Therapeutic strategies to target cancer-specific pathways and vulnerability

Lecturer: SUZUKI Motoshi

(Professor, Molecular Oncology, Fujita Health University School of Medicine)

鈴木 元 先生

(藤田医科大学医学部分子腫瘍学講座/教授)

Date: December 4 (Mon) from 5:30 p.m.

令和5年12月4日(月)17:30~



Place: Lecture room 2, Medical Education & Library Building 3F

医学教育図書棟3階 第2講義室

Abstract

Our analysis of 149 lung cancer specimens showed that some metabolic genes were aberrantly expressed in cancer. For example, high expression levels of a ceramide metabolic gene CERS6 were associated with lung cancer metastasis. To understand underlying mechanisms, we performed IP-MS/IP-WB analyses and identified an actin binding protein LASPI as a novel CERS6 interaction partner. In another analysis, we showed that low expression of DNA replication/repair gene POLD4 was associated with poor prognosis, and that POLD4 shortage might cause chromosomal instability. We performed a DNA fiber assay to show that in POLD4-high cells, but not in POLD4-low cells, ssDNA was accumulated in a PRIMPOL-dependent manner. Because cancer cells often show addictive nature to the highly active pathways for their survival, we suppose that PRIMPOL pathway could be targeted to induce a lethal phenotype. Our strategy to achieve this goal will be presented and discussed.

- ◆Inviter: Professor. OKADA Seiji (Dep. Hematopoiesis) /岡田 誠治 教授(造血・腫瘍制御学)
- ◆Essay/レポート宛先(To Prof. OKADA): okadas@kumamoto-u.ac.jp
- ◆Essay/レポート宛先(CC: Student Affairs Sec./医学教務): iyg-igaku-3@jimu.kumamoto-u.ac.jp