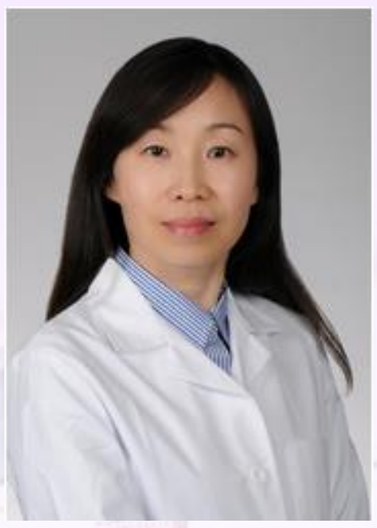
巴斯德讲坛-精英系列
Pasteur Colloquium-Elite

Exploiting vulnerabilities in therapeutically recalcitrant HER2 positive breast cancer



[Speaker] **Dr. Qi Wang**

[Time] **14:00-15:30PM, November 15, 2017**

[Host] **Prof. Changbin Chen**

[Venue] **A0201, Life Science Research Building**

[Speaker Introduction]

2014-Dec-present **Assistant Professor**, Department of Pathology and Laboratory, Medical University of South Carolina, Charleston, SC, USA.
2007-2014 **Postdoctoral Fellow,** Department of Cancer Biology, Dana-Farber Cancer Institute, Harvard Medical School, Boston, MA, USA.
2001-2004 **Ph.D.** Harbin Medical University, Harbin, China

[Abstract]

Human epidermal growth factor receptor-2 (HER2) is constitutively activated by overexpression or gene amplification in approximately 15-20% of human breast cancers. HER2 serves as a bona fide oncogene, which confers a more aggressive tumor phenotype and associates with a higher rate of recurrence and mortality. Trastuzumab (Herceptin) and lapatinib, two approved and widely used treatments for HER2 positive (HER2+) breast cancer, have significantly improved the outcome for breast cancer patients and opened up an era of cancer treatment known as targeted therapy. Unfortunately, many patients either fail to respond to targeted therapies despite the presence of the target receptor in their tumors, or respond initially but go on to develop resistant disease. The treatment options for these highly aggressive and ultimately lethal tumors are extremely limited. I have been interested in investigating the mechanisms driving breast cancer cells that have evaded HER2 targeted treatments and identifying novel avenues for therapeutic intervention. Here I will discuss three potential therapeutic strategies for therapeutically recalcitrant HER2+ breast cancer. 1) Selective requirement of p110a in PTEN loss-induced resistance to HER2-targeted therapy. 2) Overcoming therapeutic resistance in HER2+ breast cancers with CDK4/6 Inhibitors. 3) Targeting CDK7 dependent transcription in HER2+ breast cancer. The translation of these findings into clinical application is expected to significantly reduce the therapeutic resistance of targeted therapy, even augment the first-line targeted therapy, in turn increasing breast cancer patient survival.

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