production and metabolism of estrogen.

This means that two women with similar circulating estrogen levels may not necessarily experience identical estrogen activity within their tissues.

Lipedema Adipose Tissue May Respond Differently

Researchers have proposed several estrogen-related mechanisms that could contribute to lipedema, including altered ER α /ER β signaling and changes in steroidogenic enzymes within adipose tissue.

Aromatase is particularly interesting. This enzyme converts androgens into estrogens and allows adipose tissue to contribute to local estrogen production.

This local hormone activity is sometimes referred to as intracrine signaling—hormones can be produced, metabolized and act within the tissue itself rather than their effects being determined solely by hormone concentrations measured in the bloodstream.

Researchers are now asking whether abnormalities in these pathways could help explain why lipedema frequently appears or changes during major hormonal transitions.

Why Menopause Is So Interesting

Menopause presents an apparent paradox.

Circulating estradiol falls substantially, yet menopause is frequently reported as a period when lipedema first becomes apparent or progresses.

A proposed 2025 model suggests that changes in estrogen-receptor balance and local estrogen metabolism within adipose tissue could help explain this observation. The authors propose that declining systemic estrogen does not necessarily mean that all estrogen signaling within adipose tissue disappears.

This remains a proposed biological model, not a proven mechanism of lipedema progression.

But it changes the question we should be asking.

Instead of:

“Does estrogen cause lipedema?”

perhaps the more useful question is:

“How does lipedema tissue respond when estrogen signaling changes?”

And That Brings Us to Endocrine Therapy

Breast-cancer endocrine therapies provide another circumstance in which estrogen signaling is deliberately altered—sometimes for many years.

Tamoxifen modifies estrogen-receptor signaling. Aromatase inhibitors such as anastrozole, letrozole and exemestane inhibit aromatase and substantially reduce estrogen production.

If lipedema involves altered estrogen-receptor signaling, local estrogen metabolism, or hormone-sensitive adipose and stromal tissue, it becomes reasonable to ask whether these medications might influence the condition.

They might.

They might not.

And different therapies might conceivably have different effects.

At present, we simply do not have enough lipedema-specific research to know.

That uncertainty is not evidence that endocrine therapy worsens lipedema.

It is evidence that the question deserves investigation.

Estrogen, a key regulator of adipocyte lipid and glucose metabolism, and female-associated body fat distribution are postulated to play a contributory role in the pathophysiology of lipedema.

Katzer K, Hill JL, McIver KB, Foster MT. Lipedema and the Potential Role of Estrogen in Excessive Adipose Tissue Accumulation. *International Journal of Molecular Sciences*. 2021;22(21):11720.
Pinto da Costa Viana D, Caseri Câmara L, Borges Palau R. Menopause as a Crit

Two Different Ways of Changing Estrogen Signaling



For women with hormone-receptor-positive breast cancer, endocrine therapy is designed to interfere with estrogen's ability to support cancer growth.

However, not all endocrine therapies accomplish this in the same way.

That difference may be particularly important when asking whether these treatments could influence lipedema.

Tamoxifen: Changing the Signal

Tamoxifen is a selective estrogen receptor modulator (SERM).

Rather than substantially eliminating estrogen from the body, tamoxifen binds to estrogen receptors and changes how estrogen signals are received. Its effects are also tissue-specific.

In breast tissue, tamoxifen primarily blocks estrogen's effects. In other tissues, including bone, it can produce estrogen-like effects.

This makes tamoxifen particularly interesting from a lipedema perspective.

If lipedema involves altered estrogen-receptor signaling within adipose and stromal tissue, an important unanswered question is:

How does lipedema tissue respond when estrogen receptors are being selectively modulated?

At present, we do not know.

Aromatase Inhibitors: Reducing Estrogen Production

Aromatase inhibitors work very differently.

Medications such as:

Anastrozole • Letrozole • Exemestane

inhibit aromatase, the enzyme responsible for converting androgens into estrogens.

This is particularly relevant after menopause because peripheral tissues—including adipose tissue—become important sites of estrogen production.

By inhibiting aromatase, these medications substantially reduce estrogen synthesis throughout the body.

And that creates another intriguing question for lipedema research.

Aromatase and local estrogen metabolism have already been proposed as potential components of lipedema biology.

So what happens to lipedema tissue when that same enzyme is pharmacologically inhibited for several years?

Again:

We don't know.

The Treatments Should Not Be Grouped Together

This distinction may eventually prove important.

TAMOXIFEN

Estrogen remains present

↓

Estrogen receptor activity is selectively modified

↓

What happens in lipedema tissue?

AROMATASE INHIBITORS

Aromatase is inhibited

↓

Estrogen synthesis falls substantially

↓

Local and systemic estrogen environments change

↓

What happens in lipedema tissue?

These are two different biological interventions.

If lipedema is influenced by estrogen receptors, local estrogen metabolism, adipose-tissue signaling—or some combination of these mechanisms—it is possible that the two types of endocrine therapy could affect lipedema differently.

But possible is not the same as proven.

Could Endocrine Therapy Trigger a Lipedema Flare

This is where we have to be especially careful.

A woman may notice increased pain, swelling, tissue changes or progression while receiving endocrine therapy. That observation is important—but it does not establish that the medication caused the change.

Breast cancer treatment may occur alongside surgery, inflammation, changes in physical activity, alterations in body composition, natural or treatment-induced menopause, lymphatic disruption and other physiological stressors.

Separating these variables would require specifically designed research.

What we need are studies comparing women with established lipedema before and after endocrine therapy, ideally separating women receiving tamoxifen from those receiving aromatase inhibitors.

Until those studies are performed, the most scientifically accurate conclusion remains:

Breast-cancer endocrine therapy may or may not influence lipedema. We currently do not have sufficient evidence to determine whether it does—or whether different endocrine therapies affect lipedema differently.



Breast Cancer is a Major Life Event

For women with lipedema, there may be additional challenges to consider beyond the risk of cancer-related lymphedema.

POTENTIAL ADDITIONAL CONCERNS FOR WOMEN WITH LIPEDEMA

- Flare in lipedema symptoms due to hormonal changes and treatment
- Increased pain, tenderness and inflammation
- Potential for increased fibrosis and tissue changes
- Risk of lymphedema after surgery or radiation
- Emotional and mental health impact
- Importance of a multidisciplinary care team and ongoing support

You are not alone ♥

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Important Clinical Note

Endocrine therapy substantially reduces recurrence and mortality from hormone-receptor-positive breast cancer. Nothing presented here suggests that a woman should stop, avoid or modify medically indicated breast-cancer treatment because she has lipedema.

The purpose of asking this question is not to discourage treatment. It is to encourage research.

References

National Cancer Institute. Hormone Therapy for Breast Cancer. National Cancer Institute.

National Cancer Institute. Tamoxifen Citrate. Updated January 2, 2025.

National Cancer Institute. Aromatase Inhibitor. NCI Dictionary of Cancer Terms.

Katzer K, Hill JL, McIver KB, Foster MT. Lipedema and the Potential Role of Estrogen in Excessive Adipose Tissue Accumulation. *International Journal of Molecular Sciences*. 2021;22(21):11720.

Pinto da Costa Viana D, Caseri Câmara L, Borges Palau R. Menopause as a Critical Turning Point in Lipedema: The Estrogen Receptor Imbalance, Intracrine Estrogen, and Adipose Tissue Dysfunction Model. *International Journal of Molecular Sciences*. 2025;26(15):7074.

Awareness, Not Avoidance

The possibility that endocrine therapy could influence lipedema raises important questions—but it should not overshadow what we already know.

For women with hormone-receptor-positive breast cancer, medications such as tamoxifen and aromatase inhibitors are supported by extensive research demonstrating their ability to reduce breast cancer recurrence and mortality. The potential for lipedema symptoms to change does not outweigh the established benefits of medically indicated cancer treatment.

At this time, we do not even know whether endocrine therapy directly causes lipedema to progress or flare. What we do know is that these medications can produce other effects—including joint and muscle symptoms, fatigue, changes in bone health and, in some women, changes in body composition—that may make managing an existing condition such as lipedema more challenging.

The goal should therefore be awareness and management—not avoidance of treatment.

When Several Stressors Occur at Once

Cancer treatment rarely occurs in isolation.

A woman with lipedema may simultaneously be recovering from surgery, experiencing inflammation, becoming less physically active because of fatigue, undergoing natural or treatment-related hormonal changes, and adapting to endocrine therapy.

For some women, lymphatic disruption from breast cancer surgery or radiation may add another consideration: the potential development of breast-cancer-related lymphedema.

If lipedema symptoms increase during this period, it may therefore be difficult to identify one single cause. Rather than immediately attributing a flare to endocrine therapy, it may be more useful to look at the entire physiological picture.

Managing What We Can

While researchers continue investigating the hormonal biology of lipedema, there are factors women can address with their healthcare teams.

Maintaining appropriate physical activity can be particularly important when fatigue encourages a more sedentary lifestyle. Walking, aquatic exercise, resistance exercise and other individually appropriate movement can support mobility, muscle function, metabolic health and lymphatic flow.

Nutrition can also support overall health during and after cancer treatment. Rather than pursuing restrictive “anti-inflammatory” diets, the emphasis can be placed on a nutrient-dense dietary pattern rich in vegetables, fruits, whole grains, legumes, healthy fats and adequate protein while limiting foods that individually aggravate symptoms or contribute to an overall highly processed dietary pattern.

Weight and body-composition changes, pain, swelling, fibrosis and changes in mobility should also be recognized early rather than simply accepted as unavoidable consequences of treatment.

For women at risk for lymphedema, appropriate education and early assessment of persistent swelling can help distinguish changes related to lymphatic dysfunction from changes associated with lipedema.

With that said, women with Lipedema should be educated

Awareness Creates an Opportunity

Perhaps the most important message is that women should not have to choose between treating their cancer and managing their lipedema.

They can do both.

A woman beginning endocrine therapy who already has lipedema may benefit from documenting her baseline symptoms—including pain, tenderness, swelling, limb measurements, mobility and tissue changes—and monitoring those symptoms throughout treatment.

If changes occur, they can then be discussed with her oncology and rehabilitation teams rather than automatically assuming that the medication must be discontinued.

And importantly:

Never stop or alter breast-cancer endocrine therapy because of suspected lipedema symptoms without discussing it with the prescribing oncology team.



The Research Question Remains

We still need prospective research following women with established lipedema before, during and after breast-cancer endocrine therapy.

Does tamoxifen influence lipedema?

Do aromatase inhibitors?

Does menopausal status matter?

Are changes related to estrogen signaling itself—or to inflammation, body composition, decreased activity, surgery, lymphatic injury or several factors occurring simultaneously?

We don't yet have those answers.

But recognizing the question is the beginning of finding them.

The Takeaway

- Treat the cancer.
- Recognize the lipedema.
- Monitor changes.
- Manage the factors we can influence.
- And continue asking the questions that research has not yet answered.

The purpose of this conversation is not fear.

It is awareness, preparation and better care for women living with both conditions.

References

Lüchinger JE, Pavicic E, Giachino CL, Stute P. Impact of hormones on lipedema development: a systematic literature review. Archives of Gynecology and Obstetrics. 2026;313:60. doi:10.1007/s00404-026-08318-1.

Excellent primary reference for the entire article. It specifically evaluates estrogen, estrogen receptors, menopause and other hormonal mechanisms in lipedema.

Springer

Viana DPC, Câmara LC, Palau RB. Menopause as a Critical Turning Point in Lipedema: The Estrogen Receptor Imbalance, Intracrine Estrogen, and Adipose Tissue Dysfunction Model. International Journal of Molecular Sciences. 2025;26(15):7074. doi:10.3390/ijms26157074.

Best citation for Page 2, including ERα/ERβ, aromatase, intracrine estrogen and the menopause hypothesis. Importantly, this is a proposed mechanistic model rather than proof of causation.

PubMed Central (PMC)

Katzer K, Hill JL, McIver KB, Foster MT. Lipedema and the Potential Role of Estrogen in Excessive Adipose Tissue Accumulation. International Journal of Molecular Sciences. 2021;22(21):11720. doi:10.3390/ijms222111720.

This is one of the foundational papers for discussing estrogen signaling and adipose accumulation in lipedema.

PubMed

Viana DPC, Invitti AL, Schor E. Lipedema as a hormone-sensitive stromal disorder: a four-pathway translational framework. Frontiers in Cell and Developmental Biology. 2026;14:1903835. doi:10.3389/fcell.2026.1903835.

This is particularly valuable because it integrates ERα/ERβ signaling, aromatase, intracrine steroid metabolism, inflammation and stromal/adipose biology, while explicitly acknowledging that much of the mechanistic evidence still needs direct confirmation in lipedema tissue.

Frontiers

Herbst KL, Kahn LA, Iker E, et al. Standard of care for lipedema in the United States. Phlebology. 2021;36(10):779–796. doi:10.1177/02683555211015887.

Useful for the final section on exercise, compression, self-management, healthy eating and maintaining mobility.

PubMed Central (PMC)

National Cancer Institute. Hormone Therapy for Breast Cancer. Updated December 2, 2025.

This is an excellent authoritative source for explaining the differences between tamoxifen, aromatase inhibitors and ovarian suppression. NCI also notes that about 80% of diagnosed breast cancers are hormone-receptor positive.

I WISH I HAD KNOWN MORE ABOUT LIPEDEMA THEN...

This article is personal for me.

I have lipedema, and I am also a breast cancer survivor.

I was only 36 when I underwent treatment for hormone-sensitive breast cancer. My treatment included tamoxifen, hormone cessation, and later Arimidex (anastrozole)—all while my body was experiencing enormous hormonal changes. Years later, I went through another major hormonal transition: menopause.

Looking back, with everything I have since learned about lipedema, I find myself asking questions I did not even know to ask at 36.

What was happening to my adipose tissue as my hormones changed?

Could altering estrogen signaling have influenced my lipedema?

Did 2 rounds of menopause have a greater impact?

Were the changes related to treatment, inflammation, decreased activity, weight and body-composition changes—or simply the natural progression of lipedema?

The answer is: I do not know.

And the research does not know either—not yet.

That is why I wrote this article.

This is not about questioning breast cancer treatment. Those treatments were necessary, and the evidence supporting endocrine therapy for hormone-receptor-positive breast cancer is strong.

It is about recognizing that women can be dealing with more than one condition at the same time.



About Me

My experience as both a breast cancer survivor and a woman living with lipedema has profoundly influenced the way I practice.

After decades in rehabilitation, my own journey taught me to look beyond a diagnosis and consider how the lymphatic system, hormonal changes, inflammation, pain, movement, connective tissue, and overall health interact.

It shifted my focus toward a more integrated approach—combining my background as a PTA, massage therapist, and Certified Lymphedema Therapist to help people better understand and manage the many factors affecting their bodies.

My experiences have also made education a central part of my work: the more we understand, the better we can advocate for ourselves and make informed choices about our care.

Laine Kristin Miller, PTA, LMT, CLT