「分子病態セミナー」を開催

2015 年 9 月 29 日

９月２９日（火）に、がん進展制御研究所４階会議室において、乳がんや大腸がんなど固形がんの新規分子標的とその分子機構について、多くの優れた研究成果をあげている Cancer Therapeutics and Stratified Oncology, Genome Institute of Singapore 教授 Qiang YU 先生を招いて、分子病態セミナーを開催しました。

セミナーでは「Therapeutic targeting of molecular events that drives metastasis, chemo resistance and cancer stamens in refractory breast cancer」という演題で講演をしていただきました。

セミナーには、研究所内外の教職員、大学院生等約４０名が参加し、活発な質疑応答や意見交換が行われました。

分子病態セミナー

Title: Therapeutic targeting of molecular events that drives metastasis, chemoresistance and cancer stemness in refractory breast cancer.

Presenter: **Professor Qiang YU**
Senior Group Leader
Cancer Therapeutics and Stratified Oncology
Genome Institute of Singapore

Yu先生は、乳がんや大腸がんなど固形がんの新規分子標的とその分子機構について、
多くの優れた研究成果をあげています。
皆様奮ってご参加ください。

日 時：９月２９日（火）午後１７：００～１８：００

場 所：がん進展制御研究所　４階　会 議 室

Although early diagnosis and application of new therapies have significantly improved survival of breast cancer patients, many patients still develop relapse and died of metastasis years after surgery and treatment. Triple-negative breast cancer (TNBC), characterized by tumors that do not express ER, PR, or HER-2 genes, represents one of the most aggressive subtypes of breast cancers with poor outcomes. Although TNBC in general is more responsive to chemotherapy, patients often develop resistance to chemotherapy, leading to early metastatic relapse. One important goal of breast cancer research is to find solutions to reduce the drug resistance, tumor recurrence, and metastasis, which remains to be a challenging task. NF-kB-mediated inflammatory cytokine network has been considered one of the major molecular events that contribute to breast cancer metastatic progression and chemoresistance. However, clinical development of NF-kB inhibitors has not been successful due to severe liver toxicity. Alternatively, targeting oncogenic events that confer NF-kB dependency in cancer cells but not normal cells may provide new treatment opportunities. In this talk, I will describe our efforts in uncovering new molecular targets that regulates NF-kB activity in TNBC. In particular, we have identified an inflammatory kinase whose overexpression/activation promotes metastasis, cancer stemness and acquired resistance to chemotherapy. Pharmacologic inactivation of this kinase and associated gene network abolished TNBC invasion and metastasis in vitro and in vivo and effectively reversed the chemoresistance, and thus providing a new therapeutic approach for refractory TNBC which is currently incurable.

1. Feng M, et al., “RASAL2 activates RAC1 to promote triple-negative breast cancer progression.” J Clin Invest, 124, 5291-5304, 2014.
2. Wee ZN,et al., ″EZH2-Mediated Inactivation of IFN-γ-JAK-STAT1 Signaling Is an Effective Therapeutic Target in MYC-Driven Prostate Cancer.” Cell Reports 8, 204-216, 2014
3. Tan J, et al., “PDK1 signaling toward PLK1-MYC activation confers oncogenic transformation, tumor-initiating cell activation, and resistance to mTOR-targeted therapy.” Cancer Discov 3, 1156-1171, 2013.

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