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Monitoring *ESR1* Mutations *in Metastatic Breast Cancer*

Patients with advanced receptor-positive breast cancer are typically treated with aromatase inhibitors along with cyclin-dependent kinase 4/6 inhibitors. Together these drugs reduce estrogen levels in the body and block cancer cell growth. Overtime, the cancer cells adapt and develop mutations that cause this drug combination to no longer be effective. One of the more common resistance causing mutations are found in the estrogen receptor (*ESR1*) itself, resulting in estrogen-independent growth of the breast cancer cells. Recent clinical trials (described below) have demonstrated the clinical value of monitoring for these *ESR1* mutations (*ESR1m*), including modifying the drug regimen to maintain the therapeutic response. As a result, monitoring for *ESR1* mutations in advanced receptor-positive breast cancer patients is becoming more commonplace in the oncological care of breast cancer.

PADA-1 Trial

PADA-1, a randomized, open-label, multicenter, phase III trial to evaluate the safety and efficacy of palbociclib in combination with hormone therapy (HT) driven by ctDNA *ESR1* mutation monitoring in ER+, HER2-negative mBC patients.

- The PADA-1 trial aimed to show the efficacy of an early change in therapy at the onset of *ESR1* mutation in blood (b*ESR1*^{mut}), while assessing the global safety of combination fulvestrant and palbociclib.

Results

- Switching therapy to fulvestrant and palbociclib, in patients with detectable *ESR1* mutations, resulted in a doubling of median progression-free survival, from 5.7 months to 11.9 months.
- PADA-1 is the first prospective randomized trial showing that the early therapeutic targeting of b*ESR1*^{mut} results in significant clinical benefit.

PADA-1 Trial

Scan the QR Code or Visit:
www.thelancet.com/article/S1470-2045(22)00555-1/fulltext

SERENA-6 Trial

SERENA-6 is a phase III switching trial of camizestrant in *ESR1*m breast cancer during first-line treatment.

- The phase III SERENA-6 trial uses blood tests to monitor if patients with breast cancer develop *ESR1*m while being treated with an aromatase inhibitor and a CDK4/6 inhibitor. An *ESR1*m test is considered positive if one or more *ESR1* mutations that occur in HR+ mBC are detected. If *ESR1*m is detected, participants have been randomly assigned to either continue with the same aromatase inhibitor or switch to camizestrant while continuing with the same CDK4/6 inhibitor.
- The study is aimed at assessing whether switching to camizestrant prolongs the time before the cancer grows, spreads, or worsens.

SERENA-6 Trial
Scan the QR Code or Visit:
ascopubs.org/doi/10.1200/JCO.2026.44.17_suppl.LBA1007

Scan the QR code or visit
learn.bio-techne.com/49BeLTK

- Camizestrant (Etcamah™) is a newly approved treatment that blocks estrogen receptors, including mutated receptors, reducing the growth and spread of cancer. It is a next-generation oral selective estrogen receptor degrader (SERD) with broad efficacy against *ESR1* mutations as evidenced across multiple clinical studies.[1–3]
- The aim is to treat *ESR1*m clones and extend the duration of control of ER-driven tumor growth, delaying the need for chemotherapy.
- May - September 2026: Camizestrant (Ectamah™) approved in UAE, Saudi Arabia, Japan, the EU, Canada, Brazil, and by the FDA in the US.

Background

- Median PFS (updated at ASCO 2026 showed a 55% reduction in risk of death or progression from early switch to Camizestrant as compared to continued AI treatment (16.8 months vs. 9.2 months, HR=0.45).[3]
- Also updated at ASCO 2026, Median PSF2 data showed a 37% reduction in risk of progression or death of 37% from early switch to Camizestrant as compared to continued AI treatment (25.7 months vs. 19.1 months, HR=0.63).[3] Overall survival (OS) data remains immature.
- *Note: Camizestrant has been shown to be more broadly effective against acquired ESR1 mutations than fulvestrant in clinical studies.*

1. Baird RD, et al. *Clin Cancer Res*. 2025;31(20):4244-4254. doi:10.1158/1078-0432.CCR-25-1198
2. Oliveira M, Pominchuk D, Nowecki Z, et al. *Lancet Oncol.* 2024;25(11):1424-1439. doi:10.1016/S1470-2045(24)00387-5
3. Turner NC, et al. *Lancet Oncol*. 2026;27(8):941-958. doi:10.1016/S1470-2045(26)00287-1

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