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A major aspect of the research in my lab is transcriptional regulation during normal mammary gland development and breast cancer development. Among the several transcriptional regulators that we are studying, Cofactor of BRCA1 (COBRA1) binds to tumor suppressor BRCA1 and exhibits a chromatin unfolding activity. We also show that COBRA1 functions as a subunit of the negative elongation protein (NELF) complex to modulate RNA polymerase II movement and transcription of estrogen-responsive genes in breast cancer cells. This represents a novel mechanism by which hormone-mediated gene expression is regulated. We are currently using molecular and cellular approaches to examine the exact impact of COBRA1/NELF on gene regulation, stem/progenitor functions, and tumorigenesis in normal mammary glands and other major hormone-responsive organs.

In a separate research direction, we have been investigating the impact of adipose stromal cells (ASCs) on breast cancer development. ASC is a major constituent of the breast and it produces a plethora of breast cancer-related factors including estrogens. We are currently using three-dimensional (3D) co-culture systems and xenograft mouse models to study the signal transduction pathway(s) that regulate the estrogen-producing capability of ASCs. In parallel, we are also interested in identifying and characterizing novel ASC-secreted, estrogen-independent factor(s) that may influence the behaviors of breast tumor cells. By looking outside the “box” of breast tumor cells, we wish to emphasize the importance of tumor microenvironment and the impact of stromal factors on breast cancer development. Results from our work may shed light on the molecular basis of several well-documented risk factors for breast cancer, including obesity and high mammographic density.