



Basal-like breast tumors comprise a heterogeneous group that accounts for about 15% of all breast cancers. They are highly aggressive and generally fail to respond to targeted therapies. Thus, there is a great need to identify novel vulnerabilities and develop novel therapies for this aggressive breast tumor type. The epithelial-to-mesenchymal transition (EMT) is often reactivated during tumor formation. Compared to other breast cancer subtypes, basal-like breast cancers display the highest degree of mesenchymal and stem-like features, which are responsible for tumor initiation, metastasis and therapeutic resistance.

We have recently identified genes that regulate EMT in breast cancer using a genome scale screen and identified several RNA splicing factors that are upregulated in basal-like breast cancers and promote the breast tumor formation.

We propose to systematically characterize the role of alternative splicing in breast cancer and EMT using genomic, molecular and cellular biology, and computational approaches, and investigate its role in breast tumor metastasis and therapeutic resistance. These studies may lead to new approaches to treat basal-like/triple negative breast cancers.

Dr. Ji Li obtained his BSc in Biological Sciences from Peking University, China, and completed his PhD study at New York University School of Medicine. During his doctoral studies, Dr. Li investigated post-translational control of cell cycles in cancer and discovered a novel protein complex that contributes to tumorigenesis. Dr. Li is currently a postdoctoral fellow at Dana-Farber Cancer Institute/Harvard Medical School in the laboratory of Dr. William C. Hahn.