Mechanisms of leukemogenesis by

epigenetic/transcriptional regulators

Lecturer: YOKOYAMA Akihiko

(Team leader, Tsuruoka Metabolomics Laboratory, National Cancer Center)

横山 明彦 先生 (国立研究開発法人 国立がん研究センター・鶴岡連携拠点 がんメタボロミクス研究室/ チームリーダー)

Date: June I5 (WED) from 5:30 p.m. 令和4年6月15日 (水) 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

※ This seminar will be held in a face-to-face. /
 今回のセミナーは対面形式で開催されます。
 ※ This seminar will be recorded. /本セミナーは録画されます。

Abstract

Leukemia is a major cause of childhood cancer. Despite of the improvement of chemotherapy, some subtypes of leukemia remain refractory to date. Leukemia is caused by two different types of mutations. One is of signaling pathways that induce constitutive activation of proliferation signals. The other is of epigenetic/transcriptional regulators that are involved in differentiation and self-renewal. MLL, a histone methyl transferase, and MOZ, a histone acetyl transferase, are implicated in transcriptional maintenance and self-renewal. These epigenetic factors are mutated in leukemia by chromosomal translocations, which generate fusion proteins that constitutively activate their transcriptional activities. Expression of MLL fusions and MOZ fusions transforms normal hematopoietic progenitors by constitutively activating their target genes such as HOXA9 and MYC to induce refractory leukemia. We have studied the molecular mechanisms of the disease onset by these fusions, by elaborate structure/function analyses to determine which domain structures are necessary for leukemic transformation. These studies led to the development of novel drugs that inhibit these epigenetic/transcriptional regulators. I will talk about the story how understanding of molecular mechanisms of disease led to the drug development.

◆Inviter: Prof. MATSUI Hirotaka (Diagnostic Medicine)/松井 啓隆 教授(臨床病態解析学)

◆Essay/レポート宛先(To Prof. MATSUI):hmatsui@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