

BIOGRAPHICAL SKETCH

NAME: Yi-Ching Tang

eRA COMMONS USER NAME (credential, e.g., agency login): YICTANG

POSITION TITLE: Faculty Associate

EDUCATION/TRAINING

INSTITUTION AND LOCATION	DEGREE (if applicable)	Start Date MM/YYYY	Completion Date MM/YYYY	FIELD OF STUDY
I-Shou University, Kaohsiung, Taiwan	B.S.	09/2002	06/2007	Biological Science and Technology
National Chi-Nan University, Nantou, Taiwan	M.S.	09/2007	06/2009	Biomedicine and Engineering
University of Texas Health Science Center at Houston, Houston, Texas	M.S.	08/2016	07/2018	Biomedical Informatics
University of Texas Health Science Center at Houston, Houston, Texas	Ph.D.	08/2018	07/2022	Biomedical Informatics
University of Texas Health Science Center at Houston, Houston, Texas	Postdoc	12/2022	05/2024	AI for drug discovery

A. Personal Statement

As a Faculty Associate with a strong interdisciplinary background in Bioinformatics, Computational Biology, and Machine Learning, I am well-positioned to support research efforts in computational drug discovery and development. My doctoral studies focused on addressing translational challenges in in-silico drug discovery, utilizing transfer learning techniques to bridge the gap between in-vitro and in-vivo drug response. Prior to joining McWilliam School of Biomedical Informatics, UTHealth Houston, I collaborated closely with the Taiwan's Center for Disease Control (CDC) to develop computational strategies for combating drug resistance in Tuberculosis.

My extensive data science expertise and successful experience in project completion enable me to contribute significantly to project planning, data pipeline development, and data analysis. Currently, my research is focused on leveraging AI techniques to engineer therapeutic proteins, leveraging my proficiency in Bioinformatics and computational techniques to drive innovation in this field.

B. Positions, Scientific Appointments and Honors**Positions and Scientific Appointments**

2024 -	Faculty Associate
2022 - 2024	Postdoctoral Fellow, McWilliams School of Biomedical Informatics, Houston, TX
2017 - 2023	Graduate Research Assistant, School of Biomedical Informatics, UTHealth, Houston, TX
2015 - 2016	Bioinformatics Analyst, Linkou Chang Gung Memorial Hospital, Taoyuan
2012 - 2015	Bioinformatics Analyst, Center of Biodiversity, Academia Sinica, Taipei

Honors

2021	Presidential poster award, Pharmacology Global Research Network Annual Conference
------	---

Other Experience and Professional Memberships

- 2019 - 2021 Member, Pharmacology Global Research Network (PGRN)
2019 - 2021 Member, American Society of Human Genetics (ASHG)
2018 - 2020 Member, American Medical Informatics Association (AMIA)

C. Contributions to Science

1. Comparative genomics approaches for discovering therapeutic gene modules and drug targets

Characterization of genome-wide gene expression and polymorphisms can delineate phenotypic variations and be used to understand underlying biological functions. I participated in two genome projects when I worked at the sequencing center. I designed and developed a single mutation calling pipeline to characterize genome-wide genetic differences among *A. Cinnamomea* tissues and conducted statistical analysis on the bulk-RNA sequencing data to identify differentially expressed genes exclusive to each tissue. The pathway analysis results for those gene expression signatures allow future investigators to estimate the medicinal effects for industrial production. Drug-resistant tuberculosis is a global health issue. However, inhibiting the growth of the bacteria requires specific treatment because of the rapid mutation rate that will develop drug resistance during transmission. Our center sequenced the first local extensive drug-resistant TB strain, enabling us to find drug-resistant mutations specific to the local population. Using my experience developing variant calling pipelines, I developed approaches to detect single-point mutations that can cause damage to their downstream protein products. We then applied this to discover the connection between drug resistance and *M. tuberculosis*.

Specifically, our functional assay result showed that the novel mutation K247N found in gene *gyrB* reduces the binding affinity of ofloxacin to its target and causes the bacteria to become resistant to the drug.

- a. Wang WF, Lu MJ, Cheng TR, **Tang YC**, Teng YC, Hwa TY, Chen YH, Li MY, Wu MH, Chuang PC, Jou R, Wong CH, Li WH. Genomic Analysis of Mycobacterium Tuberculosis Isolates and Construction of a Beijing Lineage Reference Genome. *Genome Biol Evol.* 2020 Feb 1;12(2):3890-3905. PubMed Central PMCID: PMC7058165.
- b. Lu MY, Fan WL, Wang WF, Chen T, **Tang YC**, Chu FH, Chang TT, Wang SY, Li MY, Chen YH, Lin ZS, Yang KJ, Chen SM, Teng YC, Lin YL, Shaw JF, Wang TF, Li WH. Genomic and transcriptomic analyses of the medicinal fungus *Antrodia cinnamomea* for its metabolite biosynthesis and sexual development. *Proc Natl Acad Sci U S A.* 2014 Nov 4;111(44):E4743-52. PubMed Central PMCID: PMC4226107.

2. AI solutions for anti-cancer drug response prediction

High-throughput drug screening in cancer cell lines has been developed as a pre-clinical method to measure the drug response of tumors. Gene expression profiles in pre-treatment cell lines have been demonstrated as the most predictive feature compared to somatic mutations and copy number variations. However, the reality of tumor molecular profiles is more complex, and gene expression profiles might be best thought of as interactive networks gathering a set of genes that carry out similar functions. To test this hypothesis, I developed informatics methods integrating multiple-omics data with biological pathways and used them to predict drug response to anti-cancer drugs. We observed better generalizability and explainability of underlying molecular interactions associated with drug response. More importantly, our approaches demonstrated potential translatability from cancer cell lines to tumors.

- a. **Tang YC**, Powell RT, Gottlieb A. Molecular pathways enhance drug response prediction using transfer learning from cell lines to tumors and patient-derived xenografts. *Sci Rep.* 2022 Sep 27;12(1):16109. PubMed Central PMCID: PMC9515168.
- b. **Tang YC**, Gottlieb A. SynPathy: Predicting Drug Synergy through Drug-Associated Pathways Using Deep Learning. *Mol Cancer Res.* 2022 May 4;20(5):762-769. PubMed PMID: 35046110.
- c. **Tang YC**, Gottlieb A. Explainable drug sensitivity prediction through cancer pathway enrichment. *Sci Rep.* 2021 Feb 4;11(1):3128. PubMed Central PMCID: PMC7862690.

3. Tissue specificity analysis of transcription factors

Gene expression has been used to understand the association with disease, unraveling that signals of disease-induced transcriptional changes can delineate variations in phenotypes or disease subtypes. Transcription factors (TFs) are essential proteins that participate in gene regulation, but their roles are often overlooked by cis-based models that consider only the cis-element of the gene. In this study, we investigated genetic variability in TFs and their effect on gene expression. I provide computational support for this project's data pre-processing, model development, and evaluation. We discovered tissue-specific genetic regulation patterns from human

whole blood and skeletal muscle tissue. Specifically, our TF models can partially explain gene expression for 1,035 genes more than the cis-based models.

- a. Lu H, **Tang YC**, Gottlieb A. Tissue-Specific Variations in Transcription Factors Elucidate Complex Immune System Regulation. *Genes (Basel)*. 2022 May 23;13(5) PubMed Central PMCID: PMC9140347.

Complete List of Published Work in My Bibliography:

<https://www.ncbi.nlm.nih.gov/myncbi/yi-ching.tang.1/bibliography/public/>