CURRICULUM VITAE

Liang-Yi Hung, PhD

I. Biographical

Name: Liang-Yi Hung

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II. Education

Ph.D. (1995/8 ~ 2000/10) Graduate Institute of Life Sciences, Academia Sinica and National Defense Medical Center, Taipei, Taiwan, Republic of China

III. Experience

Associate Professor (2012/8 ~ present)

Institute of Bioinformatics and Biosignal Transduction, College of Bioscience and Biotechnology, and Center for Gene Regulation and Signal Transduction Research, National Cheng Kung University, Tainan 70101, Taiwan

Assistant Professor $(2010/8 \sim 2012/7)$

Institute of Bioinformatics and Biosignal Transduction, College of Bioscience and Biotechnology, and Center for Gene Regulation and Signal Transduction Research, National Cheng Kung University, Tainan 70101, Taiwan

Assistant Professor $(2009/2 \sim 2010/7)$

Institute of Biosignal Transduction, College of Bioscience and Biotechnology, and Center for Gene Regulation and Signal Transduction Research, National Cheng Kung University, Tainan 70101, Taiwan

Research Assistant Professor $(2005/2 \sim 2009/1)$

Department of Pharmacology, College of Medicine, and Center for Gene Regulation and Signal Transduction Research, National Cheng Kung University, Tainan 70101, Taiwan

Institute of Biomedical Sciences, Academia Sinica, Taipei, Taiwan, Republic of China

IV. Major interests

- (1) To study the functional role of CPAP in the TNF α -induced NF κ B mediated genes activation in HBV-associated HCC
- (2) To study the molecular mechanism of overexpressed Aurora-A in tumor cells
- (3) To study the target therapy in Aurora-A-overexpressing cancers
- (4) To investigate the potential role and the transcription regulation of Aurora-C in tumorigenesis

VI. Publications (From 2011~now)

- Jen-Hui Tsou, Kung-Chao Chang, Pey-Yi Chang-Liao, Shu-Ting Yang, Chung-Ta Lee, Ya-Ping Chen, Yi-Chao Lee, Bo-Wen Lin, Jenq-Chang Lee, Meng-Ru Shen, Chin-Kai Chuang, Wen-Chang Chang, Ju-Ming Wang* and <u>Liang-Yi Hung*</u>. Aberrantly expressed AURKC enhances the transformation and tumorigenicity of epithelial cells. <u>Journal of Pathology</u>. 2011; 225: 243-254. SCI.
- 2. Wu S-R, Li C-F[#], <u>Hung L-Y</u>[#], Huang A-M, Tseng J-T, Tsou J-H and Wang J-M. CCAAT/enhancer binding protein delta mediates TNFalpha-induced aurora kinase C transcription and promotes genomic instability. <u>Journal of Biological Chemistry</u> **2011**; 286(33): 28662-28670. (# equal contribution) SCI.
- 3. Hsu C-C, Lee Y-C, Yeh S-H, Chen C-H, Wu C-C, Wang T-Y, Chen Y-N, <u>Hung L-Y</u>, Liu Y-W, Chen H-K, Hsiao Y-T, Wang W-S, Tsou J-H, Tsou Y-H, Wu M-H, Chang W-C, Lin D-Y. The 58-kDa microspherule protein (MSP58) is a novel brahma-related gene 1 (BRG1)-associated protein that modulate the p53-p21 senescence pathway. <u>Journal of Biological Chemistry</u> 2012; 287(27): 22533-22548. SCI.
- 4. Ya-Ping Chen, <u>Liang-Yi Hung</u>, Yan-Shen Shan, and Kung-Chao Chang. ALK-positive large B-cell lymphoma presenting with jejunal intussusception. <u>European Journal of Haematology</u> **2013**; 90(3):261.
- 5. Shu-Ting Yang, Chia-Jui Yen, Chein-Hsien Lai, Yih-Jyh Lin, Kung-Chao Chang, Jenq-Chang Lee, Yao-Wen Liu, Pey-Yi Chang-Liao, Lu-Shin Hsu, Wen-Chang Chang, Wen-Chun Hung, Tang K. Tang, Yi-Wen Liu and <u>Liang-Yi Hung</u>*. SUMOylated CPAP is required for IKK-mediated NF-κB activation and enhances HBx-induced NF-κB signaling in HCC. <u>Journal of Hepatology</u> 2013; 58(6): 1157-1164.
- 6. Ya-Ping Chen, Hui-Ju Lin, Jiann-Shiuh Chen, Ming-Ying Tsai, Hsing-Pang Hsieh, Jang-Yang Chang, Nai-Feng Chen, Kung-Chao Chang, Wen-Tsung Huang, Wu-Chou Su, Shu-Ting Yang, Wen-Chang Chang, <u>Liang-Yi Hung</u>*, and Tsai-Yun Chen*. CDKN1A-mediated Responsiveness of *MLL-AF4*-positive Acute Lymphoblastic Leukemia to Aurora Kinase-A Inhibitors. <u>International Journal of Cancer.</u> 2014 Aug 1; 135(3): 751-762.

- 7. Kung-Chao Chang, Yu-Chu Wang[#], <u>Liang-Yi Hung</u>[#], Wan-Ting Huang[#], Jen-Hui Tsou, Jones Dan, Hsiang-Lin Song, Yu-Min Yeh, Lin-Yuan Kao and L. Jeffrey Medeiros. Monoclonality and Cytogenetic Abnormalities in Hyaline Vascular Castleman Disease. <u>Modern Pathology</u> **2014 Jun**; 27(6): 823-831. (**DOI**: **10.1038/modpathol.2013.202**) (# equal contribution)
- 8. Yu-Cheng Lee, Jenny Que, Yu-Chia Chen, Jen-Tai Lin, Yih-Cherng Liou, Po-Chi Liao, Yu-Peng Liu, Kuen-Haur Lee, Li-Ching Lin, Michael Hsiao, <u>Liang-Yi Hung</u>, Chi-Ying Huang and Pei-Jung Lu. Pin1 acts as a negative regulator of the G2/M transition through an interplay with the Aurora A/hBora complex. <u>Journal of Cell Science</u> 2013 Nov 1; 126 (Pt21): 4862-4872.
- 9. Kung-Chao Chang, Wei-Chao Chang, Yao Chang, Liang-Yi Hung, Chien-Hsien Lai, Yu-Min Yeh, Tu-Wei Chou, and Chung-Hsuan Chen. Ran GTPase-Activating Protein 1 Is a Therapeutic Target in Diffuse Large B-Cell Lymphoma. PLoS One 2013 Nov 6; 8(11): e79863.
- 10. Ding-Yen Lin, Chi-Chen Huang, Ya-Ting Hsieh, Hsin-Chuan Lin, Ping-Chieh Pao, Jen-Hui Tsou, Chien-Ying Lai, <u>Liang-Yi Hung</u>, Ju-Ming Wang, Wen-Chang Chang and Yi-Chao Lee*. Analysis of the interaction between Zinc finger protein 179 (Znf179) and promyelocytic leukemia zinc finger (Plzf). <u>Journal of Biomedical Science</u> 2013 Dec 20; 20:98. (DOI:10.1186/1423-0127-20-98)
- 11. Chen Chang, <u>Liang-Yi Hung</u>, Tung Tran Thanh, Chein-Hsien Lai, and Kung-Chao Chang*. Congenital Peribronchial Myofibroblastic Tumor with Features of Maturation in the Older Infant: Report of Two Cases with Literature Review. <u>Histopathology</u> **2014** Apr; 64(5): 755-777.
- 12. Bo-Wen Lin[#], Yu-Chu Wang[#], Pey-Yi Chang-Liao[#], Ya-Ju Lin, Shu-Ting Yang, Jen-Hui Tsou, Kung-Chao Chang, Yao-Wen Liu, Joseph T. Tseng, Chung-Ta Lee, Jenq-Chang Lee^{*} and Liang-Yi Hung^{*}. Overexpression of Aurora-C impaired the spindle checkpoint by promoting the degradation of Aurora-B. Cell Death and Disease 2014 Mar 6; 5:e1106.
- 13. Pao-Lin Kuo, Yung-Ling Huang, Christine Chin-Jung Hsieh, Jenq-Chang Lee, Bo-Wen Lin and Liang-Yi Hung*. STK31 is a cell-cycle regulated protein that contributes to the tumorigenicity of epithelial cancer cells. **PLoS ONE 2014** Mar 25; 9(3):e93303.
- 14. Ying-Ren Chen, <u>Liang-Yi Hung</u>, Kung-Chao Chang. Mucosa-associated lymphoid tissue-type lymphoma presenting as a urethral caruncle with urinary bladder involvement. <u>Int J Urol.</u> 2014 Oct; 21(10):1073-4.
- 15. En-Ju Chou, <u>Liang-Yi Hung</u>, Chieh-Ju C Tang, Wen-Bin Hsu, Hsin-Yi Wu, Pao-Chi Liao, Tang K Tang. Phosphorylation of CPAP by Aurora-A Maintains Spindle Pole Integrity during Mitosis. <u>Cell Rep.</u> **2016 Mar 29**; 14(12):2975-87.
- 16. Yi-Han Dai, <u>Liang-Yi Hung</u>, Rho-Yu Chen, Chien-Hsien Lai, Kung-Chao Chang. ON 01910.Na inhibits growth of diffuse large B-cell lymphoma by cytoplasmic sequestration of sumoylated C-MYB/TRAF6 complex. <u>Transl Res.</u> **2016 Sep**; 175:129-143.e13.
- 17. Kung-Chao Chang, Jen-Chieh Lee[#], Yu-Chu Wang[#], <u>Liang-Yi Hung</u>[#], Yenlin Huang, Wan-Ting Huang, Ren-Ching Wang, Tse-Ching Chen, Yi-Shan Tsai, L Jeffrey Medeiros. Polyclonality in Sclerosing Angiomatoid Nodular Transformation of the Spleen. **Am J Surg Pathol. 2016 Aug 11.** [Epub ahead of print] (# equal contribution)

Clinical role of epigenetic silencing of miR-137 in early colorectal carcinogenesis

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MicorRNA-137 is silenced in human colorectal cancer tissues and colon polyps. Our study showed that the decreased expression of miR-137 is significantly different in various types of polyp which maintain different potentials to lead to CRC development. The expression of miR-137 gradually decreases during the process of colorectal carcinogenesis. Receiver operating characteristic curve (ROC) analysis indicates that the loss of miR-137 expression in colon polyps can serve as a biomarker to predict the predisposition of colorectal carcinogenesis. By cell model and xenograft animal model, the enforced expression of miR-137 in colorectal cancer cells can inhibit cell proliferation and tumor formation, induce G2/M arrest, and lead to apoptosis. The expression pattern of miR-137 and Aurora-A or COX-2 is negatively correlated in human colorectal cancer tissues and colon polyps. Those effects induced by overexpressed miR-137 can be rescued by the overexpression of Aurora-A. In summary, our study suggests that the loss of miR-137 expression in colon polyps can serve as a biomarker to predict the tendency toward to CRC formation through the impaired inhibitory effect of Aurora-A. The investigation of the regulatory mechanism of miR-137-mediated Aurora-A inhibition may shed new light on the early prognosis of cancer therapy for CRC in the future.

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