Thoughts about what I learned: There was an overwhelming amount of interesting, thought provoking science presented and discussed at SABCS and I expect to be reviewing and digging into it for many months. I heard some very interesting lectures (for example, re the interplay between epigenetics, exercise and adiposity in the context of cancer care) as well as learning about potentially practice changing results from the studies presented this year at SABCS, such as the Phase 3 EMBER-3 trial. My biggest take-away however, is that patient advocate involvement in conferences such as SABCS is of vital importance and needs to be expanded. There are not enough platforms that lead to nuanced dialogue between researchers and advocates about how and what gets studied. While SABCS is one such platform I believe that we need more. Patient voices help researchers clarify and improve their questions, their protocols, the quality of their research and are accelerants to improving science and saving lives.

Summary of two sessions

1) I attended the "**Risk-Reduction and Early Detection: Mechanisms of Obesity Related Risk for Breast Cancer and Approaches to Risk Reduction**" session held on 12/10.

Catherine Duggan outlined how over a lifetime, with increased obesity, epigenetic changes in response to dietary, exercise and other environmental factors such as the social determinants of health can increase risk of cancer by modifying immune cells, reducing tumor suppressor genes and creating a pro-inflammatory environment. Epigenetic changes can persist and be passed down to children, thereby changing phenotype and metabolism in the next generation and increasing risk of breast cancer. She explained that epigenetic responsiveness to external factors raises the possibility of intervention but that there is currently no evidence that epigenetic changes are reversible. Duggan further spoke about "epigenetic age" vs chronological age and spoke about some ways that it could be used to screen for stratified breast cancer risk. She commented that while reversibility might not pan out, a better diet and exercise can slow epigenetic aging. Finally she explained that adipose cells retain epigenetic memory even after weight loss and are primed to return to their former state, making it almost impossible to maintain weightloss. Finally she discussed some future research avenues such as the use of SERMs, CRISPR, and interventions in early life for risk reduction.

Neil Iyengar spoke about fat loss and metabolic improvements in relation to the use of incretin mimics (ie GLP1 agonists and like drugs). My understanding is that it is the inflammation associated with excess adiposity that is onco-genic, and he explored this by discussing how studies of normal weight people with "metabolic obesity" (and the histological bio-marker that he said was "crown-like") showed that they had increased breast cancer. He outlined some of their ongoing research about way in which they might intervene using weight to reduce risk but I got the impression that the research was still inconclusive.

The exercise lecturer John Jakicic made it very clear that the most important factorwas the “quality” of the tissue rather than the “quantity”. By this he meant that exercise improves outcomes by increasing strength and lean mass and that interventions that focus on weightloss are off-target, that in fact weightloss without improvements in activity can cause lean mass loss which is very problematic.
I found the session to be very thought provoking and plan to follow-up with the lecturers with a few questions.
2) The Education Session called “**The Biology and Treatment Implications of Organ Specific Metastases**” was very engaging. Three lecturers covered brain, liver and bone metastases (Christine Hodgdon of GRASP represented the patient perspective).

First, Diana Cittelly spoke about brain mets and said that 15-20% of metastatic breast cancer patients go on to develop symptomatic brain mets. Interventions can be problematic because they are invasive, cause cognitive problems and medication often does not cross the blood brain barrier. She reviewed the differences in incidence based on age at time of diagnosis commenting that it suggested that estrogen is involved in neuro inflammation. She reviewed the possible benefit of combination therapy to treat brain mets as well as the role of FGFR1 expression in progression of brain mets. She made a good point that the mechanisms for brain met progression likely differ in ER- vs ER+ disease and so require separate models to conduct research.
Rochelle Johnson spoke about bone mets, reviewing how breast cancer cells disseminate to bone early-on and seed to other metastatic sites, as well as markers that might be used to monitor for bone mets. She mentioned that immune cells are excluded and are “exhausted” in bone metastases. Therapies currently include anti-resporptive agents but bone anabolic therapies (teiparatide and abaloparatide) are being explored in the metastatic setting with abaloparatide showing efficacy and safety in pre-clinical models.
Peter Vermeulen spoke about how the histopathological type of liver mets have effects on outcomes, with “encapsulated” type being associated with better outcomes as compared to “replacement” style. He said that this information should be used to design treatments specific to histological type, and that more research was needed to develop identification tools.

My experience networking with other patient advocates:
A key highlight of SABCS is the chance to spend face to face time with other patient advocates, strengthening relationships, meeting new people and learning about the excellent work being done by patients. I believe that this time together accelerates and increases the impact that we can have as advocates.

I was especially glad to be able to spend time with other lobular advocates. The ILC community might be small, but it is uniquely tight-knit and active and I left San Antonio energized and with a larger, more robust network to lean on.
I was also able to reconnect with advocates (and researchers!) that I met during my ProjectLEAD training, an earlier opportunity which wouldn't have been possible for me to take advantage of without LBCA's scholarship and support.
I am already looking forward to the 2025 conference!

How I'll use the experience and new knowledge in my advocacy:
I'm excited to incorporate the science that I learned and the personal connections that I made into my ILC advocacy. I'll use the new scientific information to better educate other patients and the general public about issues regarding ILC. The relationships I made with researchers and other members of the breast cancer community are new avenues of collaboration that I'll continue to explore.