

SEMINAR

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Hosted by A/P Liou Yih Cherng

Targeting estrogen receptors and cofactors for precision medicine in breast cancer



By Xu Wei

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Xu's laboratory focuses on targeting estrogen receptors for breast cancer therapy. Estrogen receptors (ERs) exist in two forms, ER α and ER β , which have opposing roles in cell proliferation. In a small molecule library screen, Dr. Xu identified a natural plant product Dip G that significantly decreased ER α but increased ER β stability. Via distinct mechanism from the existing agents for endocrine therapy, this compound also significantly promoted degradation of mutant ER α that is found in ~25% of patients with metastatic ER α -positive breast cancers. Biological functions of these estrogenic compounds are currently being investigated in cell-based and breast cancer mouse models. Dip G may be developed as novel agents for treating metastatic, endocrine-resistant breast cancers caused by ER α mutations.

Dr. Xu graduated from Peking Univ. with B.S. in Chemistry and Institute of Biophysics, Chinese Academy of Science with M.S. in Biophysics. In 1999, Dr. Xu received PhD in Biochemistry from U. of Iowa. After postdoc training in Dr. Ronald Evans lab in the Salk Institute, San Diego, Dr. Xu joined University of Wisconsin-Madison as a tenure tracked assistant professor in 2005. In 2014, Dr. Xu was promoted to the rank of full professor. Dr. Xu's research focuses on transcriptional and epigenetic regulation in breast cancer. She has received numerous national awards including Department of Defense Era of Hope Scholar Award and Society of Toxicology Achievement Award. Dr. Xu's research is supported by fundings from NIH, DOD, Susan Komen Breast Cancer Foundation, and Falk Research Foundation.

Dr. Xu's laboratory has also employed biochemical and functional genomic approaches, as well as mouse genetics to decipher the contribution of histone arginine methylation to the epigenetic control of cancer cells. The major focus of Xu lab is on a protein arginine (R) methyltransferase CARM1/PRMT4, a nuclear hormone receptor co-activator. Using genetically engineered CARM1 knockout cell lines, Dr. Xu has identified a number of non-histone substrates for CARM1 and elucidated the function of protein arginine methylation in cancer initiation, progression and metastasis.