Research Seminar

FYI

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FYI

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eIF5-mimic protein represses non-AUG translation by competing with eIF5

Friday, July 7, 2017
3:00 – 4:00 pm
at Seminar Room (D105)

The translation factor eIF5 interacts with the tRNAi-binding protein eIF2, thereby regulating the frequency of non-AUG initiation. The translational repressor termed eIF5-mimic protein (5MP) counteracts the activity of eIF5 by competing for eIF2, and as we have recently shown exhibits pro-oncogenic properties. In the human genome, tumor suppressor genes, including PTEN, and oncogenes, such as cMyc, possess in-frame, upstream non-AUG start codons. In addition, translation of NAT1/eIF4G2/DAP5 involved in cap-independent translation of numerous oncogenes including cMyc starts at the GUG codon. Here we show that eIF5 increases the expression of NAT1, cMyc and other non-AUG initiated proteins and, conversely, that the 5MP paralog, 5MP1 or 5MP2, decreases their expression. Strong non-AUG initiation depends on the nucleotide context beyond the Kozak consensus. Such strong non-AUG signals are the targets of converse regulation by eIF5 and 5MP. Non-AUG initiation of uORFs modifies the effect of paird uORFs involved in translational control through eIF2 inhibition. We propose that repression of non-AUG translation of cancer-regulating genes contributes to pro-oncogenic properties of 5MP proteins.

Host: Dr. Hiroshi Takagi, Lab of Applied Stress Microbiology (ex. 5420)