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Education

1992	B.S., Department Of Agricultural Chemistry, National Taiwan University Taiwan
1994	M.S., Institute of Biological Chemistry, National Taiwan University, Taiwan
2002	Ph.D., Department Of Biochemistry and Molecular Biology, University Of Nebraska Medical Center, Omaha, NE

Research and Professional Positions Held in Chronological Sequence

2003	Post-Doctoral Research Associate, Department of Biochemistry and Molecular Biology, University of Nebraska Medical Center, Omaha, NE
2003-2006	Postdoctoral Fellow, Lombardi Comprehensive Cancer Center, Georgetown University, Washington, District of Columbia.

Research Interests

Our research focuses on three integrated objectives: (1) elucidating the molecular mechanisms underlying prostate cancer progression and metastasis; (2) identifying robust biomarkers to distinguish clinically significant (lethal) from indolent prostate cancer; and (3) defining the mechanisms of action of targeted therapies and their impact on tumor-immune interactions.

We investigate the role of cell surface proteolysis in cancer biology, particularly the Type II transmembrane serine proteases (TTSPs), which regulate key processes such as extracellular matrix remodeling, cell proliferation, migration, and apoptosis. Dysregulation of TTSPs contributes to tumor growth, invasion, and metastasis. Our work specifically examines TMPRSS2, matriptase, and matriptase-2, along with their cognate inhibitors HAI-1 and HAI-2, in prostate and lung cancer progression. In parallel, we study the pathological roles of HAI-1 and HAI-2 in biliary atresia and cholangiopathies, as well as the physiological function of the HAI-1-hepsin axis in pancreatic β-cell insulin production.

To address the clinical limitations of prostate-specific antigen (PSA)-based screening, including false positives, overdiagnosis, and overtreatment, we are developing noninvasive urinary biomarkers to improve early detection and risk stratification, enabling more precise treatment decision-making.

In addition, our recent findings reveal an unexpected immunomodulatory role for the EGFR tyrosine kinase inhibitor afatinib. Sequential administration of afatinib prior to anti-PD-1 therapy enhances antitumor efficacy, promotes $CD8^+$ T-cell infiltration, and prolongs survival in preclinical models. Building on this, we aim to define how afatinib functions not only as a tumor-intrinsic EGFR inhibitor but also as a transient immune-modulating agent that augments adaptive antitumor immunity.