conferenceseries.com
939th Conference

May 2017 | Volume 2 | Issue 2 | ISSN: 2572-4118
Breast Cancer: Current Research

Proceedings of

4th World Congress on

BREAST CANCER

May 08-10, 2017
Singapore

Exhibitor

OncoDNA
THE CANCER THERANOSTIC COMPANY

Conference Series

One Commerce Center-1201
Orange St. #600, Wilmington, Zip 19899, Delaware, USA

Kemp House, 152 City Road
London EC1V 2NX, UK

Toll Free: Japan: 81-345780247 | Singapore: +65 8008526126 | USA/Canada: 1-800-216-6499 | UK: 0-800-098-8455
General Number: 1-650-889-4686

Email: breastcancersummit@cancersummit.org | breastcancersummit@oncologyseries.com | breastcancersummit@nursingconference.com

conferenceseries.com
939th Conference

4th World Congress on

Breast Cancer

May 08-10, 2017 Singapore

Keynote Forum

(Day 1)

Sudhakar Jha, Breast Can Curr Res 2017, 2:2(Suppl)
http://dx.doi.org/10.4172/2572-4118-C1-001

Sudhakar Jha

National University of Singapore, Singapore

TIP60 inhibits epithelial-mesenchymal transition program in breast cancer: A HAT with many tricks

HIV-Tat-interacting protein 60 KDa (TIP60) is a lysine acetyltransferase implicated in transcription, DNA damage response and apoptosis. It is known to be downregulated in multiple cancers. Recent studies have shown that TIP60 downregulation correlates with node positivity, metastasis and poor survival rate. Epithelial-mesenchymal transition (EMT) is considered as an important step in cancer metastasis. During this process, there is an overexpression of EMT inducers such as Snail2 (also known as Slug) and repression of cell adhesion molecules such as E-cadherin and EpCAM. Additionally, previous report has demonstrated that E-cadherin and EpCAM expression were repressed by DNA hypermethylation on their promoter region during EMT. In this conference, I will be discussing data that show TIP60 expression partially abrogates cell migration and metastatic potential of breast cancer cells both in vitro and in vivo models. Mechanistically, we show this is through its ability to destabilize DNMT1 and inhibit Snail2 expression. Depletion of TIP60 stabilizes DNMT1 and increase Snail2 level, resulting in the EMT. Activation of DNMT1-Snail2 axis in the absence of TIP60 represses expression of epithelial markers by increased DNA methylation on their promoter region. In pathophysiological scenario, we find TIP60 to be significantly down-regulated in breast cancer patients with poor Overall Survival (OS) and Disease-Free Survival (DFS) prognoses. These data suggest that levels of TIP60 can be a prognostic marker of disease progression and stabilization of TIP60 could be a promising strategy to treat cancers.

Biography

Sudhakar Jha's group is interested in understanding the regulation of chromatin remodeling complexes and their role in cancer prevention and intervention. Chromatin remodeling complexes play an important role in maintaining chromatin organization as they create a histone code that is read by specific readers resulting in an active or repressed chromatin. Dr. Jha's group has purified and characterized chromatin-remodeling complexes implicated in transcription and DNA damage response (Mol Cell 2009, 34: 521-533). Dr. Jha's group has identified the role of TIP60, a histone acetyltransferase in DNA damage response pathway (Mol Cell Biol 2008, 28: 2690-2700) and RVB1, a component of TIP60 complexes to be required for activity of this complex (Mol Cell Biol 2013, 33: 1164-74). Following which, his group has discovered Human Papillomavirus (HPV) E6 and Adenovirus (AdV) oncogenes to destabilize TIP60 (Mol Cell 2010, 38: 700-711; Oncogene 2013, 32: 5017-25 and Oncogene 2016, 35:2062-74). Dr. Jha's have recently identified and new cellular regulator of TIP60 and have demonstrated its role and significance in epithelial-mesenchymal transition and breast cancer progression (Oncotarget 2015, 6:41290-306 and J Mol Cell Biol 2016, 85: 384–399).

csisjha@nus.edu.sg

Notes:

Wang Xuefei, Breast Can Curr Res 2017, 2:2(Suppl)
http://dx.doi.org/10.4172/2572-4118-C1-001

Wang Xuefei

Peking Union Medical College Hospital, China

CTC immune escape mediated by PD-L1

Breast cancer is the most common malignant tumor in women around the world. CTC (circulating tumor cell) is associated with the breast cancer patients' cancer-related deaths and prognosis. The recently circulating clusters were found and its metastasis and tumor formation ability is 23~50 times as CTC. However, its mechanism has not been clarified. These days, researchers have successfully completed CTC cluster separation, CTC cell culture, and PD-L1 was found to be related with histological grading of tumor. Meanwhile, the high expression of PD-L1 in CTC surface has also been reported. Since PD-L1 can mediate Treg to play the role of immunosuppression, we propose that CTC with positive PD-L1 is easier to connect PD-L1, immune cells and CK cytokines etc. Treg cells can protect CTC from being attacked by the immune system through the immunosuppression. Meanwhile, they can weaken CTL killing ability and trigger more MDSC. Finally, CTC formed the metastasis. To explore this hypothesis we have analyzed CTC and PD-L1 mRNA expression on CTC in 10 metastatic breast cancer patients and 10 primary breast cancer patients. We have also analyzed the relationship between clinical pathological features and PD-L1 expression on CTC, through overall and split chi square test. The results show that in the total 20 patients, 15 have more than 1 CTC in 7.5 ml peripheral blood. Among the 15 patients, each one has at least 1 CTC showing PD-L1. We found PD-L1 on CTC is related to the tumor size (P=0.012) lymph node status (P=0.001) and PR status (P=0.037). In tumor size group, we can see statistical difference between T2 and T3 (P=0.003), while in node status group statistical difference can be found in N1 vs. N3 (P=0.000) and N2 vs. N3 (P=0.015). However, we didn't see difference of PD-L1 on CTC in metastatic and non-metastatic patients (P=0.418). Next, we are preparing for the cell experiment to further discover it.

Biography

Wang Xuefei is a Medicine Doctor graduated from PUMCH (Peking Union Medical College and Hospital). Currently, she is the fellow of the breast surgery department in PUMCH. She is also a member of Beijing Breast Disease Society of Young Academic. These years, her researches are focused on metastatic breast cancer, especially on CTC of breast cancer. She has also obtained the patent of CTC hemodialysis, meanwhile, participated in a number of national projects, including Beijing Municipal Science and Technology project, National 11th Five-Year issue, National 12th Five-Year issue. She has published more than 14 articles and books, including 4 SCI articles, and did oral and poster presentation in many breast cancer related conferences.

1210548954@qq.com

Notes: