Curriculum Vitae

Name
Yao-An Shen

Current Position
Associate Professor, Department of Pathology / Graduate Institute of Clinical Medicine, College of Medicine, Taipei Medical University, Taipei 110301, Taiwan
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Education & Professional Experiences (Times New Roman, 11 point)
Dr. Shen earned his Ph.D. in Biochemistry and Molecular Biology from National Yang Ming Chiao Tung University, Taiwan. Driven by a strong commitment to cancer research, he pursued postdoctoral training at Johns Hopkins University (JHU), where he specialized in cancer stem cells (CSCs) and translational oncology. He joined Taipei Medical University as an Assistant Professor in 2019 and was promoted to Associate Professor in 2024. He currently also serves as Director of the Core Laboratory of Good Tissue Practice (GTP), leading innovative research in precision oncology and cell therapy.

Research Interests (Times New Roman, 11 point)
Dr. Shen's research interests center on CSCs and their roles in therapeutic resistance, tumor recurrence, and metastasis. His work integrates translational oncology, cancer metabolism, and precision medicine to identify key transcription factors, metabolic vulnerabilities, and neoantigen targets in CSCs. He develops first-in-class small-molecule inhibitors, immune cell–based therapies, and nanomedicine delivery systems to overcome chemoresistance and improve clinical outcomes. His research further explores metabolic reprogramming, tumor microenvironment interactions, and advanced cell engineering platforms, aiming to bridge fundamental molecular discoveries with innovative therapeutic strategies in cancer treatment.

Selected Publications (※3-5 major papers)

- High-efficiency engineering of cell membrane nanovesicles with intrinsic homotypic targeting for precision cancer drug delivery. Ho TL, Phan TTT, Tsai IL, Huynh TQD, Shen YA*, Fan YJ*. Int J Biol Macromol. 2025 Nov 8;334(Pt 1):148793.
- Fatty acid synthase inhibitor cerulenin hinders liver cancer stem cell properties through FASN/APP axis as novel therapeutic strategies. Chen LY, Wu DS, Shen YA*. J Lipid Res. 2024 Sep 26;65(11):100660.
- Glucose Transporter 1-Mediated Transcytosis of Glucosamine-Labeled Liposomal Ceramide Targets Hypoxia Niches and Cancer Stem Cells to Enhance Therapeutic Efficacy. Yu LY, Shueng PW, Chiu HC, Yen YW, Kuo TY, Li CR, Liu MW, Ho CH, Ho TH, Wang BW, Li CE, Chen MH, Shen YA*, Lo CL*. ACS Nano. 2023 Jul 25;17(14):13158-13175.
- Systems medicine dissection of chr1q-amp reveals a novel PBX1-FOXM1 axis for targeted therapy in multiple myeloma. Trasanidis N, Katsarou A, Ponnusamy K, Shen YA, Kostopoulos IV, Bergonia B, Keren K, Reema P, Xiao X, Szydlo RM, Sabbattini P, Roberts I, Auner HW, Naresh KN, Chaidos A, Wang TL, Magnani L, Caputo VS, Karadimitris A. Blood. 2022 Jan 11:blood.2021014391.

- Inhibition of the MYC-regulated glutaminase metabolic axis is an effective synthetic lethal approach for treating chemoresistant cancers. Shen YA, Hong JX, Asaka R, Asaka S, Hsu FC, Rahmanto YS, Jung JG, Chen YW, Yen TT, Tomaszewski A, Zhang C, Attarwala N, DeMarzo AM, Davidson B, Chuang CM, Chen X, Gaillard S, Le A, Shih IM, Wang TL. Cancer Res. 2020 Oct 15;80(20):4514-4526.

Lecture Title
Turning Cancer Stem Cell Vulnerabilities into Therapeutic Opportunities: From PBX1 Targeting to Trojan-Horse Nanomedicine

Abstract (500 words maximum)
Cancer stem cells (CSCs), although representing only a small fraction of tumor populations, play a central role in therapeutic resistance, tumor relapse, and metastatic progression. Their capacities for self-renewal, metabolic plasticity, and immune evasion enable them to survive conventional treatments and regenerate tumors, posing a major obstacle to durable cancer control. Targeting the unique biological dependencies of CSCs therefore represents a promising strategy for improving therapeutic outcomes. To address this challenge, we developed an integrated framework that exploits CSC vulnerabilities at both the transcriptional and drug-delivery levels. First, we identified pre-B-cell leukemia transcription factor 1 (PBX1) as a master regulator of CSC maintenance, chemoresistance, and metastatic potential. Using structure-guided drug discovery, we developed first-in-class small-molecule inhibitors that disrupt PBX1–DNA interactions and suppress CSC-associated transcriptional programs, thereby impairing self-renewal and increasing sensitivity to chemotherapy. Second, we engineered glucosamine-labeled liposomal nanocarriers that exploit the metabolic dependence of CSCs on glucose uptake. Incorporation of glucosamine–cholesterol into ceramide-based liposomes enables GLUT1-mediated transcytosis, facilitating penetration through stromal barriers and delivery of therapeutic agents into hypoxic tumor niches enriched with CSCs. This Trojan-horse delivery strategy suppresses stemness pathways, including OCT4–SOX2 signaling, modulates immune checkpoint expression, and enhances the efficacy of chemotherapeutic agents. Third, we developed hybrid nanovesicles (hNVs) generated by fusing natural killer (NK) cell membranes with CSC-derived membranes. These biomimetic vesicles combine homotypic tumor targeting with immune recruitment, enabling selective drug delivery to CSC populations while promoting NK cell–mediated antitumor responses. Collectively, these approaches establish a multi-layered therapeutic strategy that integrates transcriptional inhibition, metabolic targeting, and biomimetic nanomedicine to overcome CSC-driven resistance and advance the development of next-generation precision oncology.