Amy H Tang, Pancreat Disord Ther 2017, 7:3 (Suppl)
DOI: 10.4172/2165-7092-C1-004

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Developing therapy-responsive prognostic biomarkers, and conquering undruggable oncogenic k-ras-driven pancreatic cancer by attacking its major k-ras vulnerability and the k-ras signaling gatekeeper – the siah proteolysis machinery

Hyperactive K-RAS signaling is a major menace that drives aggressive cancer cell dissemination, tumor progression and systemic metastasis in human pancreatic cancer. Counteracting K-RAS hyperactivation in attempt to reverse malignant transformation and inhibit latent tumor spread is an important goal in pancreatic cancer biology. Instead of targeting an upstream signaling module such as EGFR/K-RAS/B-RAF/MEK/MAPK/ERK/AKT/mTOR, we targeted the most downstream signaling module in the K-RAS signaling pathway called the SIAH-dependent proteolytic machinery. SIAHs are the human homologs of seven-in-absentia (SINA), an evolutionarily conserved RING E3 ligase, an essential downstream signaling module and a critical gatekeeper required for proper K-RAS signal transduction. We found that inhibiting SIAH function is highly effective to abolish well-established and late-stage pancreatic tumor growth and metastasis in our pre-clinical studies. These findings demonstrate that SIAH is indeed an attractive, logical and potent anti-K-RAS therapeutic target for us to develop new and effective anticancer strategy against human pancreatic cancer. Through our work, SIAH has emerged as a new and effective drug target against oncogenic K-RAS hyperactivation in pancreatic cancer. As one of the most evolutionarily conserved signaling components, SIAH is ideally and logically positioned to become a next-generation anti-K-RAS drug target in human pancreatic cancer. By attacking this most downstream gatekeeper critical for the proper oncogenic K-RAS signaling transmission, we will be in a position to halt the genesis, progression and metastasis of the deadliest forms of human pancreatic cancer in the future. We aim to translate anti-SIAH therapy to benefit our pancreatic cancer patients in the clinic.

Biography

Amy H Tang received a BS in Biophysics from Fudan University, and a PhD in Biochemistry and Molecular Biology from Pennsylvania State University. She completed her Post-doctoral training supported by two Post-doctoral fellowships, (1) NIH Post-doctoral fellowship and (2) Leukemia and Lymphomia Society (LSA) Senior Post-doctoral Fellowship at UC Berkeley. She is a Professor of Cancer Biology at Eastern Virginia Medical School. She is a lead Pancreatic Cancer Researcher and a Recipient of the highly prestigeous national cancer award – 2010 AACR-PanCAN Innovative Award. She and her research team have developed an innovative strategy and therapy-responsive prognostic biomarkers to control and eradiacte oncogenic K-RAS-driven pancreatic cancer in preclinical and clinical settings. She has published numerous papers in peer-reviewed journals, and secured NIH/DOD/AACR-pancreatic cancer action network innovative grant/lustgarden foundation for pancreatic cancer research. She has served as reviewers at multiple NIH study sections for more than 10 years, and has served as the Editorial Advisory Board Member and reviewer of numerous scientfic journals.

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