December 2015

HMT Newsletter

Dear Friends,

As I write this, 2015 is quickly drawing to a close. The year has flown by with many interesting projects and meetings. I hope 2015 was very successful for all of you. At HMT we had very good year. We met many researchers and were able to provide our metabolic profiling services. We are very happy to hear from our many satisfied clients. In 2016, the HMT team will consciously work hard to support your study. So please challenge us anytime for your metabolomics needs. Please have a happy and safe holiday season.

Sincerely,

Tsutomu Hoshiba
CEO
Human Metabolome Technologies America

HMT Updates

Campaign

Special Offer for metabolic profiling

Basic Scan or CARCINOSCOPE package: $4,000 / 6 samples

- Suitable for small polar molecules
- Variety of samples Cells, Tissues, Blood, Stool
- Full statistical analysis and biological interpretation
- Offer expires on December 31, 2015

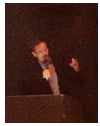
Event Information

Biotech Showcase 2016
January 11-13, San Francisco, California, USA

We are looking for the client and partner who are interested in the installation of our unique metabolomics technology. Please contact us if you will join the event.

Dr. Lewis C. Cantley presents at International Symposium

November 20, Tokyo, Japan



At the National Cancer Center (NCC) in Japan, HMT sponsored a lecture by Dr. Lewis C. Cantley about advances in metabolic signal transduction in cancer. Dr. Cantley, Director of the Cancer Center of Weill Cornell Medical College and New York-Presbyterian Hospital, is focused on understanding the biochemical pathways that regulate normal mammalian cell growth and the defects that cause cell transformation. Dr. Cantley visited Japan to provide his metabolomic perspectives for cancer research to Japanese scientists participating in the International Symposium of the Princess Takamatsu Cancer Research Fund on November 19, in addition, he kindly accepted our invitation to speak at an educational workshop opened for many researchers who could not attend the meeting.

In the mid-1980s, Dr. Cantley conducted research on mechanisms of cellular responses to hormones and growth factors that led to the discovery of phosphoinositide 3-kinase (PI3K) signaling pathway. Dr. Cantley presented a review about signal transduction in cancer metabolism, and the latest updates in related drug development. He also introduced an interesting concept for a nutritional approach to cancer as a metabolic disorder which has been published recently (*Science*, Nov 5, 2015, J. Yun *et al.*).

In addition, Dr. Hideki Makinoshima, a researcher in NCC, gave a lecture about the mechanism of EGFR inhibitor resistance, characterized by glycolytic activity as revealed by HMT's quantitative energy metabolism profiling (CARCINOSCOPE) and 13C labeling analysis (F-SCOPE) service (*J. Biol. Chem.*, Jul 10, 2015, H. Makinoshima *et al.*).

We would like to appreciate Dr. Cantley, Dr. Makinoshima and all attendees for sharing their time at this exciting seminar.

Featured articles

Comprehensive analysis of transcriptome and metabolome analysis in Intrahepatic Cholangiocarcinoma and Hepatocellular Carcinoma

Yoshiki Murakami *et al.*, *Sci. Rep.*, **5**: 16294.

Intrahepatic cholangiocarcinoma (ICC) and hepatocellular carcinoma (HCC) are liver originated malignant tumors. Of the two, ICC has the worse prognosis because it has no reliable diagnostic markers and its carcinogenic mechanism is not fully understood. The aim of this study was to integrate metabolomics and transcriptomics datasets to identify variances if any in the carcinogenic mechanism of ICC and HCC.

Effect of STAT3 Inhibition on the Metabolic Switch in a Highly STAT3-activated Lymphoma Cell Line

Cancer Genomics Proteomics, **12**, *pp*. 133-142., 2015

Signal transducer and activator of transcription (STAT)3 is involved in a metabolic shift in cancer cells, the Warburg effect through its pro-oncogenic activity. To develop efficient STAT3 inhibitors against cancer cells, novel proteomic and metabolic target molecules need to be explored using multi-omics approaches in the context of STAT3 gene inhibition-mediated tumor growth suppression.

Hepatocyte β-Klotho regulates lipid homeostasis but not body weight in mice

Kanako Kobayashi *et al.*, *FASEB J.*, in press

β-Klotho (β-Kl), a transmembrane protein expressed in the liver, pancreas, adipose tissues, and brain, is essential for feedback suppression of hepatic bile acid synthesis. Because bile acid is a key regulator of lipid and energy metabolism, we hypothesized potential and tissue-specific roles of β-Kl in regulating plasma lipid levels and body weight. By crossing β-kl-/- mice with newly developed hepatocyte-specific β-kl transgenic (Tg) mice, we generated mice expressing β-kl solely in hepatocytes (β-kl-/-/Tg). Gene expression, metabolomic, and in vivo flux analyses consistently revealed that plasma level of cholesterol, which is over-excreted into feces as bile acids in β-kl-/-, is maintained in β-kl-/- mice by enhanced de novo cholesterogenesis.

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