

Ovaries Appear to Develop a Second Role After Menopause

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STORY AT-A-GLANCE

- Scientists found that the ovaries remain biologically active after reproduction ends in mice, continuing to change at the molecular level instead of becoming inactive once their reproductive role ends
- Aging transformed the mouse ovary from a primarily reproductive organ into one with many immune-related characteristics, suggesting it may continue influencing whole-body biology long after fertility ends
- Important changes begin well before reproductive cycles stop, as communication between ovarian cells gradually becomes less coordinated, helping explain why reproductive function declines over time instead of stopping all at once
- The post-reproductive mouse ovary continued changing at the genetic level even after follicle depletion and fibrosis had stabilized, showing that the organ remains biologically dynamic despite major structural changes already being complete
- Supporting healthy aging after menopause means creating a healthier environment for your remaining ovarian tissue by lowering chronic inflammation, protecting mitochondrial function, reducing exposure to estrogen-like chemicals, and maintaining strong metabolic health

In the video above, Dr. Jen Gunter walks through the basics of what menopause is, how the hormonal transition unfolds, and what you can do about common symptoms. It's a useful primer, but it reflects the conventional understanding that the story essentially



ends once the transition is complete. The research below picks up exactly where that thinking stops.

Menopause is officially defined as the point 12 consecutive months after your last period, but the dramatic symptoms most women associate with it — hot flashes, night sweats, sleep disruption, mood changes, vaginal dryness, and accelerated bone loss — actually belong to the transition leading up to that milestone, when the ovaries are actively winding down hormone production.

That's where one of the biggest misconceptions takes hold: the assumption that by the time a woman reaches postmenopause, the ovaries have simply stopped working.

For a long time, the scientific consensus supported that view. Once the ovary's supply of follicles ran out, researchers treated the organ as little more than inactive scar tissue, a structure that had finished its job and could be safely ignored. That assumption shaped decades of medical thinking and helped justify the routine removal of ovaries during unrelated surgeries.

Yet millions of women now spend decades in postmenopause, which raises a question science has only recently begun taking seriously: what are the ovaries actually doing during all those years? Now two studies are challenging the old view from complementary directions. One investigated how the ovary's molecular and cellular identity transforms after reproduction ends and discovered an organ that, far from going quiet, appears to take on an entirely new biological role.¹

The other built extraordinarily detailed spatial maps of the aging ovary and found that decline begins not with a sudden shutdown but with a gradual loss of the precise coordination among cell types that keeps the organ functioning.² Together, they suggest the postmenopausal ovary deserves far more scientific attention than it has received, and that understanding what changes inside this organ is the first step toward understanding how it influences healthy aging throughout the rest of your body.

The Ovary Kept Changing Long After Reproduction Ended

A study published in *Molecular Human Reproduction* investigated what actually happens to the ovary after reproduction ends, a stage that scientists have historically viewed as biologically quiet. Instead of assuming the postmenopausal ovary simply remains in place without an important purpose, the researchers compared ovaries from reproductively young, reproductively old, and post-reproductive mice to determine how the organ continues to change after its reproductive years end.³

Because human postmenopausal ovarian tissue is difficult to study, the researchers used mice, whose ovarian aging follows many of the same biological patterns seen in women. The three groups were 2-month-old (reproductively young), 18-month-old (reproductively old), and 24-month-old (post-reproductive) mice, with three to four animals per group. The authors note that mice do not menstruate, but argue the model remains useful for identifying conserved mechanisms of ovarian aging.

Rather than focusing only on hormone production or egg loss, the team combined detailed tissue analysis with gene activity measurements to examine the ovary from multiple angles. This allowed them to identify structural changes, shifts in cellular behavior, and differences in which genes were switched on or off at each stage of aging. Their goal was to determine whether the post-reproductive ovary remains biologically active instead of becoming an inactive organ.

- **Researchers found that ovarian aging continued after fertility ended** — Most people assume **menopause** represents the final chapter of ovarian biology. According to the researchers, that assumption doesn't match what they observed. Although the ovaries had already exhausted their supply of follicles, the organ continued changing at the molecular level long afterward.

Instead of reaching a stable endpoint, the ovaries developed an entirely different pattern of activity. The researchers wrote that "the ovary continues to undergo molecular changes after reproductive senescence," meaning the transition didn't stop once reproduction ended. That finding suggests your ovaries continue participating in important biological processes long after fertility disappears.

- **The ovary gradually lost its reproductive identity and adopted a completely different one** — Genes associated with normal ovarian function steadily became less active while an entirely different collection of genes became much more active. Genes work like instruction manuals that tell cells which jobs to perform. When different genes switch on, cells begin behaving differently.

Instead of producing instructions related to reproduction, the post-reproductive ovary increasingly expressed genes involved in immune activity and inflammation. The researchers described this as a shift "from reproductive functionality to an immune-dominant signature." Rather than functioning primarily as a reproductive organ, the ovary appeared to take on responsibilities more commonly associated with the immune system.

- **Immune cells steadily moved into the aging ovary** — The genetic findings matched what researchers observed under the microscope. As the ovaries aged, increasing numbers of immune cells entered the tissue. These included T cells, which help coordinate immune responses, macrophages, which remove damaged cells and debris, and multinucleated giant cells, large immune cells that often appear during long-lasting tissue remodeling or inflammation.

Think of macrophages as the body's cleanup crew. They remove worn-out material and help organize tissue repair. T cells serve as coordinators that direct other immune cells where to respond. Finding substantially more of these cells inside the post-reproductive ovary tells scientists the organ remains biologically busy rather than dormant.

The authors read this shift as ovarian "inflammaging" and propose that "targeting the inflammatory milieu of the ovary may be the foundation for a non-hormonal, non-fertility therapeutic to maintain healthspan for women in the post-reproductive period." In other words, the immune identity is presented as something to counteract, not as a second job the ovary has usefully taken on.

- **Scar-like tissue remained elevated after follicles disappeared** – The researchers examined **collagen**, the structural protein that helps support tissues throughout your body. Excess collagen creates fibrosis, meaning normal tissue becomes stiffer and more scar-like. Picrosirius Red staining, which detects collagen I and III, showed an increasing trend with age that remained elevated in the post-reproductive ovary. The authors report this as a trend rather than a statistically significant increase.

Total follicle numbers were significantly reduced at both 18 and 24 months compared with 2 months, with no further decline between 18 and 24 months, indicating follicle depletion was complete by 18 months.

The continued transformation after follicle depletion occurred mainly at the molecular level. Although the ovary's physical fibrosis had reached a plateau, 230 genes were still differentially expressed between the reproductively old and post-reproductive stages. This distinction shows that the organ remained biologically dynamic even when its major structural changes had stabilized.

- **The aging ovary appeared capable of communicating with the rest of the body** – One of the study's most intriguing discoveries involved proteins that aging ovaries appear capable of releasing into circulation. Researchers identified numerous genes whose protein products are predicted to be secreted outside the ovary. Secreted proteins act like biological messages because they travel to other tissues and influence how those tissues behave.

This finding raises an important possibility. Instead of remaining isolated after menopause, the ovary could continue influencing organs throughout the body by releasing inflammatory signaling molecules.

The authors concluded that the post-reproductive ovary "could be a source of pro-inflammatory signaling mediators with the potential to modulate extra-ovarian tissues." This remains a computational prediction. The authors state that future

studies are needed to validate whether the genes they identified actually produce secreted proteins, and whether those products can be detected in the blood.

None of that undercuts the study's central finding, though. Instead of seeing the ovaries only as organs involved in fertility, this research suggests they remain active participants in whole-body aging. Scientists still need additional research, particularly in humans, but this work provides evidence that the post-reproductive ovary continues playing a meaningful biological role rather than quietly fading into inactivity.

Ovarian Cells Lost Their Perfect Timing Before Fertility Ended

That first study revealed what the ovary becomes after reproduction ends — an immune-active organ still participating in whole-body biology. But it left open the question of how the ovary gets there. A second study, published in *Nature Aging*, tackled that question by investigating how thousands of different ovarian cells coordinate their activities as the organ ages.⁴

The researchers built an exceptionally detailed spatial map of the aging mouse ovary using an advanced genetic mapping technique that captures which cells are active and exactly where they sit within the tissue. The study analyzed 22 mouse ovaries, generating 69 spatial maps that captured more than 610,000 individual measurement points across different stages of the reproductive cycle.

The investigators also developed new computer tools to identify and track 358 oocytes (immature eggs), 668 follicles, and 236 corpora lutea, the temporary structures that form after ovulation. By comparing young, middle-aged, and older mice that were still cycling, they discovered that important biological changes appeared long before reproduction stopped. The three age groups were 10 to 12 weeks, 36 to 40 weeks, and 52 to 54 weeks.

Instead of a sudden collapse at menopause, ovarian aging reflected a gradual breakdown in the precise timing and organization that normally keeps the ovary functioning efficiently.

- **The ovary began losing its internal rhythm years before reproduction stopped –** Ovarian aging started disrupting the timing of normal biological events while the animals were still reproductively active. Healthy ovaries carefully synchronize hormone signals, egg development, and tissue remodeling throughout every reproductive cycle. According to the researchers, that coordination steadily weakened with age instead of disappearing all at once.

Think of a symphony orchestra. Every musician still knows how to play, but if they gradually stop following the conductor's cues, the music drifts out of sync long before anyone stops playing entirely. The researchers concluded that ovarian aging reflects "a progressive breakdown of tissue-level coordination," meaning the individual cells still existed but no longer worked together with the same precision.

That finding matters because many women notice changes in menstrual cycles, hormone balance, and **fertility** years before menopause. This research suggests those changes begin when the ovary's internal timing system starts drifting out of sync rather than when eggs suddenly run out.

- **Egg development became less organized as neighboring cells stopped communicating efficiently –** The study found that aging disrupted folliculogenesis, the carefully controlled process through which immature follicles grow and mature before ovulation. Instead of progressing through clearly defined stages, older ovaries lost much of the orderly communication that guides healthy follicle development.

Follicles don't develop in isolation. Each one constantly exchanges chemical signals with surrounding support cells that deliver nutrients, hormones, and growth instructions. Researchers found that this coordinated conversation became

increasingly disorganized with age, making follicle development less synchronized with the normal reproductive cycle.

The investigators also found that hormone-sensing patterns became uncoupled from cycle stage. In other words, ovarian cells no longer responded to hormonal signals with the same precision seen in younger ovaries, even before reproductive cycles had completely stopped.

- **The ovary struggled to clean up after ovulation** — Every ovulation creates a temporary structure called the corpus luteum, which produces progesterone before naturally breaking down to make way for the next cycle. The study found that this cleanup process became less efficient as ovaries aged.

Researchers observed an accumulation of late-stage, regressing corpora lutea that normally would have cleared. The authors identify these structures by their transcriptional signature rather than by age — they note the method cannot determine how old a corpus luteum is and describe the finding as failed timely clearance, suggesting that normal tissue turnover slowed with age.

Because proper removal of these structures helps prepare the ovary for the next reproductive cycle, delayed clearance disrupted the normal sequence of events.

This discovery gives scientists another explanation for why reproductive cycles often become irregular before menopause. It isn't simply hormone production that changes. The ovary also becomes less efficient at resetting itself between cycles.

- **The physical layout of the ovary became increasingly disorganized** — Healthy follicles normally develop in carefully arranged neighborhoods inside the ovary. The researchers found that this spatial organization gradually disappeared with aging. Preantral and atretic follicles, which cluster tightly in young ovaries, became more scattered in aged ones, reducing the close communication that supports normal development.

Antral follicles behaved differently — they sit as relatively isolated "islands" in both young and old ovaries. Imagine trying to complete a team project after everyone has been moved into separate buildings. Communication slows, coordination suffers, and mistakes become more common. Researchers reached a similar conclusion about ovarian tissue. Loss of this spatial organization reduced the ability of neighboring follicles to influence one another through short-range signaling.

The study also found changes in the extracellular matrix (ECM), the supportive framework that surrounds cells — specifically reduced expression of matrix genes alongside increased matrix-degrading enzymes, which the authors describe as aberrant remodeling. They propose in their concluding model that loss of follicle clustering "may, in part, reflect increased ECM rigidity," citing earlier work; this study did not measure tissue stiffness directly.

- **Inflammation, tissue remodeling, and structural breakdown occurred as interconnected layers rather than separate processes** — Rather than identifying one single cause of ovarian aging, the researchers found several biological processes working together. Increased inflammatory signaling, changes in the extracellular matrix, altered immune activity, and declining tissue organization reinforce one another.

The authors describe a propagating sequence rather than simultaneous onset: disrupted clearance of corpora lutea generates persistent inflammatory niches that "may initiate" broader immune remodeling, and local defects then "propagate across niches" to produce organ-level disorganization.

The authors described these changes as "interconnected layers of ovarian aging," meaning each problem reinforced the others instead of occurring independently. As tissue organization weakened, immune remodeling increased. As inflammation increased, communication between cells deteriorated further. Together, these changes accelerated the decline in normal ovarian function.

Healthy organs depend on millions of cells working together in the right place at the right time. According to this research, ovarian aging isn't defined by one damaged cell type or one failing hormone. It reflects the gradual loss of the remarkable coordination that normally keeps every part of the ovary working as a unified system.

That gradual unraveling is exactly what millions of women experience firsthand during **perimenopause** — the irregular cycles, the unpredictable symptoms, the sense that something has shifted before any test confirms it — and this research now offers a biological explanation for why the decline feels like a slow drift rather than a clean stop.

The authors draw the same parallel to human perimenopause, while stating that "validation in human tissue will be necessary for translating these findings into clinical applications."

Note: The findings from both featured studies are from laboratory or animal research and may not directly apply to human health.

Support Healthy Ovarian Aging by Protecting the Tissue That Remains

The research shows that the ovary continues changing long after menopause instead of simply shutting down, which means the environment you create inside your body still matters. If the postmenopausal ovary is indeed releasing inflammatory signals that influence tissues throughout the body — and the early evidence points in that direction — then the internal environment surrounding that organ matters even more than previously understood.

Chronic inflammation, poor metabolic health, and reduced **cellular energy** place extra strain on tissues that are already adapting to a new biological role. Focus on creating an environment that supports healthy tissue function instead of accepting that everything after menopause is simply decline.

The studies also suggest that your goal after menopause is not simply replacing hormones. Instead, it's supporting the metabolic and cellular environment that allows the tissues you still have to function as well as possible. Many of the same habits that protect your [mitochondria](#), lower inflammation, and improve metabolic health also support healthier hormone balance throughout the rest of your body.

- 1. Reduce the inflammation that places extra stress on aging tissues** — Chronic, low-grade inflammation became a defining feature of the aging ovaries in the research. To reduce this, build most meals around minimally processed whole foods while eliminating [seed oils](#) and ultraprocessed foods.

Seed oils, like soybean, canola, sunflower, or safflower oil, are rich in [linoleic acid](#) (LA), which contributes to inflammation and mitochondrial dysfunction. Keeping LA intake below 5 grams per day while replacing those oils with traditional fats such as grass fed butter, ghee, or tallow helps create a healthier environment for aging tissues.

- 2. Lower your exposure to estrogen-like chemicals in your environment** — Even after menopause, your body continues responding to hormone signals from outside sources. Everyday plastics, personal care products, food packaging, and household products often contain [endocrine-disrupting chemicals](#) that behave like estrogen inside the body.

I recommend storing food in glass or stainless steel instead of plastic, avoiding heating food in plastic containers, choosing products without parabens or phthalates whenever possible, filtering your drinking water, and reducing unnecessary plastic use throughout your home.

- 3. Build a strong metabolic foundation instead of restricting your body** — Healthy tissues require energy to repair themselves, and a postmenopausal ovary that remains biologically active needs that energy just as much as any other organ. I

recommend eating enough healthy carbohydrates — about 250 grams daily for most adults, adjusted for activity level — along with adequate protein from bioavailable sources.

Aim for 0.6 to 0.8 grams per pound of ideal body weight (1.32 to 1.76 grams per kilogram), with one-third coming from [collagen-rich sources](#) like slow-cooked meats or bone broth.

Nutrients found in foods such as liver, pasture-raised eggs, and properly raised animal foods provide vitamin A, vitamin B6, and other compounds that are involved in normal hormone production and cellular energy metabolism. Daily sunlight, resistance exercise, regular walking, and restorative sleep further strengthen the [metabolic foundation](#) that every organ depends on.

- 4. Look beyond blood estrogen alone when evaluating hormone balance** — Even after menopause, blood [estrogen levels](#) tell only part of the story because estrogen stored inside tissues doesn't always match what appears in a blood test. One marker that I believe provides additional insight is prolactin, a hormone whose production rises in response to estrogen activity.

Elevated prolactin, particularly alongside reduced thyroid function, suggests increased estrogen signaling even when blood estrogen appears low. Looking at the [broader hormonal picture](#) provides a more complete understanding of what's happening inside your body. Talk to your health care provider about whether this testing is appropriate for you.

- 5. Focus on restoring balance instead of replacing estrogen** — If you're considering hormone support, carefully weigh the total estrogen burden from medications and environmental exposures.

For many postmenopausal women, bioidentical progesterone, not synthetic progestins, offers a different strategy because progesterone opposes many estrogen effects. Where progesterone is used, I recommend transmucosal delivery

of pharmaceutical-grade bioidentical progesterone mixed with natural vitamin E, rather than a transdermal cream.

The goal is creating an internal environment where the tissues that continue serving you after menopause remain as healthy and resilient as possible.

FAQs About the Ovaries After Menopause

Q: Do the ovaries become inactive after menopause?

A: No. The research reviewed in this article found that although the ovaries stop releasing eggs and lose their reproductive function, they remain biologically active. Both studies were conducted in mice, so this is not yet established in women.

Instead of simply becoming inactive tissue, they continue changing at the molecular level and take on many characteristics associated with the immune system, suggesting they still influence your health long after menopause.

Q: What changes inside the ovaries as they age?

A: Scientists found that the aging mouse ovary gradually loses its reproductive identity while genes involved in immune activity become more active. The ovaries also accumulate immune cells, maintain elevated fibrosis after reproductive aging, and continue changing at the molecular level even after follicle depletion is complete.

Q: Does ovarian aging begin only after menopause?

A: No. The second study found that important changes begin years before menopause. Communication between ovarian cells becomes less coordinated, follicle development becomes less organized, tissue remodeling changes, and the ovary gradually loses the precise timing that keeps the reproductive cycle functioning normally.

Q: Why does this research matter if I'm already postmenopausal?

A: These findings suggest your ovaries continue participating in whole-body biology after reproduction ends. Rather than viewing menopause as the end of ovarian function, the research indicates that the postmenopausal ovary remains an active organ whose biological changes could influence healthy aging throughout the rest of your body. Both research teams state that validation in human tissue is still needed.

Q: What lifestyle habits support healthy ovarian aging after menopause?

A: Focus on the factors that influence cellular health rather than simply replacing hormones. That includes eating a minimally processed diet while avoiding seed oils, reducing exposure to estrogen-like chemicals from plastics and personal care products, maintaining healthy metabolism with adequate carbohydrates and protein, looking beyond blood estrogen alone when evaluating hormone balance, and considering whether bioidentical progesterone is appropriate instead of adding more estrogen.

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, 3} [Molecular Human Reproduction June 10, 2026, Volume 32, Issue 2, gaag038](#)
- ^{2, 4} [Nature Aging June 17, 2026, 6, 1244-1266](#)