Stanford

Wen-yang Lin

Postdoctoral Scholar, Genetics

Bio

HONORS AND AWARDS

- AACR-Genentech Fellowship in Lung Cancer Research, American Association for Cancer Research, US (2017 - 2019)
- University of California Postdoctoral Fellowship Award, Tobacco-Related Disease Research Program, University of California, US (2017 - 2019)
- Stanford School of Medicine Dean's Postdoctoral Fellowship, Stanford University, US (2017)
- Washington Tunnicliffe Writing Fellowship, University of Washington, US (2016)
- International Provost Grants, The State of Washington, US (2012 - 2013)
- Washington Research Foundation Hall Fellowship, University of Washington, US (2011)
- Studying Abroad Scholarship, Ministry of Education, Taiwan (2008 - 2010)
- Excellent Paper Award, International Symposium on Bioengineering, Taiwan (2007)
- National Taiwan University Presidential Award, Taiwan, National Taiwan University, Taiwan (2001 - 2002)

PROFESSIONAL EDUCATION

- Bachelor of Arts, National Taiwan University (2005)
- Master of Science, University of California Los Angeles (2010)
- Doctor of Philosophy, University of Washington (2016)
- Ph.D., University of Washington, Seattle, WA, US , Biology - Neuroscience (2016)
- Master of Science, University of California, Los Angeles, CA, US , Biomedical Engineering (2010)

STANFORD ADVISORS

- Monte Winslow, Postdoctoral Faculty Sponsor

PATENTS

- Wen-Yang Lin, Ruoh-Huey Uang. "Taiwan Patent I378069 Method of Manufacturing Core-Shell Nanostructure", Industrial Technology Research Institute, Dec 1, 2012
- Wen-Yang Lin, Ruoh-Huey Uang. "China P.Rep. Patent CN101837455B Method of Manufacturing Core-Shell Nanostructure", Industrial Technology Research Institute, Oct 26, 2011
- Wen-Yang Lin, Ruoh-Huey Uang. "United States Patent US20100166976A1 Method of Manufacturing Core-Shell Nanostructure", Industrial Technology Research Institute, May 22, 2009

LINKS

- Stanford Winslow Lab: http://winslowlab.stanford.edu/people.html

Research & Scholarship

CURRENT RESEARCH AND SCHOLARLY INTERESTS

Lung cancer is a major health burden, leading to more deaths than the next four major cancer types combined. Despite advances in clinical cancer genome sequencing and the development of many targeted therapies, understanding the relationship of tumor genotype to therapeutic response remains a major obstacle to translating existing drugs into effective cancer treatments in the clinic. Pharmacogenomic analysis of tumor response is often extrapolated from the analysis of patients' tumor responses or modeled using in vitro cultured cell line systems, but investigating the effect of tumor genotype on drug response in cell lines, patient-derived xenograft models, or patients themselves all have severe limitations. Genetically-engineered mouse models have emerged as particularly rigorous in vivo systems with which to test early stage oncology therapies and represent tractable models with which to investigate the impact of tumor genotype on therapy response. Current genetically-engineered mouse models are time-consuming, cost-intensive, and have unavoidable technical and experimental variability that has limited their use in translational studies.

We have established a novel multiplexed somatic genome-editing approach that will allow the quantification of genotype-specific drug responses. This in vivo approach will increase in precision and scope of translational cancer pharmacogenomics studies. To quantify the effect of tumor suppressor gene inactivation on lung cancer growth, we established a system that combines somatic Cas9-mediated gene inactivation with existing genetically-engineered mouse models to generate ~30 different lung tumor genotypes. To quantify the exact size of each tumor and determine the size distribution of each tumor genotype, we induce tumors with barcoded vectors and use high-throughput sequencing and statistical approaches to determine the number of cancer cells in each tumor. We will combine our quantitative pooled genome-editing approach with pre-clinical treatments to uncover genotype-specific therapy responses. We will quantify the responses of ~30 different genotypes of tumors to several therapies that have been shown to have genotype-specific effects in lung adenocarcinoma models. This will extend our understanding of the genomic modifiers of treatment responses and define the experimental and statistical parameters to enable the most efficient use of these models for translational studies. Finally, by performing pre-clinical/co-clinical trials for targeted therapies across >30 tumor genotypes in parallel we will generate a pharmacogenomic map connecting lung adenocarcinoma genotype to targeted therapy response. Our ongoing clinical interactions will allow validation of our pharmacogenomic predictions in lung adenocarcinoma patients.

This flexible system can incorporate additional tumor suppressors, allows for the investigation of genotype-specific responses to other therapies including immuno-therapies, and be adapted to other cancer types. The techniques described in this proposal are ideally positioned to become a mainstay of pre-clinical/co-clinical trial design.

PROJECTS

- A Quantitative Multiplexed Platform for the Pharmacogenomic Analysis of Lung Cancer - Stanford University (September 20, 2016 - September 19, 2019)

Publications

PUBLICATIONS

- **Functions of the SLC36 transporter Pathetic in growth control** *FLY*
  Lin, W., Williams, C. R., Yan, C., Parrish, J. Z.
  2015; 9 (3): 99-106
- **The SLC36 transporter Pathetic is required for extreme dendrite growth in Drosophila sensory neurons** *GENES & DEVELOPMENT*
  Lin, W., Williams, C., Yan, C., Koledachkina, T., Luedke, K., Dalton, J., Bloomsburg, S., Morrison, N., Duncan, K. E., Kim, C. C., Parrish, J. Z.
  2015; 29 (11): 1120-1135
- **Coordinate control of terminal dendrite patterning and dynamics by the membrane protein Raw** *DEVELOPMENT*

Lee, J., Peng, Y., Lin, W., Parrish, J. Z.
2015; 142 (1): 162-173

- **PINK1-Parkin Pathway Activity Is Regulated by Degradation of PINK1 in the Mitochondrial Matrix** *PLOS GENETICS*
  Thomas, R. E., Andrews, L. A., Burman, J. L., Lin, W., Pallanck, L. J.
  2014; 10 (5)

- **Antioxidant effects of betulin on porcine chondrocyte behavior in gelatin/C6S/C4S/HA modified tricopolymer scaffold** *Materials Science and Engineering: C*
  Lin, W., Lin, F., Sadhasivam, S., Savitha, S.
  2010; 30 (4): 597–604

- **The dose dependent effects of betulin on porcine chondrocytes** *Process Biochemistry*
  Lin, W., Sadhasivam, S., Lin, F.
  2009; 44 (6): 678–684

PRESENTATIONS

- Functions of the SLC36 transporter Pathetic in growth control - 73rd Society for Developmental Biology Annual Meeting (July 17, 2014 - July 21, 2014)
- Identification of an amino acid transporter for extreme dendrite growth in Drosophila sensory neurons - 2014 Northwest Developmental Biology Meeting (March 13, 2014 - March 16, 2014)
- The effects of Chinese herbal medicine on chondrocytes for cartilage tissue engineering - 3rd World Congress on Regenerative Medicine (October 18, 2007 - October 20, 2007)