2026 NYCU-UCSD Bilateral Symposium

Converging Biology, Engineering & Medicine for a Healthier Future

August 8 (Sat.) - 10 (Mon.)

HO YING TSAI Memorial Hall, Shou-Ren Building, Yangming Campus, NYCU

International Conference Hall, Xianqi Building, Boai Campus, NYCU

主辦單位 | 國立陽明交通大學 NATIONAL YANG MING CHIAO TUNG UNIVERSITY
腫瘤與免疫學研究中心 Cancer and Immunology Research Center
智慧型藥物與智能生物裝置研究中心 Center for Intelligent Drug Systems and Smart Bio-devices
工程醫學推動辦公室 Senior Vice President Office for Engineering Medicine

補助單位 | 教育部 高等教育深耕計畫 Higher Education SPROUT Project, Ministry of Education
NSTC 國家科學及技術委員會 National Science and Technology Council
教育部大專校院動物實驗替代科技人才培育計畫
NYCU 跨學院及產業3R跨域人才培育聯盟 NYCU 3R-TCA

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NYCU 跨學院及產業3R跨域人才培育聯盟 NYCU 3R-TCA

Agenda

August 8 (Sat.)

2026 NYCU-UCSD Bilateral Symposium

08:30-08:50	Registration	
08:50-09:00	Opening Remarks: Academician Shu Chien (Online)、Vice President Muh-Hwa Yang	
09:00-09:10	NYCU & UCSD MOU Signing Ceremony	
Moderators：Muh-Hwa Yang		Talk Title
09:10-09:50	Plenary Speech： Vice Chancellor John M. Carethers (UCSD)	
Session 1：Computational Medicine		
09:50-10:30	Hannah Carter (UCSD)	Host genetic factors modulate anti-tumor immunity and immunotherapy responses
10:30-10:50	Chun-Yu Lin (NYCU)	Patient-specific network-based interpretable machine learning predicts immune checkpoint inhibitor response in cancer patients
10:40-11:00	Coffee Break	
Session 2：Neuroscience		
Moderators：Shih-Pin Chen		Talk Title
11:10-11:30	Jin-Wu Tsai (NYCU)	From Cortical Development to Precision Neurotherapeutics
11:30-11:50	Shih-Pin Chen (NYCU)	The role of neuroinflammation in migraine
11:50-12:10	Cheng-Chang Lien (NYCU)	Predicting Threat in Time: A New Role for the Hippocampus
12:10-12:20	Group Photo	
12:20-13:30	Lunch	
Session 3：Cancer and Immunology		
Moderators：Muh-Hwa Yang		Talk Title
13:30-14:10	Barbara Jung (UCSD)	CDI2 Strategic Plan - Follow-Up Conversations
14:10-14:50	Li-Fan Lu (UCSD)	Functional specialization of regulatory T cells in controlling intestinal inflammation
14:50-15:10	Wei-Chien Yuan (NYCU)	Epigenetic Control of Hippo-YAP Signaling in Liver Regeneration and Cancer
15:10-15:30	Coffee Break	
Session 4：Chemical Biology		
Moderators：Jhih-Wei Chu		Talk Title
15:30-16:10	Terry Hwa (UCSD)	Resource Allocation in Starving Bacteria
16:10-16:30	Chin-Yuan Chang (NYCU)	Biosynthesis of Capreomycin and Enzyme-Catalyzed Pentacyclic Skeleton Formation in Cyclopiazonic Acid
16:30-16:50	Yu-Yuan Hsiao (NYCU)	A Nucleic Acid-Binding Intrinsically Disordered Loop Mediates Active-Site-Independent Inhibition of TREX1 through Fuzzy Interactions
16:50-	Closing Remarks: Vice President Muh-Hwa Yang	

Agenda

August 9 (Sun.) - 10 (Mon.)

2026 NYCU-UCSD Bilateral Symposium

08:30-08:50		Registration
08:50-09:00		Opening Remarks: Director Miwako Waga、 Vice President Hsiao-Wen Zan
Session 5：Tissue Engineering		
	Moderators： Jui-I Chao	Talk Title
09:00-09:40	Adam J. Engler (UCSD)	Understanding and Exploiting Cancer Mechanobiology
09:40-10:20	Robert L. Sah (UCSD)	Joints by Nature and by Design
10:20-10:40	Chih-Hsin Lin (NYCU)	A machine learning liver-on-a-chip system for safer drug formulation
10:40-11:00		Coffee Break
Session 6：Molecular and Biomedical Engineering		
	Moderators： Tzong-Yi Lee	Talk Title
11:00-11:40	Tzyy-Ping Jung (UCSD)	From Prognosis to Actuation: The Next Engineering Frontier for Brain-Computer Interfaces
11:40-12:00	Li-Wei Ko (NYCU)	AI-driven Wearable Brain Computer Interface for Smart Healthcare
12:00-13:30		Lunch
Session 7：Biosensing Technologies		
	Moderators： Dar-Bin Shieh	Talk Title
13:30-14:10	Albert P. Pisano (UCSD)	Wearable Sensors for Healthcare Engineering
14:10-14:30	Paul C.-P. Chao (NYCU)	Achieving High Accuracy in Predicting Blood Glucose Levels Using Dual Long Wavelengths 1480 nm and 1640 nm PPG and Machine Learning
14:30-14:50	Wen-Liang Chen (NYCU)	Comprehensive Electrochemical Platform for Disease Prevention and Detection
14:50-15:10		Coffee Break
Session 8：Systems & Synthetic Biology		
	Moderators： Ethan I. Lan	Talk Title
15:10-15:50	Yu-Hwa Lo (UCSD)	Label-Free, AI-Guided Cell Sorting for Single-Cell Multi-Omics and Cell Therapy
15:50-16:10	Dar-Bin Shieh (NYCU)	Nano-Enabled Biosensing Platforms: Bridging Rapid Virus Diagnostics and AI-Guided Multi-Cancer Detection
16:10-16:30	Ethan I. Lan (NYCU)	Development of a novel de novo biosynthetic pathway for 3-hydroxy-3 methylbutyrate production
16:30-		Closing Remarks: Senior Vice President Dar-Bin Shieh

Monday, August 10th, 2026
Delegation Visit from the University of California San Diego (UCSD), U.S.A

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2026 NYCU-UCSD Bilateral Symposium

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2026 NYCU-UCSD Bilateral Symposium

Hannah Carter

Position : Professor

Affiliation : Department of Medicine,Division of Genomics and Precision Medicine,University of California San Diego

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Biography

Dr. Carter is a Professor in the Department of Medicine and the Division of Genomics and Precision Medicine at UCSD. The Carter Lab develops and applies computational approaches to aid the interpretation of genetic variation and to advance precision cancer medicine. A major focus of the lab is developing methods for immuno-oncology. By studying the interplay between the inherited immune system and the evolving tumor genome, Dr. Carter seeks to identify the molecular pathways critical for immune response and evasion. She has developed computational approaches to prioritize neoantigens, model immunoediting and predict immunotherapy response. Dr. Carter is co-lead of the Structural and Functional Genomics Program at the Moores Cancer Center, co-Director of the Bioinformatics and Systems Biology Program and a member of the UCSD Institute for Genomic Medicine. She is an NIH Director’ s Early Independence Awardee, a Mark Foundation Emerging Leader and Jaime Wyatt Miller Fellow, a CIFAR Azrieli Global Scholar and a Siebel Scholar.

Host genetic factors modulate anti-tumor immunity and immunotherapy responses

Hannah Carter
Department of Medicine,Division of Genomics and Precision Medicine,University of California San Diego

Abstract

The success of immune checkpoint inhibitors (ICI) has demonstrated the potential for immunotherapy to revolutionize cancer treatment. However, while some patients have had remarkable response rates to ICI, response rates have been disappointingly low in many tumor types, and it has proved difficult to accurately predict which patients will benefit. To better understand the factors that lead to differences in ICI response across individuals, we studied genetic factors related to host-immunity and somatic characteristics of tumors in a dataset of > 700 cancer patients across 8 different ICI studies covering 3 different tumor types. We used interpretable machine learning approaches to train gradient boosting models to predict response from these germline and somatic features. Models trained to use both germline and somatic information from DNA outperformed models that used either data type alone. In the subset of samples that had RNA profiling, DNA scores predicted whether beneficial RNA measures, such as checkpoint expression or T cell infiltration levels, would be associated with response. Using Shapley Additive eXPlanations to interpret the learned model, the most predictive features related to T follicular helper cell infiltration, antigen presentation genes, folate metabolism, cell adhesion, loss of MHC-I, tumor subclonal heterogeneity and evidence of immunoediting. Examining feature interactions revealed that a SNP associated with T follicular helper cell infiltration could rescue response in the presence of class I HLA loss. Among ICI responders, tumors with a higher proportion of MHC II presented neoantigens obtained superior responses. Collectively, these findings reveal considerable influence of host genetics on potential for ICI response, and emphasize the importance of CD4+ T cell activity for effective anti-tumor immunity.

Chun-Yu Lin 林峻宇

Position : Associate Professor

Affiliation : Institute of Bioinformatics and Systems Biology, National Yang Ming Chiao Tung University

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Biography

Dr. Chun-Yu Lin is an Associate Professor at the Institute of Bioinformatics and Systems Biology, National Yang Ming Chiao Tung University (NYCU), Taiwan. After earning his Ph.D. from the former National Chiao Tung University in 2014, he expanded his international experience as a JSPS Postdoctoral Fellow at Kyoto University, Japan (2017–2019). His research focuses on integrating multiomics data with biological networks to develop next-generation personalized diagnostics. To achieve this, his team has developed innovative computational algorithms, including CaMPNets, SWEET, Gscore, and SINUM, that accurately decipher single-cell profiles and disease-driving pathways. These robust tools have been successfully translated into practical biomedical applications, such as cancer subtyping, clinical decision support, and drug repurposing. To date, he has published 37 SCI or conference papers, including in high-impact journals (e.g., Nature Communications, Nucleic Acids Research, Advanced Science, and Briefings in Bioinformatics). His outstanding contributions have been widely recognized with notable awards, including the JSPS 'Research Fellowship' (2017), the NCTU 'BioICT Junior Chair Professor' (2019), the MOST 'Young Scholar Fellowship' (2020), the NSTC 'Ta-You Wu Memorial Award' (2024), and the Pharmacogenomics Society of Taiwan 'Academician Yuan-Tsong Chen Young Scholar Award' (2024).

Patient-specific network-based interpretable machine learning predicts immune checkpoint inhibitor response in cancer patients

Chun-Yu Lin
Institute of Bioinformatics and Systems Biology, National Yang Ming Chiao Tung University

Abstract

Immune checkpoint inhibitors (ICIs) have transformed cancer therapy by producing durable responses in a subset of patients. However, response rates remain limited, and commonly used biomarkers, such as PD-1/PD-L1 expression and tumor microenvironment-related features, often show insufficient predictive accuracy and limited cross-cohort generalizability. Network-based biomarkers have been proposed for ICI response prediction, but many rely on aggregate protein-protein interaction networks compiled across diverse cell types and biological states or cohort-level co-expression networks. Such networks may obscure patient-specific variation in pathway activity and tumor-immune biology. Although single-sample network (SIN) inference provides a promising strategy to capture inter-patient heterogeneity, predictive frameworks that effectively leverage SINs for ICI response modeling remain limited.

In this talk, I will present SNIPE (Single-sample Network-based analysis for predicting ImmunotheraPy Efficacy), an interpretable machine-learning framework for predicting ICI response from patient-level transcriptomic data. SNIPE builds on our prior work in network medicine, particularly sample-specific weighted correlation networks (SWEET), which infer patient-specific SINs that capture shared and individualized molecular wiring more effectively than conventional cohort-level networks. SNIPE constructs high-confidence SINs (hcSINs) to represent individual tumor biology and integrates single-sample gene set correlation enrichment analysis (ssGscore), an extension of our Gscore method, to quantify personalized pathway activity from each patient’ s inferred network context. Together with ssGSEA, this strategy generates personalized ICI-response pathway signatures (PIPSs), which are used to train and compare multiple machine-learning models, including logistic regression, random forest, SVM, and XGBoost. SHapley Additive exPlanations (SHAP) are further applied to quantify pathway contributions and provide biologically interpretable explanations of model predictions.

Across five independent melanoma cohorts comprising more than 300 ICI-treated patients with matched transcriptomic and response data, SNIPE outperformed predictors based on aggregate PPI-network features and conventional ICI biomarkers. SHAP-based interpretation further highlighted clinically relevant inflammatory pathways contributing to SNIPE-based responder prediction, including rheumatoid arthritis- and inflammatory bowel disease-related pathways. Overall, SNIPE demonstrates how patient-specific network modeling and Gscore-derived pathway interpretation can be integrated into interpretable AI for precision immuno-oncology.

Jin-Wu Tsai 蔡金吾

Position : Secretary-General/Distinguished Professor

Affiliation : Institute of Brain Science,College of Medicine,National Yang Ming Chiao Tung University

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Biography

Jin-Wu Tsai, Ph.D., is Secretary-General of NYCU, Director of the Advanced Therapeutics Research Center, and Distinguished Professor at the Institute of Brain Science. He also serves as President of the Taiwan Neuroscience Society. Dr. Tsai received his Ph.D. from Columbia University and was previously a Scientific Manager at Genentech Inc.

Dr. Tsai’ s research focuses on the cellular and molecular mechanisms underlying cortical development and neurodevelopmental disorders. His work integrates human genetics, molecular biology, and advanced imaging approaches to uncover how disruptions in neuronal migration, cytoskeletal dynamics, and transcriptional regulation lead to brain malformations and neurological disease. His group has identified key disease-associated genes, including CEP85L, NDEL1, BICD2 and BAIAP2, and elucidated their roles in centrosomal function, cytoskeletal organization, and neuronal positioning. More recently, his research has expanded to transcriptional regulation, including the identification of FOXJ3 as a critical factor in neuronal differentiation and cortical development.

In addition to mechanistic discoveries, Dr. Tsai is actively translating basic science findings into clinical applications. His work on FOXG1 syndrome has led to the development of a diagnostic and predictive platform that integrates genetic and phenotypic data for improved disease classification and risk assessment. His broader research vision aims to establish a multiscale framework linking genes, cells, and cortical layering to enable precision diagnostics and targeted therapeutic strategies for neurodevelopmental and neurodegenerative disorders.

Dr. Tsai has published extensively in leading journals, including Neuron, Nature Genetics, Nature Communications, Cell Death and Differentiation, Molecular Psychiatry, Acta Neuropathologica, Developmental Cell, and Journal of Biomedical Science. He has received Outstanding Research Award from the National Science Council (2020, 2026), TienTe Lee Award for Medical and Pharmacological Technologies (Young Investigators), and Academia Sinica Research Award for Junior Research Investigators (2019).

From Cortical Development to Precision Neurotherapeutics

Jin-Wu Tsai
Institute of Brain Science,College of Medicine,National Yang Ming Chiao Tung University

Abstract

Neurodevelopmental and neuropsychiatric disorders remain among the most complex and least predictable human diseases, largely due to the multiscale nature of brain development spanning genes, cells, circuits, and behavior. In this talk, I will present our integrative efforts to bridge fundamental mechanisms of cortical development with emerging strategies for precision neurotherapeutics. Our work begins with human genetics-driven discovery of key regulators of cortical development. We identified CEP85L as a critical centrosomal protein governing neuronal migration, where disease-associated mutations disrupt microtubule organization and lead to malformations of cortical development. Beyond cytoskeletal regulation, we have uncovered transcriptional programs that shape neuronal fate and cortical architecture. Our recent studies identify FOXJ3 as a novel transcriptional regulator that coordinates gene expression networks essential for progenitor differentiation and neuronal maturation. These findings highlight how transcriptional control integrates with cellular machinery to orchestrate cortical development. Importantly, we have translated these mechanistic insights into clinically relevant applications. Focusing on FOXG1 syndrome, a severe neurodevelopmental disorder, we developed a diagnostic and predictive platform that integrates genetic, molecular, and phenotypic data to achieve high accuracy in risk stratification. This work establishes a foundation for early diagnosis and targeted intervention. Ultimately, this framework provides a path toward precision neurotherapeutics, where mechanistic understanding informs early diagnosis, patient stratification, and the development of targeted interventions for neurodevelopmental diseases.

Shih-Pin Chen 陳世彬

Position : 1. Professor & Director/ 2. Attending Physician & Chief

Affiliation : 1. Institute of Clinical Medicine, National Yang Ming Chiao Tung University; 2. Division of Translational Research, Department of Medical Research, Taipei Veterans General Hospital

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Biography

Dr. Chen obtained his MD degree from the School of Medicine and his PhD degree from the Institute of Clinical Medicine at National Yang-Ming University, Taiwan. He completed his neurology residency training at the Department of Neurology, Taipei Veterans General Hospital, followed by postdoctoral fellowship training at Massachusetts General Hospital, Harvard Medical School. He has served as an attending physician in the Department of Medical Research and as an adjunct attending neurologist at the Neurological Institute of Taipei Veterans General Hospital.

Dr. Chen is dedicated to unraveling the pathophysiology of headache and neurovascular disorders using bidirectional translational approach encompassing clinical, neuroimaging, genetics, and preclinical animal studies. He has authored over 180 research articles, including those from esteemed journals such as JAMA Neurology, Brain, Annals of Neurology, and Neurology, etc. He is currently the Secretary General of the Asian Regional Headache Consortium (ARCH), an Associate Editor of The Journal of Headache and Pain, a board member of the Science and Research Committee in the International Headache Society, and the Convener of the Neurology Division, Department of Life Sciences, National Science and Technology Council, Taiwan.

The role of neuroinflammation in migraine

Shih-Pin Chen

1. Institute of Clinical Medicine, National Yang Ming Chiao Tung University; 2. Division of Translational Research, Department of Medical Research, Taipei Veterans General Hospital

Abstract

Migraine is a complex neurological disorder with substantial socioeconomic impact, yet it remains under-recognized and under-treated. Despite advances in the field, its pathophysiology is still incompletely understood. Increasing evidence implicates neuroinflammation as a key contributor to migraine; however, direct histopathological evidence in humans is lacking because brain tissue is inaccessible for biopsy. Recent advances in neuroimaging have begun to provide indirect in vivo evidence of neuroinflammation within the central nervous system and adjacent tissues of patients with migraine, findings that are strongly supported by preclinical studies.

Cortical spreading depolarization (CSD), the electrophysiological substrate of migraine aura, is the only established intrinsic brain event capable of triggering trigeminovascular activation. Neurogenic inflammation within the meninges is thought to mediate the sensitization of trigeminal nociceptors and subsequent headache. Our recent work demonstrates that activation of the neuronal P2X7–Panx1–NLRP3 inflammasome pathway is a critical driver of CSD-induced neuroinflammatory cascades and trigeminovascular activation. We further show that CSD elicits an initial surge of perivascular cerebrospinal fluid influx accompanied by rapid trigeminal activation, followed by prolonged dysfunction of the glymphatic and meningeal lymphatic systems. Impaired brain waste clearance likely delays the removal of inflammatory mediators, thereby sustaining neuroinflammation and pain. Together, these findings identify a mechanistic link between cortical neuroinflammation, dysfunctional glymphatic–meningeal lymphatic transport, and trigeminovascular activation in migraine. Ongoing studies will need to define the underlying molecular and neural circuit mechanisms, with the goal of identifying novel therapeutic targets for migraine.

Cheng-Chang Lien 連正章

Position : Chair Professor & Dean

Affiliation : Institute of Neuroscience, College of Life Sciences,National Yang Ming Chiao Tung University

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Biography

Dr. Cheng-Chang Lien is Chair Professor and Dean of the College of Life Sciences at National Yang Ming Chiao Tung University (NYCU), Taiwan. He received his M.D. degree from China Medical University in 1997 and earned his Ph.D. in Physiology and Neuroscience from the University of Freiburg, Germany, under the mentorship of Professor Peter Jonas, graduating summa cum laude in 2003. He subsequently conducted postdoctoral research with Professor Mu-Ming Poo at the University of California, Berkeley, USA. He joined the Institute of Neuroscience at NYCU in 2006, was promoted to Professor in 2015, and served as Institute Director from 2017 to 2021.

Dr. Lien’ s research focuses on the physiological mechanisms underlying neural circuit function, excitation-inhibition balance, cognition, emotion, and neurological and psychiatric disorders. His laboratory integrates electrophysiology, optogenetics, chemogenetics, calcium imaging, mouse genetics, behavioral analysis, and computational approaches to investigate neural function from molecules to behavior.

His work has advanced the understanding of hippocampal circuit organization, particularly dentate gyrus mossy cells and GABAergic interneurons. Through optogenetic-assisted circuit mapping and in vivo functional studies, he has identified long-range excitatory and inhibitory mechanisms that regulate information processing, anxiety-related behaviors, and memory formation. He also discovered inhibitory fear memory engrams in the amygdala, providing a new framework for understanding emotional memory storage and stabilization. His research further elucidates how circuit remodeling and altered inhibitory signaling contribute to chronic pain, Huntington’ s disease, and affective disorders.

Dr. Lien has received numerous honors, including election as a Fellow of the IUPS Academy of Physiology (2026), the 4th Chiung-Lin Chen Memorial Award for Translational Medicine (2025), the NSTC Outstanding Research Award (twice; 2016, 2024), the TienTe Lee Young Scientist Award (2015), and the NHRI Integrated Research Grant Award (twice). International recognitions also include the DAAD Scholarship, the Alexander von Humboldt Research Fellowship, and the NeuroCure Fellowship. He currently serves as Chair of the Alexander von Humboldt Fellow Alumni Association in Taiwan and has held leadership positions including Convener of the NSTC Division of Morphology and Physiology (2023–2025) and Director of the NSTC Life Sciences Research Promotion Center (2024–2026).

Predicting Threat in Time: A New Role for the Hippocampus

Cheng-Chang Lien
Institute of Neuroscience, College of Life Sciences,National Yang Ming Chiao Tung University

Abstract

Adaptive defensive behavior depends not only on learning that a stimulus predicts danger, but also on anticipating when an aversive outcome is expected to occur. Although the hippocampus is well known for its role in associative and contextual learning, whether and how hippocampal circuits encode internal predictions of threat timing has remained unclear. Here, we identify a previously unrecognized predictive timing mechanism mediated by mossy cells in the ventral dentate gyrus during conditioned fear learning. Using in vivo recordings combined with circuit-specific manipulations, we show that ventral mossy cells respond robustly to both conditioned and unconditioned stimuli during fear conditioning. Following learning, these cells exhibit a reproducible neural response during memory retrieval that is precisely time-locked to the expected onset of the unconditioned stimulus, even in its absence. This internally generated activity pattern, which we term a conditioned US-timing signal, reflects a learned prediction of threat timing rather than a sensory-driven response. Disruption of mossy cell activity or selective attenuation of this timing signal during retrieval significantly reduces freezing behavior and promotes movement, demonstrating a causal role in fear expression. These findings reveal that ventral dentate gyrus mossy cells encode temporally precise threat predictions and provide a novel hippocampal mechanism linking associative memory to behavioral control. By furnishing a temporal scaffold for information routing to downstream affective circuits, this mechanism offers new insight into how internal threat predictions guide defensive behavior and how their dysregulation may contribute to pathological fear and anxiety.

Barbara Jung, M.D.

Position : Associate Vice Chancellor and Dean

Affiliation : Department of Medicine, Division of Gastroenterology,University of California San Diego

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somdeanassistant@health.ucsd.edu

Biography

Barbara Jung, M.D. is the associate vice chancellor and dean for UC San Diego School of Medicine.

She is a practicing gastroenterologist and physician-scientist, with a focus on developing therapies to target stromal immune regulation in GI disease. She is an academician at heart and passionate about leading with empathy and through empowerment of others and training the next generation of diverse clinicians, educators, scientist and leaders.

Prior to joining UC San Diego in January 2025, Dr. Jung served as professor and chair of the Department of Medicine and held the Robert G. Petersdorf Endowed Chair in Medicine at the University of Washington.

Dr. Jung completed her medical degree at the Ludwig-Maximilians University in Munich, Germany. She then moved to the United States and completed postdoctoral studies in colon cancer on the mesa in San Diego, followed by internal medicine residency and gastroenterology fellowship at UC San Diego.

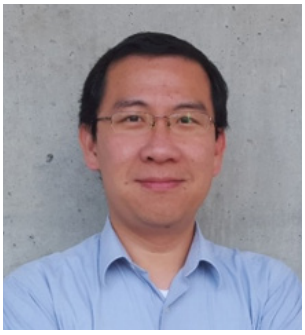
Her continuously federally funded laboratory centers on activin, a TGFb family member signaling in GI disease, which is an extension from her studies that originated from her time as a fellow in the Division of Gastroenterology & Hepatology at UC San Diego.

CDI2 Strategic Plan - Follow-Up Conversations

[1]Mark B. Wiley Ph.D., [1]Jessica Bauer Ph.D., [1]Yuchen Wang, [1]Barbara Jung M.D.
[1]Department of Medicine, Division of Gastroenterology, University of California, San Diego, San Diego, California

Abstract

The five-year survival rate for pancreatic cancer patients remains at 13%. More than 90% of these patients are diagnosed with pancreatic ductal adenocarcinoma (PDAC). Acute pancreatitis and chronic pancreatitis are risk factors for developing PDAC suggesting a strong inflammatory role for disease initiation. Recently, we found that activin a (activin), a TGF β superfamily member, drives inflammation in acute pancreatitis via recruitment of macrophages and activation of neutrophils. In keeping with this, we recently showed that PDAC patients with high activin expression in the stroma have a worse prognosis and that inhibition of activin in mice decreased metastasis suggesting PDAC patients might benefit from activin inhibition. Here, we expand our studies in PDAC in the setting of inflammation and activin signaling. Bulk RNA-sequencing analysis was performed on open source data available through the Cancer Genome Atlas and the Genotype Tissue Expression portal to compare the expression of activin signaling components across normal and cancerous pancreatic tissue. Digital Spatial Profiling (DSP) was performed on a tissue microarray of PDAC patients which permitted visualization and separation of images based on PanCK and activin localization. Tumor and stroma compartments were separated via PanCK expression and the quantification of 56 proteins was performed in activin (+) and (-) areas within each compartment. Western blots were performed to quantify pSMAD2/3, pERK, and PI3K on pancreatic cancer cells stimulated with activin in the presence/absence of anti-activin neutralizing antibody or a highly specific activin receptor subtype-2A (ACVR2A) inhibitor. Preliminary RNA-sequencing analysis identified that the genes encoding activin A and ACVR2A are significantly upregulated in PDAC tissue when compared to healthy donor tissue. DSP data revealed activin (+) areas in the tumoral compartment had reduced total immune, total T cell, T helper, and memory T cell infiltrations when compared to activin (-) areas. This effect was coupled to increased markers of the MAPK and PI3K pathways in activin (+) areas. In vitro, activin stimulated phosphorylation of p-38, p-90, and SMAD2/3 in primary pancreatic cancer cells, but not in metastatic pancreatic cancer cells suggesting that activin may mediate stage-specific outcomes. Our data also suggests that ACVR2A regulates SMAD2/3 phosphorylation in pancreatic cancer cells. Taken together, these data suggest that activin is a targetable molecule promoting a cancer supportive microenvironment in PDAC.



Li-Fan Lu

Position : Professor and Co-Director

Affiliation : Program in Immunology, Department of Molecular Biology,University of California San Diego

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Biography

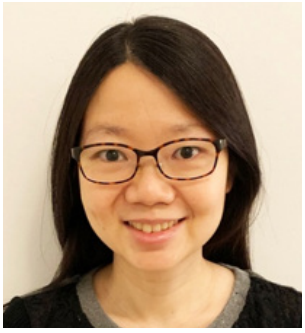
Dr. Lu is a Professor in the Department of Molecular Biology and Co-Director of the Program in Immunology at the University of California, San Diego. He earned his Ph.D. in Immunology from Dartmouth College and completed postdoctoral training at the University of Washington and Memorial Sloan-Kettering Cancer Center. Dr. Lu’ s research focuses on the role of regulatory T (Treg) cells in maintaining immunological tolerance to self-antigens and in modulating immune responses against pathogens and tumors. He has authored numerous publications on Treg cell biology in premier journals and is the holder of several patents.

Functional specialization of regulatory T cells in controlling intestinal inflammation

Li-Fan Lu
Program in Immunology, Department of Molecular Biology,University of California San Diego

Abstract

In the last three decades, regulatory T (Treg) cells have been extensively studied for their role in maintaining immunological tolerance, revealing significant heterogeneity that allows them to regulate various immune responses. However, the specific mechanisms by which different Treg cell populations exert their effects remain poorly understood. Here, we examined various tissue-specific Treg cell subsets during active autoimmune inflammation. Specifically, we uncovered key molecular pathways that govern Treg cell-mediated immune regulation, particularly within the intestinal tissues. Collectively, our work establishes a powerful model to explore the effector mechanisms of Treg cells and supports the development of strategies to manipulate their function, which could ultimately lead to novel therapies for a wide range of human diseases.



Wei-Chien Yuan 袁維謙

Position : Assistant Professor

Affiliation : Department of Life Sciences and Institute of Genome Sciences,National Yang Ming Chiao Tung University

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Biography

Dr. Wei-Chien Yuan is an Assistant Professor in the Department of Life Sciences and the Institute of Genome Sciences at National Yang Ming Chiao Tung University, Taiwan. She received her B.S., M.S., and Ph.D. from National Taiwan University and completed her postdoctoral training with Dr. Fernando D. Camargo at Boston Children's Hospital, Harvard Medical School, where she studied Hippo-YAP signaling in tissue regeneration and cancer.

Dr. Yuan's research focuses on the epigenetic regulation of cellular plasticity, tissue regeneration, and cancer progression. Her laboratory combines functional genomics, in vivo CRISPR screening, single-cell epigenomics, organoid models, and genetically engineered mouse models to uncover how chromatin regulators control cell fate decisions and tumor development. Her recent work has identified novel epigenetic mechanisms that regulate YAP-dependent transcription during liver regeneration and liver cancer, providing new insights into regenerative biology, tumor immunity, and therapeutic targeting.

Dr. Yuan has published in leading journals, including Cell, Cancer Cell, Cell Stem Cell, Molecular Cell, Nature Communications, and The EMBO Journal. She has received several prestigious awards, including the Outstanding Women in Science Award- Emerging Young Scientist, the National Health Research Institutes Career Development Grant, the Taiwan Ministry of Science and Technology Emerging Young Scholars Award, and the Ministry of Education Yushan Young Fellow Award.

Epigenetic Control of Hippo-YAP Signaling in Liver Regeneration and Cancer

Pei-Chi Chang[1, 2, 3,] Yen-Ting Chu[1], Liang-Hsun Shen[1], Yu-Yun Weng[1], Szu-Shuo Lee[3], Yu-Ching Wang[3,4,5]Andrew S. Earl,[6] Sai Ma,[6, 7] Karel Alcedo,[6] Jacquelyn O. Russell,[6,8] Yu-Ru Lee,[3] Jason D. Buenrostro, [6] and Fernando D. Camargo, [6, 8] Wei-Chien Yuan [1, *]

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Abstract

The Hippo-YAP pathway is a master regulator of tissue regeneration, cellular plasticity, and tumor progression, yet how epigenetic mechanisms shape YAP function remains poorly understood. Here, we demonstrate that distinct chromatin regulators direct YAP-dependent transcriptional programs to either constrain regenerative plasticity or promote malignant progression in the liver. Using single-cell chromatin profiling and in vivo CRISPR screening, we identify the histone acetyltransferase HBO1 as an epigenetic brake that restricts YAP-driven hepatocyte-to-biliary epithelial cell reprogramming during liver injury. Loss of HBO1 enhances chromatin remodeling and YAP activity, thereby accelerating hepatocyte plasticity and liver regeneration. In contrast, we identify the histone methyltransferase SETD8 as an essential epigenetic effector that enables YAP-driven oncogenesis by establishing a permissive chromatin landscape for sustained transcriptional activation. Beyond promoting tumor growth, SETD8 also orchestrates an immunosuppressive tumor microenvironment, whereas its pharmacological inhibition suppresses tumor progression, restores anti-tumor immunity, and sensitizes tumors to immune checkpoint blockade while sparing normal liver tissue. Together, these findings reveal that the biological outputs of YAP signaling are governed by epigenetic regulators that either restrain regenerative cell fate plasticity or reinforce oncogenic and immune-evasive programs. Our work provides a unifying epigenetic framework for understanding YAP function and highlights new therapeutic opportunities for regenerative medicine and YAP-driven cancers.

Terry Hwa

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Biography

Terry Hwa is the Presidential Chair and Distinguished Professor of Physics and Molecular Biology at UC San Diego, and the founding director of its Quantitative Biology Graduate Program. Hwa completed his B.S. degree in Physics, Biology, and Electrical Engineering from Stanford University, and Ph.D. in Statistical Physics at MIT. He has been at UC San Diego since 1995, with a mid-career transition to microbiology 20 years ago. Hwa is a member of the National Academy of Sciences and a recipient of the Max Delbruck Prize of the American Physical Society. Hwa develops theoretical and experimental approaches to gain quantitative, predictive understanding of living systems, focusing on bacterial physiology. The Hwa lab is continuing to extend their physiology-based approaches to characterize bacterial species singly and in consortium, to uncover underlying principles governing the spatiotemporal dynamics of microbial communities, and to apply these principles to expand synthetic biology applications.

Resource Allocation in Starving Bacteria

Terry Hwa
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Abstract

Synthesis of stress proteins is crucial for the survival of starving bacteria. Quantitative proteomic analysis revealed a starvation-induced protein degradation (SPD) program, widely conserved in bacteria and analogous to autophagy in eukaryotes, to be the primary process fueling protein synthesis and energy biogenesis during starvation. SPD is surprisingly inefficient, non-specifically targeting a vast number of cytoplasmic proteins including those newly synthesized during starvation. Yet, SPD substantially prolongs cell survival, accounting for a major share of viability maintenance. We characterize factors turning on and off SPD during starvation, and describe the resulting tradeoff between prolonging survival and maintaining metabolic activities during starvation. The latter leads to a simple approach to enhancing bio-production by non-growing cells as microbial cell factories.

Chin-Yuan Chang 張晉源

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Biography

Chin-Yuan Chang received his B.S. degree (2004) in Chemistry at Kaohsiung Medical University and his Ph.D. degree (2011) in Biological Science and Technology at National Chiao Tung University. He then joined Prof. Tsung-Lin Li’ s lab in Genomics Research Center, Academia Sinica, as a postdoctoral fellow. In 2015, he moved to Prof. Ben Shen’ s lab as an Academia Sinica Fellow at Scripps Research Institute in Jupiter, FL. After three years in Scripps, he joined Department of Biological Science and Technology at National Yang Ming Chiao Tung University. Currently, his lab employs biochemical and structural biology approaches to investigate natural product biosynthesis, including unique NRPS systems and the biosynthesis of nonproteinogenic amino acids, resistance mechanisms, and new chemistries within enzyme systems

Biosynthesis of Capreomycin and Enzyme-Catalyzed Pentacyclic Skeleton Formation in Cyclopiazonic Acid

Chin-Yuan Chang
Department of Biological Science and Technology,National Yang Ming Chiao Tung University

Abstract

We employ biochemical and structural biology approaches to investigate the biosynthetic pathways of natural products and the catalytic mechanism of the enzymes involved. Our research focuses on the following two topics:

1. Capreomycin (CMN) is a nonribosomal peptide (NRP) antituberculosis antibiotic, the structure of which is composed of four nonproteinogenic amino acids, including L-2,3-diaminopropionic acid (L-Dap), β -ureidodehydroalanine, L-capreomycidine (L-Cap), and β -lysine. These nonproteinogenic amino acids play critical roles for the mechanisms of action and resistance of CMN. We combine biochemical characterization and structural determination to elucidate the biosynthesis of these nonproteinogenic amino acids, as well as the operation of an unusual NRPS machinery. Our studies modify the originally proposed biosynthetic pathway and provide insights into nonproteinogenic amino acids formation in CMN and other related natural products.

2. We investigate enzyme-catalyzed cyclization reactions in cyclopiazonic acid (CPA) biosynthesis. This work resolves a long-standing biosynthetic mystery and expands the known catalytic repertoire of flavoenzymes. Moreover, the identified cyclization enzyme can be repurposed as a biocatalyst for the chemoenzymatic synthesis of structurally diverse indole alkaloid derivatives, highlighting its potential utility in molecular diversification.

Yu-Yuan Hsiao 蕭育源

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Biography

Professor Yu-Yuan Hsiao received his B.S. degree in Chemistry before moving into the field of Biochemistry. During his Ph.D. training, he developed a strong foundation in structural biology, with particular expertise in exonucleases. In 2013, he established his independent laboratory, which focuses on the structural and mechanistic studies of disease-related nucleic acid-binding proteins (NBPs) and on the development of structure-based inhibitors targeting these proteins.

In recent years, Professor Hsiao has expanded his research interests to NBPs containing intrinsically disordered regions (IDRs). His work seeks to understand how these proteins interact with nucleic acids, how IDRs contribute to protein function and regulation, and whether IDR-containing regions can serve as druggable targets for therapeutic inhibition.

A key strength of Professor Hsiao’s laboratory is its integrated application of multiple structural biology approaches, including cryo-EM, X-ray crystallography, NMR, and SAXS, together with a broad range of biochemical and biophysical techniques such as activity assays, Electrophoretic Mobility Shift Assay (EMSA), Intrinsic Tryptophan Fluorescence (ITF), Microscale Thermophoresis (MST), and fluorescence anisotropy. This multidisciplinary strategy enables a comprehensive understanding of protein structure, dynamics, and function, and is also applied to elucidate the mechanisms by which inhibitors and other compounds modulate protein activity. To further investigate protein dynamics, his laboratory has also incorporated all-atom molecular dynamics simulations and single-molecule Förster resonance energy transfer (smFRET).

Professor Hsiao’s research has made significant contributions to the understanding of NBPs and their mechanisms of action. His work has been published in leading journals, including Nature Chemical Biology, Nature Communications, Nucleic Acids Research, PLoS Biology, and JACS Au. Through these efforts, his laboratory has become a recognized and pioneering research group in the fields of biochemical and structural biology of NBPs.

A Nucleic Acid-Binding Intrinsically Disordered Loop Mediates Active-Site-Independent Inhibition of TREX1 through Fuzzy Interactions

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Abstract

TREX1 is a central exonuclease that functions in cytosolic immune regulation and nuclear DNA repair, making it an attractive therapeutic target with broad biomedical applications, including cancer immunotherapy, anticancer and antiviral intervention, and enhancement of genome-editing efficiency such as CRISPR-based approaches. In our studies, we identified three major classes of TREX1 inhibitors that target distinct regulatory regions of the enzyme, including the catalytic active site, an allosteric site, and an intrinsically disordered region (IDR)-defined modulation site. Here, we focus on the IDR-targeting inhibitors.

At the substrate-binding interface adjacent to the DEDDh catalytic core, TREX1 contains an intrinsically disordered α 7–8 loop. We identified a series of small-molecule inhibitors that bind to this IDR-defined modulation site and induce distinct disorder-to-order transitions. High-resolution X-ray crystallography reveals a conserved triangular clamping mode through which these inhibitors stabilize the flexible loop into defined conformations. Structural analysis combined with molecular dynamics simulations further shows that these conformational transitions arise from fuzzy interaction patterns between the IDR and the inhibitors in aqueous solution. Biochemical assays, together with in vitro and in vivo functional studies, demonstrate that stabilization of this loop disrupts DNA binding, interferes with substrate recognition, and thereby suppresses TREX1 activity.

These findings establish an active-site-independent mechanism for TREX1 inhibition through targeting an IDR-mediated conformational switch. Given the prevalence of IDRs in the mammalian proteome and their potential as highly specific drug targets, our study provides rare atomic-level evidence of inhibitor-induced structural ordering within an IDR and offers mechanistic insight into the druggability of IDRs in nucleic acid-binding proteins.

Adam J. Engler

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Biography

Adam J. Engler is a Professor and Chair of the Shu Chien-Gene Lay Department of Bioengineering at UC San Diego, where he has been on the faculty since 2008. Dr. Engler also holds the Kenneth Bowles Endowed Chair and is a resident scientist at the Sanford Consortium for Regenerative Medicine. Prior to starting his independent career, Dr. Engler was awarded his PhD from the University of Pennsylvania and performed postdoctoral training at Princeton University.

Dr. Engler has published more than 130 peer-reviewed manuscripts, and his seminal work has shown how physical and chemical properties of the extracellular matrix influence or misregulate cell function and modify genetic mechanisms of disease. His lab currently studies this phenomenon in the context of cardiovascular diseases and cancer.

Dr. Engler has received numerous awards in recognition of this research, including young investigator or mid-career awards from International Society for Matrix Biology (2008), Biomedical Engineering Society (2008 and 2023), American Society of Matrix Biology (2014), American Society of Mechanical Engineering (2015), and American Society for Engineering Education (2018). Dr. Engler is a NIH New Innovator Award grantee (2009) and is a Fellow of the American Institute for Medical and Biomedical Engineering (2018), the Biomedical Engineering Society (2021), the International Academy of Medical and Biological Engineering (2024), and American Society of Mechanical Engineers (2025).

Understanding and Exploiting Cancer Mechanobiology

Adam J Engler
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Abstract

Epithelial cells are classically known to respond to differences in extracellular matrix (ECM) stiffness by transitioning to a malignant, non-polarized state on stiffer ECM, i.e. Epithelial-Mesenchymal Transition (EMT). While this is akin to stiff tumors that one can detect with manual palpation, cancer fibrosis is dynamic and stiffening occurs over months to years. I will describe our efforts to mimic the onset of tumor-associated fibrosis using dynamic methacrylated-hyaluronic acid (MeHA) hydrogels. Contrary to previous observations, we find that collective decisions by mammary epithelial cells in 3D aggregates–called acini–indicate partial protection from the stiffened niche via molecular mechanisms that interpret stiffness. After cells leave this niche, however, mechanical changes can be remembered. In a parallel story, I will share our results on oral cancer, where cells that disseminate from the niche exhibit "mechanical memory" of their surroundings, and that memory can be used to "educate" their neighboring epithelial cells. I will conclude my presentation with the discovery of a physical marker–adhesion strength–that predicts both metastatic potential and survival rates. Cells disseminating from mammary tumors are weakly adherent, and when presorted by adhesion, primary tumors created from strongly adherent cells exhibit fewer lung metastases than weakly adherent cells or unsorted populations. Admixed cancer lines can be separated by label-free adhesive signatures using a next-generation flow chamber. When applied to metastatic tumors, the chamber retrospectively predicted metastatic disease from stromal samples with 100% specificity, 85% sensitivity, and AUC of 0.94. The chamber can also differentiate more weakly adherent human metastatic mammary tumors, e.g., invasive ductal carcinoma (IDC) or invasive lobular carcinoma (ILC), from non-invasive tumors, e.g., ductal carcinoma in situ (DCIS). These results together suggest how, unlike other senescent cells, metastatic cancer cells use weak adhesion to navigate against stiffness gradients and how we can use this to our advantage to assess metastatic potential of patient tumors.

Robert L. Sah

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Biography

Robert Sah is Professor of the Shu Chien–Gene Lay Department of Bioengineering and co-director of the Center for Musculoskeletal Research at UC San Diego. He received the B.S. and M.S. in Electrical Engineering and the Sc.D. in Medical Engineering from M.I.T.; and the M.D. from Harvard. After post-doctoral research at Massachusetts General Hospital; he joined UC San Diego Bioengineering in 1992. Dr. Sah’ s research interests are the biomechanics; mechanobiology; and tissue engineering of cartilage; synovial fluid; and synovial joints with the ultimate goal of improving the treatment; diagnosis; and prevention of diseases of skeletal growth; injury; and degeneration; including osteoarthritis.

He is a member of the Society of Professors of the Howard Hughes Medical Institute (HHMI), a recipient of the Van C. Mow medal, a fellow of the American Institute for Medical and Biological Engineering (AIMBE), the Biomedical Engineering Society (BMES), and the Orthopaedic Research Society. He received the Kappa Delta Award three times from the American Academy of Orthopaedic Surgery / Orthopaedic Research Society, the Arthritis Foundation Hulda Irene Duggan Investigator Award, and an NSF Young Investigator Award. He is an active member of the Orthopaedic Research Society, Tissue Engineering and Regenerative Medicine International Society, and International Cartilage Repair Society.

He was formerly Vice Chair of the Department of Bioengineering at UC San Diego, Chair of the Undergraduate Studies Committee, and Advisor to the Undergraduate Student Chapter of the BioMedical Engineering Society. H was also director of an NIH Training Grant on Translational Musculoskeletal Research. He currently directs the Bioengineering Research Scholars outreach program for High School Students.

Joints by Nature and by Design

Robert L Sah, MD, ScD, Van Wong, BS, and Albert Chen, PhD
Shu Chien-Gene Lay Department of Bioengineering, University of California-San Diego

Abstract

Articular cartilage normally serves as a load-bearing, low-friction, and wear-resistant material that effectively covers and protects the ends of bones over a lifetime. My lab studies the biomechanics and mechanobiology of this remarkable tissue, as it grows to a stable adult form, adapts to imposed mechanical demands, and when it fails in osteoarthritis.

The macroscopic load-bearing properties of cartilage depend on the molecular balance between the swelling properties of polyanionic glycosaminoglycans and the constraint of the dense collagen network. Growth of cartilage and the skeleton is driven in part by swelling stress that exceeds collagenous restraint. Homeostasis of healthy adult articular cartilage depends on a metabolic balance and maintenance of the depth-variation from the articular surface to the subchondral bone. Deterioration of articular cartilage biomechanical function with aging and osteoarthritis occurs at the surface and superficial zone in early stages, and then in the middle-deep zones at later stages.

The tribology of synovial joints involves the relative motion of apposing cartilage surfaces, sliding past each other in synovial fluid. The low-friction and low-wear properties of articular cartilage are due in part to lubricating molecules in synovial fluid, particularly proteoglycan 4 and hyaluronan. The shear to which the articular cartilage and indwelling chondrocytes are subjected is governed by the friction-dependent shear stress within, and the shear modulus of, cartilage in the superficial zone. Chondrocytes respond to shear by increasing production of proteoglycan-4. The lubricating properties of synovial fluid are diminished after acute injury due to diminished concentration and altered structure of hyaluronan. Such alterations may disrupt the mechanobiology of the synovial joint.

The bioengineering of articular cartilage and synovial fluid may be useful for scientific investigations and future therapies. Biotechnologies targeting cell expansion and assembly as well as bioreactor culture can facilitate formation of a cartilage-like material and synovial fluid-like liquid lubricant. The quest to attain material and solution phenotypes present in synovial joint health and disease challenges our understanding at multiple scales. Achieving fabrication of osteochondral structures in joint-scale bioreactors may lead to future-generation therapies such as biological joint replacement.

Chih-Hsin (Melody) Lin 林芷歆

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Biography

Melody received her B.S. in Life Science from National University of Kaohsiung in 2007, her M.S. in Zoology from National Taiwan University in 2010, and her Ph.D. in Dentistry from National Yang Ming Chiao Tung University in 2016 under the supervision of Prof. Shyh-Yuan Lee and Prof. Yuan-Min Lin. During her doctoral training, she was a visiting student at Harvard School of Dental Medicine (2013–2014), where she conducted developmental biology research under Prof. Bjorn Olsen. She completed her postdoctoral training from 2017 to 2019 in Prof. Robert S. Langer's laboratory at the Massachusetts Institute of Technology (MIT), where she developed biomaterials and tissue engineering technologies for drug delivery and regenerative medicine. She subsequently joined the Center for Tissue Engineering at Chang Gung Memorial Hospital as a postdoctoral fellow under Prof. Ming-Huei Cheng (2019–2020). Melody began her independent academic career as an Assistant Professor at Taipei Medical University in 2020 and was promoted to Associate Professor in 2024. During 2022–2023, she was also a Visiting Professor in the Department of Chemistry at the University of Oxford, conducting research with Prof. Hagan Bayley. She is currently an Assistant Professor at the Institute of Molecular Medicine and Bioengineering, National Yang Ming Chiao Tung University, where her laboratory develops biomaterials, vascularized tissue models, and advanced biofabrication technologies for tissue engineering, regenerative medicine, and drug screening, with the goal of translating bioengineered human tissue models into clinically relevant therapeutic platforms.

A machine learning liver-on-a-chip system for safer drug formulation

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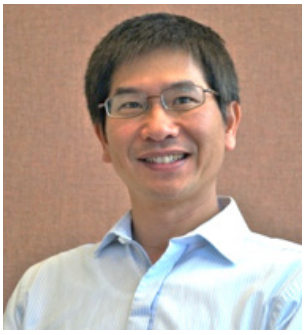
Abstract

Drug metabolism governs the biotransformation of pharmaceutical compounds, influencing their efficacy, toxicity, and potential drug–drug interactions. However, existing preclinical models often fail to accurately predict human-relevant metabolic responses, limiting the development of safer therapeutics and formulations. Here, we present a whole-tissue ex vivo liver screening platform that preserves native hepatic architecture and metabolic function, enabling real-time evaluation of drug metabolism and hepatotoxic metabolite formation. Using acetaminophen (APAP) as a model compound, we characterized its metabolic pathways and identified interventions that reduce the generation of toxic metabolites ex vivo. By integrating this experimental platform with state-of-the-art machine learning, we discovered two previously unrecognized functional excipients that effectively prevented APAP-induced hepatotoxicity in vivo in mice. To demonstrate translational potential, we further developed a solid oral dosage form that enables the controlled co-release of APAP and the protective excipients. Together, this integrated platform provides a scalable strategy for predicting drug metabolism, identifying safer formulation components, and accelerating the development of next-generation therapeutics.



Tzyy-Ping Jung

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Biography

Dr. Tzyy-Ping Jung serves as the Director of the Swartz Center for Computational Neuroscience at the University of California, San Diego. He also holds adjunct professorships at esteemed institutions in Taiwan and China, including National Yang Ming Chiao Tung University (NYCU). An internationally recognized pioneer in neural engineering, he focuses his research on real-world neurophysiological monitoring, computational neuroscience, multimodal signal processing, brain-computer interfaces (BCIs), and NeuroAI.

Deeply committed to international academic exchange, Dr. Jung has collaborated closely with numerous research institutes in Taiwan over the past 22 years, co-authoring more than 80 of his 240+ journal articles. Over 70 of these involved co-authors from NCTU/NYMU/NYCU. He has also hosted more than 50 visiting students and 10 visiting professors from Taiwan. His interdisciplinary work spans cognitive science, computer science, bioengineering, and electrical engineering. Globally celebrated for his seminal contributions to blind source separation in biomedical applications, he is an IEEE Fellow and a Fellow of the Asia-Pacific Artificial Intelligence Association (AAIA). According to Google Scholar, his work has received over 54,000 citations and has an h-index of 98, reflecting his profound scientific impact.

From Prognosis to Actuation: The Next Engineering Frontier for Brain-Computer Interfaces

Tzyy-Ping Jung
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Abstract

Much of the research on Brain-Computer Interfaces (BCIs) still emphasizes reactive detection, which involves classifying a state after it has already occurred, rather than anticipating it. Drawing on three decades of our work with EEG-based BCIs, this presentation outlines two engineering approaches for developing proactive BCIs: predictive architectures that decode continuous EEG data to anticipate performance declines in real time, and forecasting architectures that identify risk markers from brief resting-state recordings. Case studies on sleep, migraines, and cognitive-state monitoring illustrate the challenges these architectures face in signal acquisition, feature selection, and generalization, particularly as large EEG foundation models change what is feasible in the field.

Our recent review of over 300 published systems reveals that only 5.1% (16 papers) implement closed-loop actuation, highlighting a Protection Gap. This indicates that while generalizable prediction is necessary, it is not sufficient on its own. (Protection here is a conceptual term, not standard terminology in the field: it encompasses prevention — interventions before onset — as well as treatments or mitigations when prevention fails.) Thus, bridging the gap between forecasting and actuation represents the next frontier for this field.

Li-Wei Ko 柯立偉

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Biography

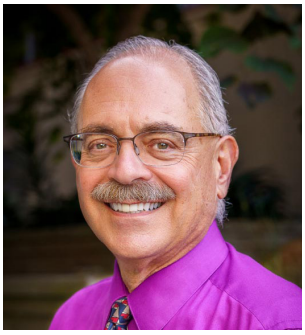
Li-Wei Ko is currently a professor at the Institute of Electrical and Control Engineering, Department of Electronics and Electrical Engineering, National Yang Ming Chiao Tung University, Taiwan. He is also an affiliate scholar at the Institute for Neural Computation at UCSD. Dr. Ko’ s primary research focuses on neural engineering, digital IC App. for brain signal computing, AI machine learning, especially in brain-computer interface, real-world neuroimaging, and neural computation in clinical applications. Dr. Ko’ s work is well-cited by peers (8398 total citations, h-index = 50). For three consecutive years (2022 to 2025), and received Taiwan NSTC’ s 2025 Outstanding Research Award. Dr. Ko was selected in the field of Artificial Intelligence & Image Processing, Single Year, as part of the Top 2% Scientist Rankings by Stanford/Elsevier. He also serves as the Associate Editors of the IEEE Transactions on Fuzzy Systems (IF =10.2), Archives of Gerontology and Geriatrics, International Journal of Fuzzy Systems.

AI-driven Wearable Brain Computer Interface for Smart Healthcare

Li-Wei Ko
Institute of Electrical and Control Engineering National Yang Ming Chiao Tung University

Abstract

Wearable brain-computer interfaces (BCIs) are becoming a practical way to decode brain intention beyond controlled laboratory settings. In this talk, we will present how next-generation wearable BCI is enabled by the combined progress of wireless EEG and AI-driven signal processing algorithms. We will briefly review the evolution of wearable and wireless EEG, then highlight how modern AI algorithms can enhance BCI performance in intention decoding, robustness to noise and individual variability, and real-world usability for continuous and long term use. We will also discuss key considerations for translating these methods into deployable systems, such as real-time processing and reliable operation in everyday environments. Finally, we will showcase smart healthcare applications, including next-generation neurorehabilitation systems, research that helps decode the neural mechanisms of migraine, and clinically oriented solutions such as assistive diagnosis neurofeedback for ADHD, and sleep improvement systems. Together, these advances point toward a scalable and clinically impactful wearable BCI for future smart healthcare.



Albert P. Pisano

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Biography

A self-described technology polymath, Albert P. Pisano’ s research is driven by a passion for developing, mastering and advancing technologies to solve problems. Recent research includes 1) micro-electro-mechanical systems (MEMS) wireless sensors for harsh environments (600° C) such as gas turbines and geothermal wells, and 2) new, additive, MEMS manufacturing techniques such as low-temperature, low-pressure nano-printing of nanoparticle inks and polymer solutions. Other research interests and activities include MEMS for a wide variety of applications, including RF components, power generation, drug delivery, strain sensors, biosensors, micro inertial instruments, disk-drive actuators and nanowire sensors. He is a co-inventor listed on more than 20 patents in MEMS and has co-authored more than 300 archival publications.

Pisano is also developing larger sensors that can be manufactured at extremely low cost and made from sustainably sourced polymers for use in health, environmental monitoring, food safety and other applications.

Pisano is a co-founder of ten start-up companies in the areas of transdermal drug delivery, transvascular drug delivery, sensorized catheters, MEMS manufacturing equipment, MEMS RF devices and MEMS motion sensors. In 2008, he was named one of the 100 Notable People by Medical Devices and Diagnostic Industry (MD&DI) Magazine.

Since 1983, Pisano has graduated over 40 Ph.D. and 75 MS students. He has hosted four visiting industrial fellows in his lab since 2005.

Wearable Sensors for Healthcare Engineering

Albert P. Pisano
Walter J. Zable Distinguished Dean
Jacobs School of Engineering
Special Adviser to the Chancellor
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Abstract

Advances in microelectronics, flexible electronics, and sensors have made possible a new class of wearable sensors, which can be called "unawarables" due to their low profile and minimal invasiveness. These sensors have the potential of gathering longitudinal metabolic data from the ambulatory population, and uploading it to the digital medical health records. This technology will provide the data that medical AI systems need, reduce the cost of obtaining metabolic data from patients and provide physicians the ability to monitor their patients in a proactive manner. This talk will review the technologies of the wearable sensors, and provide several examples of prototypes already developed and tested.

Paul C.-P. Chao 趙昌博

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Biography

Paul C.-P. Chao received his Ph.D. degree from Michigan State University, USA, and then with Chrysler Corp, Detroit, USA before joined National Yang Ming Chiao Tung University (NYCU) Taiwan. He is currently a Distinguished Professor and University Chair of the Electrical Engineering Department at NYCU; a Distinguished Lecturer for IEEE Sensors Council, 2018 – 2020, USA. His research interests focus on sensors, actuators and their interface circuitry. Dr. Chao has published more than 450 peer-reviewed papers (books, journal papers, conferences, reports) and 38 patents. Dr. Chao was the recipient of the 1999 Arch T. Colwell Merit Award from Society of Automotive Engineering, Detroit, USA; The 2019 Technical Achievement Award, IEEE Sensors Council; The 2017 Presidential Outstanding Professor of Engineering in Nation (Taiwan) (awarded by the president of the nation in the Presidential House of Taiwan, ROC); The Distinguished Institutional Award, American Society of Mechanical Engineering (ASME), USA in 2019. Dr. Chao has served as University Associate Vice Presidents of NYCU for academic affairs (2009-2010) and research and development (2015). For editorial services, he is currently the Editor in Chief of IEEE Journal of Selected Areas in Sensors; Topical Editors of IEEE Sensors Journal and IEEE IoT Journal. He is a fellow for both IEEE and ASME.

Achieving High Accuracy in Predicting Blood Glucose Levels Using Dual Long Wavelengths 1480 nm and 1640 nm PPG and Machine Learning

Duc Huy Nguyen, Duc Thang Ngo, Paul C.-P. Chao
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Abstract

Blood glucose is a vital physiological sign for managing and detecting diabetes, particularly given the rising global prevalence of obesity and its associated risks. This study proposes a novel, non-invasive glucose monitoring system that integrates photoplethysmography (PPG)-based sensing, deep learning-based signal quality assessment, and machine learning-based glucose prediction. A dual long-wavelength PPG device of 1480 and 1640 nm was used to capture blood volume changes, enabling continuous glucose monitoring without the discomfort of finger-prick or implantable devices. A convolutional neural network combined with long short-term memory (CNN-LSTM) architecture was employed to evaluate signal quality in real-time, achieving 96.3% accuracy, 94.9% sensitivity, and 96.7% specificity. High-quality PPG segments were then used to train and test glucose prediction models, including XGBoost, LightGBM, and Random Forest. Among them, XGBoost achieved the best performance with an RMSE of 12.474 mg/dL, MAE of 7.279 mg/dL, R^2 of 0.955, and a Pearson correlation of 0.980, demonstrating competitive performance compared with previously reported approaches. These results demonstrate well that the adoption of 1480 and 1640 nm plays a key role for the first time to offer successfully accurate prediction of glucose, presenting a clinically meaningful, real-time, and user-friendly solution for ubiquitous home-based diabetes management.

Wen-Liang Chen 陳文亮

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Biography

Wen-Liang Chen received doctoral training in biotechnology from National Yang Ming Chiao Tung University (NYCU), Taiwan, where he currently serves as a Distinguished Professor and Chair of the Department of Biological Science and Technology.

Professor Chen’ s research spans smart agriculture, immunodiagnostics, antibody engineering, and biosensor and biochip development. His work is distinguished by a strong emphasis on interdisciplinary integration, bridging biological science, sensor engineering, the Internet of Things (IoT), artificial intelligence, and translational biomedical technologies.

A central focus of his research is the development of intelligent sensing and decision-support platforms. These systems employ distributed sensors to continuously acquire physiological, biological, and environmental data, which are transmitted through IoT-enabled networks to cloud-based infrastructures for real-time integration and analysis. By applying advanced machine-learning and artificial-intelligence approaches, his research group develops predictive and decision-support models for both agricultural and biomedical applications.

In the field of smart agriculture, Professor Chen has successfully established multiple AI-driven models for the early prediction, risk assessment, and management of plant diseases and insect pests. These technologies support precision crop management, improve agricultural productivity, reduce unnecessary agrochemical inputs, and contribute to more sustainable farming practices.

In parallel, he has developed highly sensitive biosensing and biochip technologies for rapid disease detection. His team has further advanced these platforms toward scalable and standardized manufacturing, thereby strengthening their potential for industrial production, clinical validation, and commercialization. Through the integration of engineering innovation with biological and medical applications, Professor Chen’ s research contributes to the translation of intelligent diagnostic technologies from laboratory development to real-world implementation.

Comprehensive Electrochemical Platform for Disease Prevention and Detection

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Abstract

Rapid, accurate, and decentralized diagnostics are fundamental to disease prevention and precision healthcare. We propose a Comprehensive Electrochemical Platform for Disease Prevention and Detection centered on an advanced electrochemical point-of-care testing (POCT) chip based on electrochemical impedance spectroscopy (EIS) technology. Unlike conventional optical assays, the label-free EIS platform directly measures biomolecular interactions through impedance changes at the electrode interface, enabling highly sensitive, rapid, and quantitative detection while eliminating complex labeling procedures. The miniaturized sensor requires only a small sample volume, provides results within minutes, and is readily scalable for low-cost mass production.

The POCT chip integrates microfabricated gold electrodes, nanostructured surface modification, and highly specific antibody or aptamer recognition elements to achieve exceptional analytical sensitivity, specificity, and reproducibility. Its modular sensing architecture supports multiplex detection of diverse biomarkers, including proteins, antibodies, nucleic acids, and extracellular vesicles, allowing early diagnosis and continuous monitoring of cardiovascular diseases, cancers, infectious diseases, neurodegenerative disorders, and metabolic diseases. Furthermore, the platform is compatible with standardized manufacturing processes and portable handheld readers, facilitating clinical translation and large-scale deployment.

To extend diagnostic capability beyond biomarker detection, the electrochemical platform is integrated with artificial intelligence (AI), cloud computing, and Internet of Medical Things (IoMT) technologies. Electrochemical signatures are combined with longitudinal physiological and clinical data to establish predictive models for disease risk assessment, early warning, and personalized health management. This intelligent framework transforms conventional diagnostics into a proactive disease prevention system capable of real-time monitoring and precision clinical decision support.

By integrating high-performance EIS biosensing, intelligent data analytics, and portable POCT technologies, the proposed platform establishes a next-generation diagnostic ecosystem that bridges laboratory-grade analytical performance with decentralized healthcare. This comprehensive electrochemical platform has significant potential to accelerate precision medicine, improve healthcare accessibility, reduce medical costs, and support sustainable disease prevention and management in both clinical and community settings.

Yu-Hwa Lo

Position : Professor

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Biography

Yu-Hwa Lo (羅彧華) received his B.S. degree in electrical engineering from National Taiwan University in 1981 and his M.S. and Ph.D. degrees in EECS from the University of California, Berkeley, in 1986 and 1987, respectively. From 1987 to 1990, he was a member of the technical staff at Bellcore. He joined Cornell University as an assistant professor in 1991 and became an associate professor in 1996. Since 1999, he has been a professor in the Department of Electrical and Computer Engineering at the University of California, San Diego, where he currently holds the Distinguished William S. C. Chang Endowed Chair. He has also served as a visiting professor in the Department of Bioengineering at National Chiao Tung University, now part of National Yang Ming Chiao Tung University, in Taiwan.

Professor Lo is the founding director of the San Diego Nanotechnology Infrastructure and site director of the NSF National Nanotechnology Coordinated Infrastructure at UCSD. He serves as a member of the Advanced Research Advisory Committee and chair of the Technical Advisory Committee for Electronics and Optoelectronics at Taiwan’ s Industrial Technology Research Institute (ITRI). He previously served as chief technology officer and a board director of Nova Crystals. He is a co-founder and advisor of NanoCellect Biomedical and Colligo Biomedical and has consulted for biotechnology and pharmaceutical companies.

His research interests include biophotonics, single-photon detectors, condensed-matter physics for advanced device concepts, microfluidics, biosensors, artificial intelligence, and biomedical systems. He has published approximately 600 articles and holds 59 patents in semiconductor and optoelectronic materials and devices, as well as biomedical devices and systems. Many of his inventions have been commercialized and broadly applied in the semiconductor, optoelectronics, and biotechnology industries.

His received NSF CAREER Award, the Lilly Foundation Award, a NASA Innovation Award, and several best-paper and teaching awards. He is a Fellow of IEEE, Optica, the International Academy of Artificial Intelligence Sciences, and the National Academy of Inventors (NAI).

Label-Free, AI-Guided Cell Sorting for Single-Cell Multi-Omics and Cell Therapy

Yuhwa Lo
Electrical and Computer Engineering,University of California San Diego

Abstract

We have developed a unique technology that integrates image-guided cell sorting, real-time artificial intelligence (AI) inference, and generative adversarial networks (GANs) to analyze, classify, and isolate biological cells without labeling. Genomic, proteomic, and metabolic analyses of the sorted cells enable correlations between single-cell morphological features and multi-omic profiles. Once trained using these paired datasets, the system can predict the molecular properties, functional states, and potential fates of individual cells directly from their images.

This approach provides a powerful new platform for multi-omics research and cell therapy, particularly for identifying and isolating cells that lack known or specific biomarkers. Unlike conventional fluorescence-activated cell sorting, which relies primarily on predefined molecular markers, our technology uses information-rich cellular morphology to classify known cell populations and discover previously unrecognized cell types or states. It can characterize the current condition and behavior of cells while also predicting their future properties and developmental trajectories with high accuracy.

To further expand the information available for AI analysis, we have extended the platform from 2D imaging to 3D single-cell tomography. The system can generate 3D images at a throughput of approximately 1,000 cells per second—roughly 1,000–10,000 times faster than conventional fluorescence confocal microscopy. Three-dimensional cellular images capture substantially richer morphological and subcellular information than 2D images, strengthening the ability of AI and machine-learning models to associate cell structure with genomic, proteomic, and metabolic characteristics.

By combining high-throughput, label-free 3D imaging, intelligent cell sorting, and multi-omic validation, this platform offers a broadly applicable strategy for cell classification, rare-cell discovery, phenotype prediction, and the selection of therapeutically valuable cell populations.

Dar-Bin Shieh, DDS, DMSc 謝達斌

Position : Senior Vice President and Chair Professor

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Biography

Prof. Dar-Bin Shieh is Senior Vice President of National Yang Ming Chiao Tung University (NYCU) and Chair Professor at the Institute of Oral Medicine, National Cheng Kung University (NCKU). He holds a Doctor of Medical Science in Molecular Biology from Harvard School of Dental Medicine (1997) and practices as a clinician-scientist in oral pathology, oncology, and translational nanomedicine. His research spans nanomedicine, biophotonics, mitochondrial medicine, and cancer biology. Key contributions include zero-valent iron nanoparticles (ZVI-NPs) as dual-function cancer immunotherapeutics — simultaneously inducing ferroptotic tumor-cell death and reprogramming the immunosuppressive microenvironment — and a sustained program on gelsolin's role in chemoresistance across gynecologic, head-and-neck, and lung cancers. His group produced consecutive cover-story publications in JACS, the Journal of Dental Research, Theranostics, Nature and more. He recently pioneered exosome-based biosensing platforms for early cancer diagnosis and chemotherapy-response prediction. These programs are reflected in an h-index of 48, over 9,300 citations, and more than 140 publications, sustained by nine consecutive years of National Nanotechnology Program funding and a six-year Taiwan–Canada joint collaboration in molecular imaging. Since 2013, Prof. Shieh has served as Founding Chair of iMANI (International Institute of Macromolecular Analysis and Nanomedicine Innovations) at NCKU, an institute he designed to combine CryoEM and computation to translate molecular discoveries to the clinic — producing multiple technology transfers and one product now in Phase III clinical trials. At NCKU he also united physician-scientists with engineering and science faculty into cross-disciplinary teams that have continuously secured large-scale national program grants in genomics, nanotechnology, and medical device development. From 2019 to 2022 he served as Deputy Minister of Taiwan's Ministry of Science and Technology, shaping national policy on biomedical innovation and international research collaboration. As Senior Vice President of NYCU, he leads institutional strategy across international partnerships, interdisciplinary research development, and science-policy engagement. His honors include the National Innovation Award six times (2014–2020, including the 2020 Renewal and Excelsior Award), the Y.Z. Hsu Chair Professor Award in Nano Science and Technology (2018), Future Tech Awards (2021, 2023), the Moderna Taiwan mRNA Innovation Award (2024), the Best Basic Science Research Award from the International Conference on Oral Cancer, and the IEEE Tainan Section Outstanding Technical Achievement Award.

Nano-Enabled Biosensing Platforms: Bridging Rapid Virus Diagnostics and AI-Guided Multi-Cancer Detection

Dar-Bin Shieh[1,2,3]

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Abstract

Emerging infectious diseases and cancer share a common diagnostic bottleneck: conventional methods are too slow, too complex, and too costly for the speed and scale modern healthcare demands. We address both challenges through two complementary nano-enabled biosensing platforms that translate materials engineering directly into clinical utility.

The first is a flexible surface-active metal nano–thin-film (M-NTF) electrode for electrochemical biosensing. Biomarkers or binding partners conjugate directly to its surface within 20–30 minutes, enabling rapid, reconfigurable detection without elaborate sample preparation. Applied to SARS-CoV-2 stability, the platform demonstrated that the virus retains substantially higher infectivity at − 20 ° C than at 4 ° C over 30 days — corroborating cold-chain transmission risks identified in the WHO–China joint investigation. The same architecture probes novel infection pathways, screens antiviral peptides and drugs, and reconfigures readily for enteroviruses or other emerging pathogens by swapping variant-specific probes.

The second targets cancer through an AI-guided extracellular vesicle (EV) biochip. A 250 MHz shear-horizontal surface acoustic wave (SH-SAW) sensor detects intact tumour-derived exosomes label-free via mass-loading-induced phase shifts, achieving a detection limit of 9.6 × 10^6 particles/mL — substantially faster than ELISA. Using oral squamous cell carcinoma (OSCC) and pancreatic ductal adenocarcinoma (PDAC) as clinical models, an AI-selected seven-biomarker panel (CDH3, DSC3, EPHA2, PD-L1, CD44, GPC1, CD9) drove machine-learning classification to 94.1% accuracy and 97% specificity in single-blinded tests across NCKU Hospital and Chi Mei Medical Center biobanks, embodied in the "Smart CExoSense One" device.

Together, these platforms form a versatile, low-cost toolkit spanning viral surveillance, cold-chain food-safety monitoring, antiviral drug screening, and early multi-cancer detection — a convergent foundation for next-generation precision medicine and biosecurity.

Ethan I. Lan 蘭宜錚

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Biography

Ethan received his B.S. degree in Chemistry from the University of Maryland College Park in 2009. After getting his B.S. degree, he joined UCLA Biomedical Engineering Interdepartmental Program in 2009 where he worked with Professor James C. Liao (member of the National Academy of Science, National Academy of Engineering, and Academia Sinica) on the development of microbial cell factory for biofuel production. His research in the metabolic engineering of cyanobacteria achieved the first direct photosynthetic conversion of CO2 into 1-butanol. After completing his PhD degree, he briefly continued his work with Professor James Liao on the development of novel metabolic pathways. He started his academic career as an assistant professor in the Department of Biological Science and Technology of National Chiao Tung University in Fall of 2014. He is currently a professor at the Department of Biological Science & Technology and has served as the Associate Dean of the College of Engineering Bioscience since 2020. He is also the recipient of Ministry of Science & Technology Young Scholar Fellowship and International Outstanding Young Scholar Award from the Foundation for the Advancement of Outstanding Scholarship. His recent research achieved the first de novo biosynthesis of 3-hydroxy-3-methylbutyrate, as well as the synthesis of several platform chemicals from CO2 using metabolically engineered cyanobacteria.

Development of a novel de novo biosynthetic pathway for 3-hydroxy-3-methylbutyrate production

Ethan Lan
Department of Biological Science & Technology,National Yang Ming Chiao Tung University

Abstract

3-Hydroxy-3-methylbutyric acid (HMB) is an anti-catabolic agent currently used as a dietary supplement to assist treating sarcopenia and athletic muscle training. Its current synthesis relies on chemical processes that use and produce harmful products and is non-renewable. Using a retro-synthesis approach, we first developed a novel de novo biosynthetic pathway for producing HMB from central metabolite acetyl-CoA. This synthetic pathway was introduced to lab-strain of Escherichia coli BL21(DE3) and achieved a production titer of 18 g/L with a yield of 0.16 g/gglucose. To our surprise, HMB biosynthesis using this synthetic pathway by the probiotic E. coli Nissle 1917 was significantly less successful with a titer of approximately 10% of that from BL21(DE3). Upon systematic evaluation, we identified that availability of coenzyme A (CoA) is crucial to the functional performance of our synthetic HMB-producing pathway. With further host engineering, we were able to develop a antibiotic-resistance-free strain of E. coli Nissle 1917 that produces HMB to the g/L level. The results from our study provided a green manufacturing route to the production of HMB. Furthermore, HMB-producing probiotic strain also offers a potential engineered live biotherapeutic application for treating sarcopenia.

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