



Department of Biotechnology
Ministry of Science and Technology
Government of India

DBT



National Institute of
Advanced Industrial Science
and Technology

AIST



DBT - AIST International Laboratory for Advanced Biomedicine



DAILAB

Classroom for Advanced & Frontier Education CAFE

DAiLAB-CAFE

Series - 026

Date & Time: April 9, 2018 (4:00 to 5:00 p.m. JST)

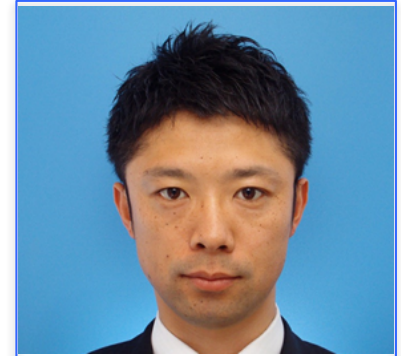
Venue: Central 5-41; 2F (Conference Room # 1)

Speaker: Shinichi HASAKO

Title: Drug discovery targeting EGFR mutations for lung cancer therapy

Affiliation: TAIHO PHARMACEUTICAL CO., LTD.

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Abstract: Among cancer-related deaths, deaths from lung cancer are the most common in the world, and approximately 80% to 85% of lung cancers are classified as non-small cell lung cancer (NSCLC). Somatic mutation of the epidermal growth factor receptor (EGFR), most of which are concentrated in the region of exon 18-21, is a major oncogenic driver and is present in approximately 30-50% and 10-20% of NSCLC in Asians and in Americans and Western Europeans, respectively. EGFR is a transmembrane glycoprotein and belongs to the erbB family of tyrosine kinase receptors. Activating mutations in the EGFR kinase domain induce ligand-independent constitutive activation and subsequent downstream molecule phosphorylation, leading to cancer cell growth and survival. Various tyrosine kinase inhibitors (TKIs) targeting EGFR mutations have been developed as anticancer agents: gefitinib, erlotinib, afatinib, for the primary treatment of patients with NSCLC harboring activating mutations, including exon 19 deletions and L858R, and osimertinib has been approved for treating patients with NSCLC with T790M acquired resistance mutations. However, the clinical response of NSCLC driven by EGFR exon 20 insertion mutations to EGFR-TKIs is much lower because plasma concentrations of these drugs in clinical settings are kept low by dose-limiting toxicity caused by wild type (WT) EGFR inhibition. TAS6417 was discovered as a mutant EGFR selective inhibitor. In preclinical studies, TAS6417 exhibited cellular potency to inhibit EGFR exon 20 insertion mutations rather than WT EGFR, leading to in vitro antiproliferative activity and efficacious in vivo anti-tumor effect against cancer cells harboring EGFR exon 20 insertion mutations.



Series 26

Speaker: Shinichi HASAKO

Topic: Drug discovery targeting EGFR mutations for lung cancers

Date: 8th April., 2018
(16:00-17:00 h JST)

Host:
DAILAB@AIST,
Japan



Thanks for participation!

National Institute of Advanced Industrial Science & Technology, Japan



IIT-Delhi, India

Guru Nanak Dev University, India



University of Sri
Jayewardenepura, Sri
Lanka



Peking University, China



Manipal University, India



Brawijaya Univ., Indonesia

