

「がん進展制御研究所 国際セミナー・女性研究者セミナー」開催

2019年9月17日

9月17日（火）、金沢大学がん進展制御研究所 4F 会議室において、エモリー大学 ウィンシップがん研究所（アメリカ）准教授 Sumin Kang 先生をお招きして、がん進展制御研究所国際セミナー・女性研究者セミナーを開催しました。

セミナーでは、「Targeting cancer signaling nodes to overcome metastasis and chemotherapy resistance」という演題で、1. がんの転移プロセスにおいて、グルタミン代謝酵素である GDH1 が、アノイキスに抵抗し、転移を促進することを、またその阻害剤が転移の抑制に効果を発揮することを示すこと、2. シスプラチン耐性機構において、MAST1 の MAPK 経路における役割を果たしていることについて講演していただきました。がんの薬剤耐性および転移の克服など、本研究所が取り組むべき課題に対してのアプローチを考える上で、大変有意義なセミナーとなりました。

セミナーには、研究所内外の教職員、大学院生等 31 名が参加し、活発な質疑応答や意見交換が行われました。



“Targeting cancer signaling nodes to overcome metastasis and chemotherapy resistance”

Dr. Sumin Kang

Associate Professor
Winship Cancer Institute
Department of Hematology and Medical Oncology,
Emory University School of Medicine, USA

September 17, 2019, Tues, 5pm~6pm

Cancer Research Institute, 4F Conference Room

Metastatic or therapy resistant cancers are often considered incurable. Although dysregulated metabolism and kinase signaling have been demonstrated in cancer cells, the precise mechanism remains unclear. We performed transcriptomics screen and identified GDH1 as a critical metabolic factor which provides anti-anoikis and pro-metastatic signals through activating CamKK2 and AMPK that promotes tumor metastasis in lung cancer. Targeting GDH1 with a GDH specific inhibitor R162 attenuated tumor metastasis in mice. We also performed cisplatin synthetic lethal partner screen using kinome shRNA library and identified MAST1 as a promising target to overcome cisplatin resistance. Mechanistically, we demonstrated that cisplatin dissociates cRaf from MEK1 to inhibit the MAPK pathway and identify MAST1 as a main cisplatin resistance driver that replaces cRaf to reactivate the MAPK pathway. Through a drug repurposing study, lestaurtinib was identified as a MAST1 inhibitor. Lestaurtinib effectively inhibits MAST1 kinase activity and cancer cell proliferation in combination with platinum-based compounds including cisplatin and carboplatin.

Biography: Dr.Kang's research has focused on how intricate molecular communication networks evolve to control cell growth, survival, and proliferation in cells, and how disruption of these processes leads to cancer, with a particular focus on the role of cellular protein kinase signaling and metabolic reprogramming in tumorigenesis, tumor metastasis, and chemotherapy resistance in human cancers. The findings have been published in journals including *Cancer Cell*, *Nat Cell Biol*, *Mol Cell*, *J Clin Invest*, and *Oncogene*.

がん進展制御研究所 平尾 敦

(連絡先：遺伝子・染色体研究分野 内線6755)