

39⁴年物醫學 聯合學術年會

Advancing Therapies in Cancer and Diseases

2025 The 39th Joint Annual Conference of Biomedical Science

大會手冊

時間 03.22 sat. 03.23 sun.

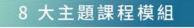
地點

國防醫學院

中華民國免疫學會 | 台灣分子生物影像學會 | 台灣生物化學及分子生物學學會 | 中華民國細胞及分子生物學學會 中華民國臨床生化學會 | 台灣毒物學學會 | 中國生理學會 | 台灣藥理學會 | 中華民國解剖學學會



動物實驗 3R 科學埕是匯聚動物實驗專業教育資源的平台,以「專業職能再造 3R 落地生根」為願景目標,依循跨部會人才培育分工,規劃建構動物實驗科學 職能導向的繼續教育課程模組與試證體制,並透過課程審查與學習時數認列的 方式,串接國科會、教育部、農業部的教育課程,匯集跨部會教育能量共同組建 動物實驗 3R 科學埕,持續完備與擴充教育資源。



將動物實驗科學梳理成各個主題課程,以能夠落實於實務應用的教育內容展開 核心課綱,網羅相關專業課程,提供動物實驗參與者汲取新知、增強知能、持續 學習的資源管道。



7 項專業職能檢定考試

依據不同工作角色的專業能力需求,制定專業職能檢定考試,通過職能檢定考試, 代表已經完成該職能內容基本知能之學習,並能配合所投入職場之工作需求, 進行實務訓練。



歡迎參與動物實驗的夥伴們一起加入科學埕,開始規劃自己的學習計畫,透過 持續學習、提升職能、瞭解最新趨勢,為自己的職涯加分升級!









JACBS transal Conference of Biomedical Science		
		h生非 聯佔
		int Annual Con
	2	大會會長的話
	3	理事長的話
	4	交通示意圖 &
	6	會場平面圖
7	10	參與學會暨理
4	11	會議資訊暨特
	12	大會議程
-	15	大會特別演講
	19	陳炯霖轉譯醫
	23	學會特別演講
4	45	研討會演講
	143	科技新知研討
	147	論文報告資訊
5	228	贊助廠商
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接駁車訊息

事長與秘書長名單

別演講及會員大會時間表

學講座 特別演講暨頒獎典禮

大會會長的話

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各位尊敬的來賓、學界先進:

JACBS

歡迎您參加本屆生物醫學聯合學術年會!此次盛會匯聚了來自生物醫學領域的專家學 者,此次由中華民國免疫學會主辦,以及八大協辦學會,包含分子生物影像學會、生物化學 及分子生物學學會、細胞及分子生物學學會、臨床生化學會、毒物學學會、生理學會、藥理 學會、解剖學學會,致力於推動醫學與科技的進展。

今年,我們將聚焦於主題「Advancing Therapies in Cancer and Diseases」,探討如何在 癌症、免疫疾病等方面推動創新治療,這些疾病不僅影響著患者的生活品質,也對社會與醫 療領域貢獻了許多寶貴的研究與心力。

非常榮幸邀請到瑞士洛桑大學及路德維希癌症研究所的何秉智博士擔任本屆年會的主題 演講嘉賓。何博士專精於癌症及自體免疫疾病中的免疫反應代謝調節研究,他深入探索代謝 重編程如何恢復免疫功能,並以此作為新穎的治療策略。他的研究成果不僅發表於《Cell》 《Nature Immunology》等國際頂尖期刊,並獲得多項專利及獎項肯定。何博士的研究進展 對於癌症及免疫疾病的治療方法提供了重要啟發,也與本屆大會主題密切契合。

著生物技術的發展與精準醫療的普及,我們正處於一個醫學快速革新的時代。本次年會 透過何博士的演講及其他前沿研究的分享,旨在啟發與會者在癌症、神經退行性疾病等領域 持續探索,為人類健康提供更多前瞻性方案。

此外,為了推動更多年輕學者與研究者的投入,我們也特別設立了「大會主題競賽獎」, 鼓勵在癌症和重大疾病治療研究上表現優異的年輕人才,期盼通過這些激勵措施,為未來的 科研工作注入新的活力與創新動能。

在此,謹代表本屆年會籌備委員會, 衷心感謝所有辛勤投入的夥伴與學者們, 感謝各界 學會的支持與協助,還有眾多廠商的參展與贊助,讓此次大會能順利進行。相信在這樣的努 力下,本屆年會將成為促進交流與合作的平台,為癌症及重大疾病的療法開創新的契機。

祝福本次年會圓滿成功,並期待大家能有所收穫!

家子

第 39 屆生物醫學聯合會學術年會 大會會長

理事長的話

各位先進您好:

生物醫學聯合學術年會(JACBS)是國內歷史悠久、規模宏大且極具指標性的學術研討會 由國內九大基礎醫學學會聯合主辦(分別為免疫學會、藥理學會、解剖學學會、分子生物影像學 會、生物化學及分子生物學學會、細胞及分子生物學學會、臨床生化學會、毒物學學會與生理 學會)。歷屆大會均吸引超過 2,300 名學者與研究人員踴躍參與。今年,我們誠摯邀請您參加第 39 屆生物醫學聯合學術年會(The 39th Joint Annual Conference of Biomedical Science, JACBS), 本屆會議由中華民國免疫學會負責籌備與規劃,將於 114 年 3 月 22 日至 23 日在台北市國防醫 學院隆重舉行。

本屆大會以「Advancing Therapies in Cancer and Diseases」為主題,旨在促進會員掌握該領 域最新科技與研究成果。我們特別邀請瑞士洛桑大學及路德維希癌症研究所的何秉智博士擔任 主題演講嘉賓。何博士專注於探討免疫反應中代謝調節在癌症及自體免疫疾病中的角色,並致 力於以代謝重編程(metabolism reprogramming)恢復免疫功能的創新治療方法。他的眾多研究 成果已刊登於 Cell、Nature Immunology、Nature Medicine、Immunity 等頂尖期刊,並屢獲專利 與獎項肯定,對癌症及免疫疾病的治療帶來深遠影響與啟示。

此外,各大學會也邀請了多位國內外知名研究學者參與,其中包括美國 Emory 大學 Emory Vaccine Center 主任 Rafi Ahmed 教授(專注於 T 細胞記憶生成與維持及其在病毒感染中的作用) 以及美國哈佛醫學院 Bertarelli Rare Cancers Fund 的 Marcia Haigis 博士(專研罕見及難治性癌症 的分子機制與治療策略)。會議議程豐富多元,精彩內容不容錯過。 為了激勵學術創新,聯合年會將同步舉辦大會主題論文競賽,誠邀各學會優秀年輕學者踴 躍參與。每年發表的研究論文數量均超過千篇,充分展現出台灣基礎研究的實力與創新活力, **並促進各界間更多万動與交流。**

相信為期兩天的會議將為來自各大學院與研究機構的教授、學者、專家、研究人員及研究 生帶來豐碩收穫。我們誠摯邀請所有對生物醫學懷有熱忱的夥伴,共同參與並推廣 JACBS 的各 項活動,藉由精彩的演講與熱烈的交流,激發青年學子投身醫藥與生物科技研發,進一步厚植 台灣科技創新能量。

諽代表第三十九屆生物醫學聯合學術年會籌備委員會,誠摯歡迎您的蒞臨,期待與您在會 中相見。祝您健康快樂!

總召

台灣生 中華民

第三十九屆生物	n醫學聯合學術年會
3集人:中華民國免疫學會	理事長 葉國偉
中華民國解剖學學會	理事長 郭余民
台灣分子生物影像學會	理事長 林康平
上物化學及分子生物學學會	理事長王育民
R國細胞及分子生物學學會	理事長 司徒惠康
中華民國臨床生化學會	理事長 徐慧貞
台灣毒物學學會	理事長 王應然
中國生理學會	理事長 李昆澤
台灣藥理學會	理事長 林建煌

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交通資訊

前往國防醫學院交通示意圖

年會舉辦地點:

JACBS

國防醫學院 (114 臺北市內湖區民權東路六段 161 號)

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大眾交通工具

搭乘公車:

- 國防醫學院周邊公車:民權幹線(原紅 32)、藍 36、284 直、617、645、903(於「國 防醫學院(網球中心)」下車,步行約5分鐘)。
- 三軍總醫院周邊公車:市民小巴 10、小 3、藍 20、藍 27、棕 9、214、256、278、284、 551、617、630、645、652、903(請於「國防醫學中心」下車,步行約10分鐘)。
- 進入三軍總醫院公車:市民小巴 10、藍 20、藍 27、紅 29、0 東、28、278、284、521、 551、617、645(請於「三總內湖站」下車,繞駛時間為0800-2130時)。

自行開車

行經中山高速公路,內湖成功路交流道出口下,往內湖方向往北走,直行至民權東路與成功 路交叉口後,右轉約 500 公尺左側至國防醫學院大門。

附近停車場資訊

- **臺北市網球中心停車場**(步行約5分鐘) 地址:臺北市內湖區民權東路六段 208 號
- 內湖停車場(步行約5分鐘) 地址:臺北市內湖區民權東路六段 180 巷旁
- 福華商業藝術廣場前 ViVi Park 石潭二站平面停車場(步行約 5 分鐘) 地址:臺北市內湖區民權東路六段 180 巷 23 號 內湖民權星巴克後方
- **三軍總醫院停車場**(步行約 10 分鐘) 地址:臺北市內湖區成功路二段 325 號

接駁車時刻表

 3/22(六)+3/23(日) 接駁時段 08:00-10:30 (人滿即發車)

昆陽捷運站4號出口→國防醫學院(只進不出)

上午班次	昆陽捷運站4號出口 四輛遊覽車人滿即發車,僅開以下時段
1	08:00
2	08:30
3	09:00
4	09:30
5	10:00
б	10:30

3/22(六)+3/23(日)

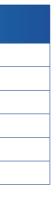
接駁時段 16:00-17:30 (人滿即發車)

國防醫學院 → 昆陽捷運站 4 號出口(只進不出)

上午班次	昆陽捷運站4號出口 四輛遊覽車人滿即發車·僅開以下時段
1	16:00
2	16:30
3	17:00
4	17:30

• 其他時段無接駁車



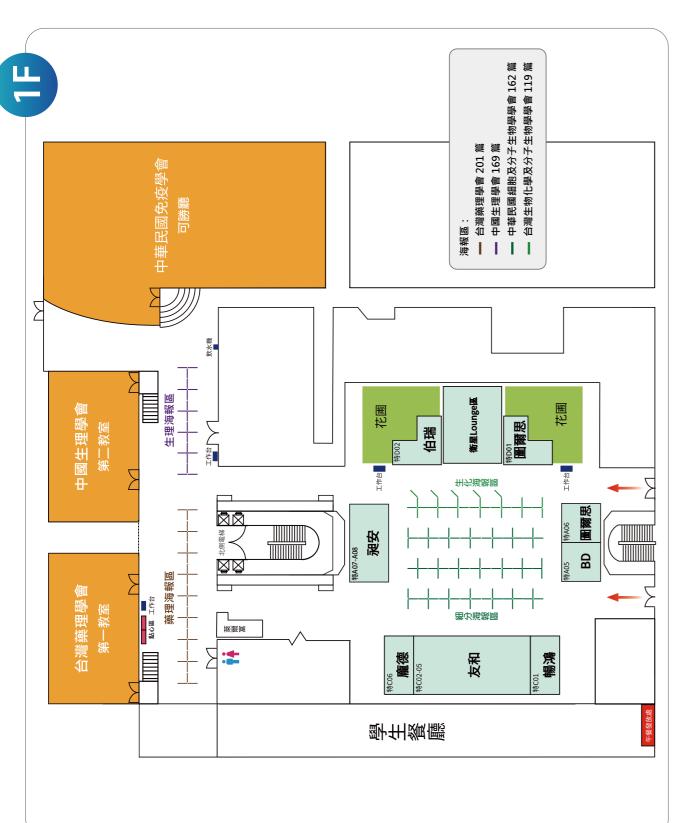


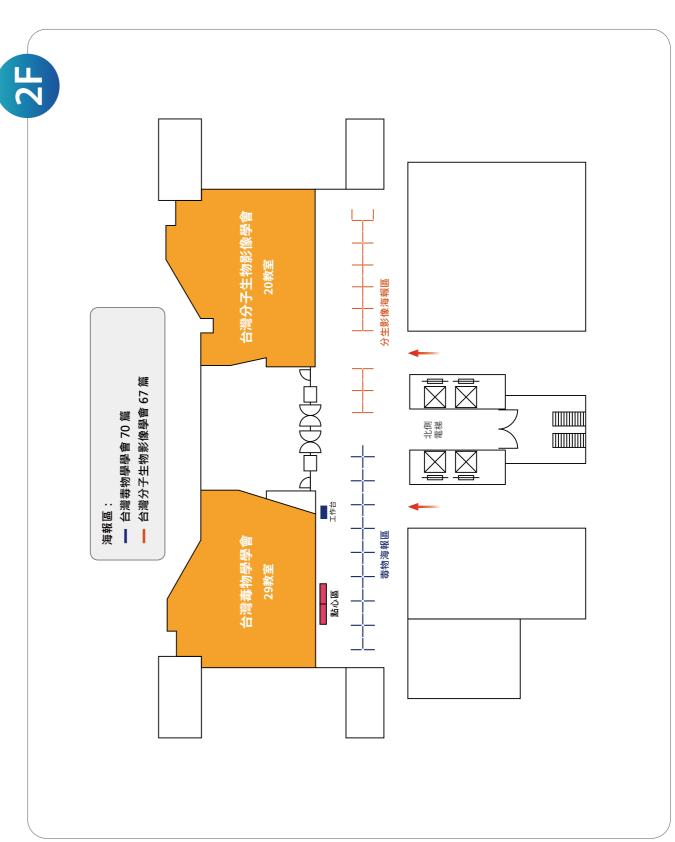
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2025 The 39th Joint Annual Conference of Biomedical Science 生物醫學聯合學術年會

會場平面圖









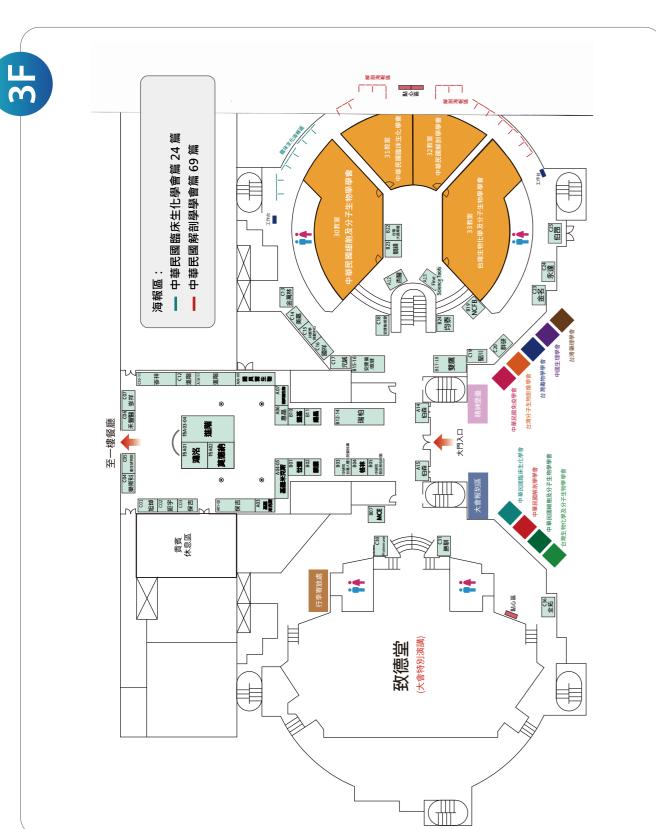
2025 The 39th Joint Annual Conference of Biomedical Science 宝物 合學術 百靈 醫學聯

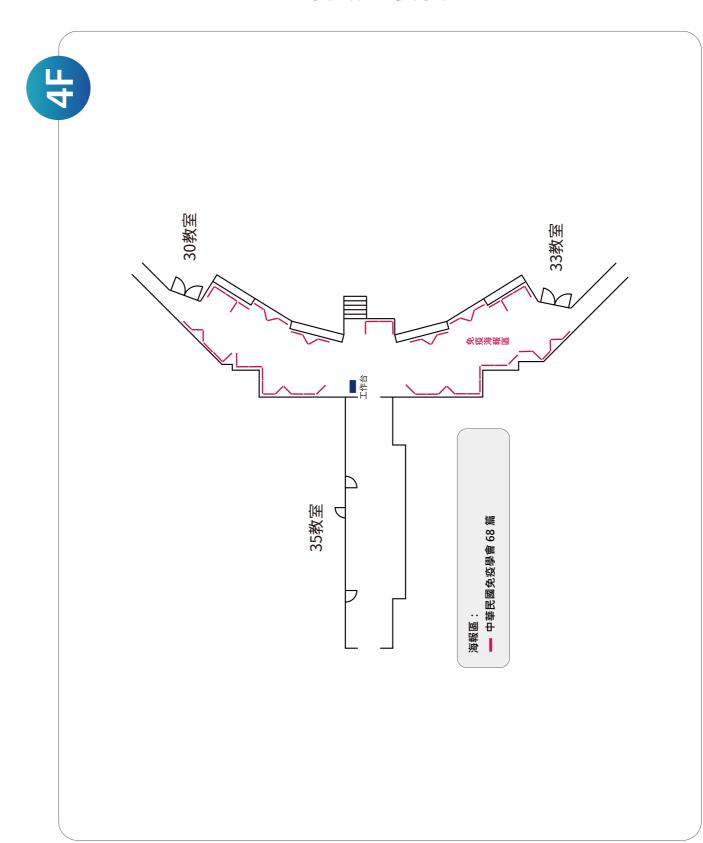
會場平面圖

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JACBS JACBS









大會核心籌備小組

大會會長	大會秘書長	大會財務長
許秉寧	莊雅惠	俞欣慧

第 39 屆生物醫學會聯合學術年會 參與學會暨理事長與秘書長名單

學會名稱	理事長	秘書長
中華民國免疫學會	葉國偉	蘇冠文
台灣分子生物影像學會	林康平	楊邦宏
台灣生物化學及分子生物學學會	王育民	林士鳴
中華民國細胞及分子生物學學會	司徒惠康	李岳倫
中華民國臨床生化學會	徐慧貞	饒梓明
台灣毒物學學會	王應然	夏興國
中國生理學會	李昆澤	林雅婷
台灣藥理學會	林建煌	許銘仁
中華民國解剖學學會	郭余民	王仰高

會議資訊暨特別演講及會員大會時間表

第 39 屆生物醫學聯合學術年會 會議資訊

內容	時間	地點
大會開幕式	114年3月22日 09:30-09:40	
大會特別演講	114 年 3 月 22 日 09:40-10:30	
大會主題口頭論文競賽	114 年 3 月 22 日 15:20-17:00	3 樓致德堂
陳烱霖轉譯醫學講座特別演講	114 年 3 月 23 日 10:50-11:50	
大會主題口頭論文競賽頒獎	114年3月23日 11:50-12:00	

學會特別演講及會員大會時間表

學會名稱	特別演講時間	會員大會時間	地點
中華民國免疫學會	114年3月22日 10:50-12:00		3 樓 30 教室
台灣分子生物影像學會	114年3月23日 10:50-11:40	114年3月23日 11:45-12:00	2 樓 20 教室
台灣生物化學及分子生物學學會	114年3月22日 11:00-12:00	114年3月23日 15:50-16:30	3 樓 33 教室
中華民國細胞及分子生物學學會	114年3月22日 10:50-12:00		3 樓 30 教室
中華民國臨床生化學會	114年3月22日 10:50-12:00	114年3月22日 14:00-14:20	3 樓 31 教室
台灣毒物學學會	114年3月23日 09:00-10:30	114年3月22日 14:00-15:00	2 樓 29 教室
中國生理學會	114年3月22日 10:50 - 12:00	114年3月23日 16:15 - 17:15	1 樓第二教室
台灣藥理學會	114年3月22日 14:00 - 15:00	114年3月22日 15:20-17:00	1 樓第一教室
中華民國解剖學學會	114年3月22日 10:50-11:50	114年3月22日 11:50-12:20	3 樓 32 教室

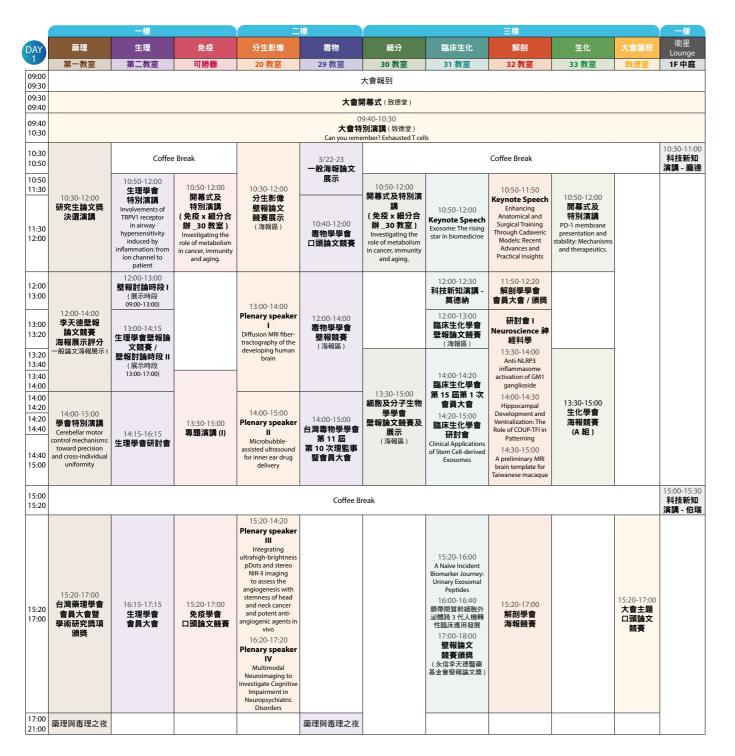


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th 2025 The 39th Joint Annual Conference of Biomedical Science 生物醫學聯合學術年會

大會議程



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DAY	藥理	生理	免疫	分生影像	毒物	細分	臨床生化	解剖	生化	大會	衛星
2	第一教室	第二教室	可勝廳	20 教室	29 教室	30 教室	31 教室	32 教室	33 教室	致德堂	Loung 1F中J
09:00 09:20 09:20 09:40 09:40 10:00	09:00-10:50 學會學術演講	08:30-10:30 生理 口頭論文競賽	09:00-10:30 免疫學會	09:00-10:30 分生影像學會	Keynote Lecture 09:00-09:45 Marijuana: A new risk factor for cardiovascular disease 09:45-10:30	09:30-10:30 細分學會 特別演講		09:00-10:30 解剖學會	09:00-10:30 生化學會		
10:00 10:20	(—) Glymphatic System in Brain Disorders	10:00-11:00 生理學會 壁報討論時段 III (展示時段 09:00-13:00)	海報競賽 (海報區)	口頭報告競賽 (海報區)	Ca ²⁺ release- activated Ca ²⁺ (CRAC) channels as a potential new therapy for treating environmental allergens-house dust mite	Visualizing Connexin Dynamics: Imaging- Based Insights into Cellular Communication and Trafficking	09:30-10:30 臨床生化學會 口頭論文競賽	□頭論文 競賽演講	海報競賽 (B 組)		
10:30 10:50					Ca	offee Break					
10:50	11:00-12:00	10:50 陳炯霖轉譯醫 學 (致得		10:50-11:40 Keynote speaker Theranostics:			10:50-11 陳炯霖轉譯醫學 調 (致德望	冓座特別演講			
10:50 12:00	一般論文海報 展示 II 12:00-13:00 一般論文海報 展示 III	11:50- 大會主題口頭 (致行		Current concept and prospection in the era of personalized medicine 11:45-12:00 分生影像 會員大會、頒獎			11:50-12 大會主題口頭論 (致德堂	文競賽頒獎			
12:00 13:00		12:10-13:30 生理學會餐會					12:00-12:10 臨床生化學會 口頭論文競賽 頒獎				
13:00 13:20					13:00-13:30 研討會 I						
13:20 13:20 13:40					暴露農藥對於腸道 微生物群及代謝體 與腎臟功能下降之 影響探討			研討會 II 數位影像和創新 教學			
13:40 14:00	13:00-15:00 學會學病演講 (二)	13:30-14:30 生理學會 壁報討論時段 Ⅳ (展示時段 13:30-17:00)	13:10-14:10 (免疫 x 細分 合辦) What is T cell exhaustion (30 教室)		13:30-14:00 研討會 II Wastewater-Based Epidemiology for Monitoring the Use of 68 NPS and Conventional Drugs in the Taipei Metropolitan Area, Taiwan, During and After the COVID-19 Pandemic	13:10-14:10 (免疫 x 細分合 辦) What is T cell exhaustion		於解剖教學的應用 13:00-13:30 3D 列印技術在解剖 學教學之應用 13:30-14:00 3D printing in Anatomy Education 14:00-14:30 Decoding the Body:	13:30-14:30 生化學會學會 專題演講 Translational biology		
14:00 14:20	Innate Immunity and Inflammation		14:10-15:00 114 年國科會 微免及檢驗醫學 學門規劃研究		14:00-14:30 研討會 III The Impact of Environmental Pollutants on Tumorigenesis and Therapeutic Efficacy of Anti-Cancer Drugs			The Advantages and Limitations of Virtual Reality in Anatomy Education 14:30-15:00 Redesigning a Flipped Classroom Course and			
14:20 14:40			推動計畫研究 成果發表會:		14:30-15:00 研討會 IV	1		Evaluating Effectiveness in	14:30-14:50 Break Time		
14:40 15:00		14:30-16:30 新進學者 專題演講	計畫申請說明暨申 請經驗分享 (30 教室)		Differential proteomic profiles of lung injury in rat models upon pulmonary exposure to air pollution			Medical Education: Case Study of the Course of "Anatomy"	14:50-15:50 生化學會學會 專題演講 Translational biology		
15:00 15:20					В	Break Time					
15:20 15:20 17:00		16:30-17:00 生理學會口頭及 壁報論文 競賽頒獎典禮	15:20-16:50 專題演講II (免疫 x 細分 合辦) Metabolism and aging (30 教室) 16:50-17:00 免疫學會 閉幕式及頒獎		15:00-15:30 研討會 V Detecting fluorescent-labeled nanoplastics in digestive fluids and tissue using Nano- tracking analysis and near-infrared fluorescence imaging 15:40-16:40 请初學會 問幕式暨論文	15:20-16:50 専題演講 II (免疫×細分 合辦) Metabolism and aging 16:50-17:00 細分學會 開幕式及頒獎		15:20-17:00 解剖學會 特別活動 (全動抽獎)	15:50-16:30 生化學會 會員大會暨 頒獎典禮		

會議資訊 Conference Information



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2025 The 39th Joint Annual Conference of Biomedical Science 合學 術年











Current Position

University of Lausanne Full Professor Department of Oncology Lausanne, Switzerland

Education/Training

- 2015 OTHERS, Yale University Department of Immunobiology, School of Medicine New Haven, CT, USA
- 2012 PhD, University of Minnesota Department of Pharmacology, School of Medicine Minneapolis, MN, USA
- 2008 OTHERS, University of Minnesota Department of Pharmacology, School of Medicine Minneapolis, MN, USA

Professional and Research Experience

- 2023-Present University of Lausanne Full Professor Department of Oncology Lausanne, Switzerland
- 2023-Present Ludwig Institute for Cancer Research Full Member Lausanne, Switzerland
- University of Lausanne Associate Professor Department of Oncology(Tenured) 2019-2022 Lausanne, Switzerland

Awards and Honors

- 2024 Clarivate Highly Cited Researchers
- Henry Kunkel Society member 2024
- 2023 Clarivate Highly Cited Researchers

Can you remember? Exhausted T cells

何秉智 Ping-Chih Ho University of Lausanne Full Professor Department of Oncology Lausanne, Switzerland

Cancer immunotherapies that harness tumoricidal activity of tumor-reactive T cells represent a major breakthrough of current paradigm for treating cancer patents. However, the unstable immunogenicity of tumor cells and highly immunosuppressive tumor microenvironments in solid tumors present the challenges for current immunotherapies. Deciphering the underlying mechanisms utilized by tumor cells to impede tumoricidal activity of infiltrating immune cells and to reduce their immunogenicity is direly needed. Recent studies revealed that the metabolic competition over nutrients between tumor and immune cells in the tumor microenvironment causes metabolic crisis for infiltrating immune cells, especially T cells. This process impairs metabolic fitness of tumor infiltrating T cells and results in T cell dysfunction and formation of an immunosuppressive tumor microenvironment. Therefore, the intensive metabolic communication between tumor and T cells could determine the aggressiveness and immunogenicity of tumor cells. Here, I will discuss how T cell mediated immunosurveillance shapes the metabolic activity of tumor cells via an "immunometabolic editing" process. Tumor cells could acquire the edited metabolic advantages to support their unrestricted growth and immune evasion through this undefined editing process. Given that deregulated metabolic activity is hallmark of most solid tumors that contributes to the outgrowth of tumor cells, new knowledge gained from this new dimension of immunoediting will be transformative for developing new immunotherapies and metabolism targeting strategies to successfully eradicate a broad range of malignancies.



3/22 (Sat.) 09:40-10:30 3樓,致德堂







ila 2025 The 39th Joint Annual Conference of Biomedica



JACBS

謝清河 peaker / Patrick C.H. Hsieh

Current Position

Distinguished Research Fellow and Chief, Division of Cardiovascular and Metabolic Diseases, Institute of Biomedical Sciences, Academia Sinica, Taipei, Taiwan Professor, National Taiwan University College of Medicine and Kaohsiung Medical University Member, Healthy Taiwan Promotion Committee, Presidential Office of R.O.C., Taiwan

Education/Training

1992 MD, Medicine, Kaohsiung Medical College 2003 PhD, Bioengineering, University of Washington, Seattle

Professional and Research Experience

2017-2021 Affiliate Attending Surgeon, Cardiovascular Surgery Division, NTU Hospital 2013-Present Professor, Institute of Medical Genomics and Proteomics, NTU College of Medicine 2009-Present Assistant/Associate/Full/Distinguished Research Fellow, Institute of Biomedical Sciences, Academia Sinica

Awards and Honors

- 2024 Academia Award, Ministry of Education
- 2024 Tien-Te Lee Outstanding Biomedical Award
- Distinguished Alumnus Award, Kaohsiung Medical University 2021

Gut Bacteria and Heart Healing: The Hidden Players in Post-**Infarction Resilience**

謝清河 Patrick C.H. Hsieh

Distinguished Research Fellow and Chief, Division of Cardiovascular and Metabolic Diseases, Institute of Biomedical Sciences, Academia Sinica, Taipei, Taiwan, Professor, National Taiwan University College of Medicine and Kaohsiung Medical University Member, Healthy Taiwan Promotion Committee, Presidential Office of R.O.C., Taiwan

Discover the surprising connection between gut bacteria and heart healing after a heart attack. This presentation explores how the trillions of microbes living in our gut influence the recovery process, particularly through their impact on the immune system and the production of key compounds called short-chain fatty acids. Special attention is given to butyrate-producing bacteria, which have been shown to play a vital role in protecting the heart after injury. Learn about studies in humans and animals that reveal how these beneficial microbes can boost heart health by producing beta-hydroxybutyrate, a molecule linked to improved heart function. This talk sheds light on how gut microbes and their metabolites interact with the body's immune system to support heart repair. It also opens the door to exciting possibilities for new therapies that harness the gut-heart connection to improve recovery and overall cardiovascular health.

陳烱霖轉譯醫學講座 特別演講暨頒獎典禮 The Chiung-Lin ChenTranslational Medicine Award

3/23 (Sun.) 10:50-11:50 3樓,致德堂



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2025 The 39th Joint Annual Conference of Biomedical Science 合學術年 會







th 2025 The 39th Joint Annual Conference of Biomedical Science 生物醫學聯合學術年會





Current Position

Associate Professor, Institute of Pharmacology, College of Medicine, National Taiwan University, Taiwan

Attending Physician, Department of Medical Research, National Taiwan University Hospital, Taiwan

Education/Training

- 2014 Ph.D., Institute of Physiology, National Taiwan University College of Medicine
- 2004 M.D., National Taiwan University College of Medicine

Professional and Research Experience

- 2020-2024 Attending Physician, Division of Hematology-Oncology, Kaohsiung Change Memorial Hospital
- 2019-2022 Assistant Professor, Institute of Pharmacology, College of Medicine, National Taiwan University
- 2011-2019 Attending Physician, National Taiwan University Hospital

Awards and Honors

- 2024 Wu Ho-Su TBF Medical Award, Taiwan Bio-developmental Foundationg Physician
- 2024 Outstanding Research Award, National Science and Technology Council
- 2020 National Innovation Award

Cerebellar motor control mechanisms: toward precision and crossindividual uniformity

潘明楷 Ming-Kai Pan

Associate Professor, Institute of Pharmacology, College of Medicine, National Taiwan University, Taiwan, Attending Physician, Department of Medical Research, National Taiwan University Hospital, Taiwan

Scientific revolutions have often been driven by the discovery of mechanisms characterized by mathematical precision and uniformity. Newton's laws of motion laid the foundation for mechanical engineering, while the deciphering of the genetic code transformed molecular biology. In contrast, human motor control theory has largely remained descriptive, lacking precise mathematical frameworks for the fine-grained kinematic control seen in physics. The inherent complexity and variability of neuronal networks across individuals raise a fundamental question: does a precise motor control mechanism exist at the systems level? In this talk, we present recent findings demonstrating how the cerebellum employs frequency coding to regulate the fine kinematics of movement. We show that disruptions in this frequency-based control can manifest as tremors (too much rhythm) or ataxia (loss of rhythm), providing a unifying framework for understanding diverse movement disorders. Furthermore, we explore how cerebellar neurons achieve precise frequency computations through population coding, shedding light on the mechanisms of cross-individual consistency in motor control.

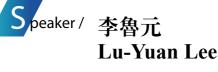


台灣藥理學會 3/22 (Sat.) 14:00-15:00 1 樓,第一教室



th 2025 The 39th Joint Annual Conference of Biomedical Science 生物醫學聯合學術年會





Current Position

Professor Emeritus, Department of Physiology, University of Kentucky

Education/Training

- 1969 BS, (Mechanical Engineering), National Taiwan University, Taiwan
- 1975 PhD, (Physiology and Biophysics), University of Mississippi Medical Center, USA
- 1978 OTHERS, (Pulmonary Physiology), University of California San Francisco, Cardiovascular Research Institute, USA

Professional and Research Experience

- 1981-Present Member of Regular and Special Study Sections and Review Panels, NIH
- 1992-1993 Karolinska Institute, Stockholm, Sweden
- 1994-1997 Director of Research, Department of Physiology, University of Kentucky (1992-Present Professor)

Awards and Honors

- 2002-2022 Fred Zechman Endowed Professor, University of Kentucky
- 2010 Elected Fellow, Biomedical Engineering Society (USA)
- 2016 Elected Fellow, American Physiological Society

Involvements of TRPV1 receptor in airway hypersensitivity induced by inflammation: from ion channel to patient

李魯元 Lu-Yuan Lee Professor Emeritus, Department of Physiology, University of Kentucky

Transient receptor potential vanilloid type 1 (TRPV1) receptor is a nonselective cation channel and a polymodal transducer; in the respiratory tract, it is expressed predominantly in nonmyelinated (C-fiber) sensory nerves. Stimulation of these TRPV1-expressing sensory endings in the lung can elicit reflex responses such as bronchoconstriction, cough, dyspnea and other characteristic symptoms of airway inflammatory diseases. Studies in our lab have demonstrated that a number of endogenous inflammatory mediators (e.g., eosinophil granular-derived cationic proteins, tumor necrosis factor-alpha, hydrogen ion, etc.) activated TRPV1 and/or up-regulated its sensitivity in airway sensory nerves. Furthermore, we have reported that allergen sensitizationinduced airway inflammation markedly enhanced the expression of TRPV1 and the sensitivity of pulmonary C-fiber afferents in an animal model of allergic asthma. More importantly, our recent studies have revealed a lower temperature threshold for activating TRPV1 expressed in pulmonary vagal sensory neurons than that previously reported in DRG neurons. An important implication of this finding is related to the fact that inflammatory reaction is known to lead to an increase in tissue temperature. In the patch-clamp studies of isolated rat vagal pulmonary sensory neurons, increasing temperature to ~39°C significantly elevated their baseline activity and sensitivity to various chemical stimuli, and an involvement of TRPV1 was primarily responsible. This hypothesis was then further tested in human studies; in patients with mild and stable asthma, a brief isocapnic hyperventilation (at ~40% of maximum voluntary ventilation for 4 min) of humidified warm air (HWA) triggered an immediate and pronounced increase in airway resistance (Raw) and coughs. In sharp contrast, the same challenge failed to evoke any significant change in Raw or cough in healthy individuals. Pretreatment with inhaled ipratropium bromide, a cholinergic antagonist, completely prevented the bronchoconstriction in asthmatic patients, but did not abolish their cough responses; these results suggested an involvement of airway sensory nerves and cholinergic reflex. Hyperventilation of humidified air at room temperature did not cause bronchoconstriction or cough in the same patients. Similarly, the same challenge with HWA also triggered vigorous cough responses and evoked throat irritation in patients with allergic rhinitis and laryngopharyngeal reflux. In summary, increasing airway temperature stimulated bronchopulmonary C-fiber afferents via an activation of TRPV1, which plays an important role in the manifestation of various common symptoms of airway hypersensitivity in patients with chronic inflammatory airway diseases. (Supported in part by NIH grants HL67379, ES026529, AI123832 and UL1TR001998)



中國生理學會 3/22 (Sat.) 10:50-12:00 1 樓[,]第二教室



h 2025 The 39th Joint Annual Conference of Biomedical Science 生物醫學聯合學術年會





Current Position

Co-Director of Bertarelli Rare Cancers Fund, HMS Co-Director of Paul F. Glenn Center for Biology of Aging Research at Harvard

Education/Training

2006 OTHERS, Massachusetts Institute of Technology
2002 PhD, University of Wisconsin - Madison
1996 BS, University of New Hampshire

Professional and Research Experience

2021-2024 Inaugural Director, Gender Equity for Faculty in Science, HMS2021-2024 Co-Chair, HMS Diversity Committee

Awards and Honors

- 2024 Elected to National Academy of Medicine
- 2023 Samsung Ho-Am Prize in Medicine
- 2022 Plenary Speaker in Opening session of 2022 Annual AACR conference

Investigating the role of metabolism in cancer, immunity and aging.

MARCIA HAIGIS

Co-Director of Bertarelli Rare Cancers Fund, HMS,Co-Director of Paul F. Glenn Center for Biology of Aging Research at Harvard

Metabolic rewiring is a hallmark of cancer and supports the increased biosynthetic and energetic requirements of cancer cells. Tumor metabolism may be regulated by tumor cell intrinsic mechanisms. In addition, the tumor microenvironment provides a unique niche that supports the metabolic reprogramming of the tumor but may be suppressive to cytotoxic T cells. Finally, the systemic metabolic fitness of an individual may affect on tumor cell mechanisms and incidence. Here, we will discuss the how aging and obesity impacts mechanisms of cancer and immunity.



免疫學會X細分學會合辦 3/22 (Sat.) 10:50-11:50 3 樓, 30 教室



2025 The 39th Joint Annual Conference of Biomedical Science





Current Position

Chair and Professor, Department of Plant Pathology and Microbiology, National Taiwan University, Taipei, Taiwan

Director - NTU College of Medicine Global Innovation Joint-Degree Program (GIP-TRIAD)

Education/Training

PhD, Cancer Cell Biology in the Department of Molecular Medicine, Cornell University, USA MS, Plant Virology in the Institute of Plant Pathology, National Taiwan University, Taiwan BS, Plant Pathology in the Department of Plant Pathology and Entomology, National Taiwan University, Taiwan

Professional and Research Experience

Post-doctoral Fellow, Department of Molecular Medicine, Cornell University (American Heart Association)

Visiting scholar, Weill Medical College of Cornell University, New York, NY, USA

Awards and Honors

2022 WW the Most Prestigious Medical Doctor Award (史懷哲風雲醫師獎), International Albert Schweitz er Foundation (史懷哲基金會).

The 16th and 18th National Innovation Award, Development of a small molecule enhancement for erythropoiesis, in the Academic Research Category. Dec. 6, 2019,「利用腸腦軸線概念開發 改善睡眠之植萃原料 Bugu-STM」2021

2016 The 6th Breast Cancer Outstanding Research Award, Breast cancer prevention foundation, Taipei, Taiwan

Exosome: The rising star in biomedicine

沈湯龍 Tang-Long Shen

Chair and Professor, Department of Plant Pathology and Microbiology, National Taiwan University, Taipei, Taiwan, Director - NTU College of Medicine Global Innovation Joint-Degree Program (GIP-TRIAD)

Exosomes are small extracellular vesicles with a phospholipid bilayer structure, measuring approximately 30-150 nm in diameter. They play a crucial role in intercellular communication, pathophysiological progression, waste disposal, regeneration, immune modulation etc. In recent years, exosomes have attracted increasing attention for their potential clinical applications, with 116 ongoing clinical trials exploring their use in biomarker discovery, therapeutics, drug delivery, and vaccine development. They have shown promise in the diagnosis and treatment of various diseases, including COVID-19, sepsis, osteoarthritis, and cancer. Compared to cell-based therapies, exosomes offer several advantages, such as high permeability, ease of storage, and non-proliferative properties, making them a valuable focus in biomedical research. Furthermore, exosomes have been widely studied in cancer (e.g., breast and colorectal cancer) and metabolic disorders (e.g., diabetes), where their biomarker potential enhances early disease detection. However, challenges such as heterogeneity, standardization of production, bioengineering modifications, and safety concerns still need to be addressed. Future research will focus on enhancing exosome-based drug delivery, expanding applications in personalized medicine, and developing scalable production methods to accelerate their clinical translation. Keywords: exosomes, biomarkers, drug delivery, intercellular communication, clinical applications



中華民國臨床生化學會 3/22 (Sat.) 10:50-12:00 3 樓,31 教室



h 2025 The 39th Joint Annual Conference of Biomedical Science 生物醫學聯合學術年會





Current Position

Professor of the Dept. of Anatomy, The Catholic University of Korea, Korea Director of the Catholic Institute for Applied Anatomy (CIAA), The Catholic University of Korea, Korea

Education/Training

- 2005 OTHERS, Medical School at Houston, University of Texas, TX, USA
- 2003 PhD, Graduate School, The Catholic University of Korea, Korea
- 1995 MD, College of Medicine, The Catholic University of Korea, Korea

Professional and Research Experience

- 2020-Present Secretary General, Organizing committee, Congress of International Federation of Associations of Anatomists (IFAA)
- 2018-2023 Division Chair, Science Program Committee, The 20th International Microscopy Conference (IMC20)
- 2018-2022 Director, Catholic Brain Bank, Seoul, Korea

Awards and Honors

- 2023 PRS Best Paper Award, American Society of Plastic Surgeons, USA
- 2022 Best Teacher Award, College of Medicine, The Catholic University of Korea
- 2013 Hangil Award for Excellent Research, The Korean Association of Anatomists

Enhancing Anatomical and Surgical Training Through Cadaveric Models: Recent Advances and Practical Insights

In-Beom Kim

Professor of the Dept. of Anatomy, The Catholic University of Korea, Korea, Director of the Catholic Institute for Applied Anatomy (CIAA), The Catholic University of Korea, Korea

Human cadavers have long been recognized as the gold standard for teaching anatomy to medical students and refining surgical techniques among clinical practitioners, particularly surgeons. Despite various challenges-including limited availability, potential decomposition, rigidity, and the risk of infection—cadaver-based training remains unrivaled in providing highfidelity simulations of operative environments. In recent decades, Korea has made notable progress in safeguarding human rights by implementing measures such as reducing working hours. Yet these reforms have also curtailed opportunities for hands-on clinical training, prompting the pursuit of more effective and efficient educational methods. Meanwhile, the proliferation of minimally invasive surgery (MIS)—encompassing laparoscopic and robot-assisted procedures—has resulted in fewer traditional open surgeries, thereby reducing surgical practice time for novices. To address these challenges, a variety of training modalities have emerged, including synthetic models, living animals, and virtual reality (VR) simulators. Nevertheless, human cadavers continue to offer the most realistic and comprehensive framework for developing surgical expertise. To optimize both specimen longevity and tissue fidelity, several embalming techniques have been introduced. In my talk, I will briefly introduce the diverse types of cadaveric specimens currently used for surgical skills training, detailing their properties, benefits, and limitations. I will also highlight our recent advances in creating "fresh cadavers with pulsation," which enhance realism and better support procedure training for vascular surgeons. Additionally, I will share insights from our latest initiatives, where medical students and residents practice essential clinical procedures—ranging from posterior nasal packing for epistaxis, tracheostomy, airway intubation, central venous catheterization, ascites paracentesis, bone marrow aspiration, pericardiocentesis, and spinal tap—using cadaveric models. A key focus will be placed on a straightforward, simple method for preparing cadavers specifically tailored to spinal tap training. By sharing our recent experiences with cadaver development and cadaverbased learning, I hope this talk will help you reflect on your identity as an anatomist and provide valuable insights into clinical medicine education in today's rapidly evolving technological environment.



中華民國解剖學學會 3/22 (Sat.) 10:50-12:00 3 樓,32 教室



2025 The 39th Joint Annual Conference of Biomedical Science 生物醫學聯合學術年會





Current Position

Director and Chair Professor, Department of Pharmacology, College of Medicine, National Cheng Kung University, Taiwan.

Education/Training

1993 PhD, Genetics, Michigan State University, USA.

Professional and Research Experience

2015-Present	Chair Professor, Department of Pharmacology & Institute of Basic Medical Sciences,
	College of Medicine, National Cheng Kung University.

- 2006-2015 Distinguished Professor, Department of Pharmacology, College of Medicine, National Cheng Kung University.
- 1999-2006 Professor, Department of Life Science, National Taiwan Normal University.

Awards and Honors

2024 國科會傑出特約研究員獎

Merit Research Fellow, National Science and Technology Council, Taiwan.

- 2023 第 19 屆永信李天德醫藥科技獎一卓越醫藥科技獎 Tien Te Lee Biomedical Foundation for Excellent Biomedical Award, Taiwan.
- 2022 第 66 屆教育部學術獎 The Ministry of Education's 66th Annual Academic Award, Taiwan.

PD-1 membrane presentation and stability: Mechanisms and therapeutics.

王憶卿 Wang Yi-Ching Director and Chair Professor, Department of Pharmacology, College of Medicine, National Cheng Kung University, Taiwan.

To date, immune checkpoint inhibitor therapies targeting the programmed cell death-1 (PD-1) pathway, including PD-1 or PD-L1 inhibitors, have emerged as frontline treatments in cancer therapy. Nevertheless, our current understanding of PD-1-mediated regulation in T cells is still limited, underscoring the urgent need to gain a deeper insight into how PD-1 contributes to T cell exhaustion and tumor immune escape. Our recent findings reveal novel mechanisms of intracellular trafficking and plasma membrane presentation of PD-1 mediated by Rab37 small GTPase to sustain T cell exhaustion, thereby leading to poor patient outcomes. In addition, post-translational modifications (PTMs) such as phosphorylation, ubiquitarian, and glycosylation of PD-1 influence its stability, membrane presentation, and T-cell activity within the immunosuppressive tumor microenvironment. By identifying key enzymes and effectors involved in these PTMs, we strive to shed light on the crosstalk between PTMs and PD-1 function, providing new insights into regulating immune responses in cancer. Moreover, we have developed therapeutic strategies targeting PD-1 PTMs using co-culture cell systems, transgenic mice, and syngeneic animal models. These strategies involve the use of neutralizing antibodies, inhibitors, or our in-house developed antagonists targeting key enzymes identified in the PTM process. Clinically, multiplex fluorescence immunohistochemistry of tumor specimens from cancer patients has shown a high enrichment of aberrant trafficking and PTM-modified PD-1 in CD8 exhausted T cells, correlating with tumor progression.



台灣生物化學及分子生物學學會 3/22 (Sat.) 11:00-12:00 3 樓,33 教室

th 2025 The 39th Joint Annual Conference of Biomedical Science 生物醫學聯合學術年會



peaker/ 高潘福 Pan-Fu Kao

Current Position

中山醫學大學 醫學系 核子醫學科 教授 中山醫學大學附設醫院 核子醫學科 主治醫師

Education/Training

2018 PhD, 中山醫學院 臨床醫學研究所
1994 MS, Johns Hopkins University, Radiation Health Sciences
1985 MD, 中山醫學大學 醫學系

Professional and Research Experience

2020-2025 副院長,中山醫學大學 醫學院
2018-2021 理事長,臺灣醫用迴旋加速器學會
2013-2025 教授,中山醫學大學 醫學系

Awards and Honors

2017 台灣醫學教育學會雜誌 最佳論文獎2016 原子能科技學術合作研究計劃 成果發表優良獎2012 中山醫學大學 教學特優教師

Theranostics: Current Concept and Future Perspectives in the Era of Personalized Medicine

高潘福 Pan-Fu Kao 中山醫學大學 醫學系 核子醫學科 教授 中山醫學大學附設醫院 核子醫學科 主治醫師

Theranostics(治療診斷學)是一種結合診斷與治療的個人化醫療技術,當今特別著重應用 於癌症治療。它利用放射性標記物進行分子影像診斷(如 SPECT/CT 或 PET/CT),再使用相 同的放射性核種藥物進行治療,最早應用放射性碘I-123和1-131進行甲狀腺癌診斷與治療, 以及[I-123]MIBG和[1-131]MIBG進行腎上腺髓質瘤診斷與治療。隨後Theranostics的觀念 拓展到以相同生物特性的製劑,標定上不同特性的放射核種,例如以[Ga-68]DOTATATE PET/ CT影像診斷和[Lu-177]DOTATATE治療神經內分泌腫瘤(Neuroendocrine Tumors),以及近 年蓬勃發展的以[Ga-68]PSMA PET/CT影像診斷和[Lu-177]PSMA治療去勢抗性的轉移性前列 腺癌(metastatic castration-resistant prostate cancer, mCRPC)。優勢包括提高診斷準確性、降 低副作用,以確保精準、高效的個人化療法。近年Theranostics更拓展到運用合併不同種類 的 PET 製劑的影像,例如合併[Ga-68]PSMA和[氟-18]去氧葡萄糖(FDG)確認腫瘤內部是 否有基因的異質性表現,再合併不同放射藥物治療與其他標靶或化學治療的可能性,以實現 個人化治療及改善預後。未來發展方向更涵蓋新型放射性藥物、AI影像分析及更多疾病應 用,如阿茲海默症的診斷與治療指引,使Theranostics成為個人化精準醫療的重要技術。



台灣分子生物影像學會 3/23 (Sun.) 10:50-11:40 2 樓, 20 教室





Current Position

JACBS

Associate Professor, Department of Pharmacology, National Taiwan University, Taiwan Faculty Member, Taiwan International Graduate Program in Chemical Biology and Molecular Biophysics (TIGP-CBMB), Academia Sinica

Education/Training

- 2015 PhD, Department of Pharmacology, National Taiwan University, Taipei, Taiwan
- 2010 MS, Department of Pharmacology, National Cheng Kung University, Tainan, Taiwan
- 2008 BS, School of Pharmacy, Taipei Medical University, Taipei, Taiwan

Professional and Research Experience

- 2019-2023 Assistant Professor, Department of Pharmacology, National Taiwan University, Taiwan
- 2016-2019 Postdoctoral Fellow, Cardiovascular Institute (CVI), Stanford University, USA
- 2015-2015 Postdoctoral Fellow, Department of Pharmacology, National Taiwan University, Taiwan

Awards and Honors

- 2024 FutureTech Award, National Science and Technology Council (NSTC), Taiwan
- 2024 NARLabs R&D Service Platform Achievement Award, National Applied Research Laboratories (NARLabs), Taiwan
- 2024 NTU SPARK Program, National Science and Technology Council (NSTC), Taiwan

Marijuana: A new risk factor for cardiovascular disease

魏子堂 Tzu-Tang Wei

Associate Professor, Department of Pharmacology, National Taiwan University, Taiwan, Faculty Member, Taiwan International Graduate Program in Chemical Biology and Molecular Biophysics (TIGP-CBMB), Academia Sinica

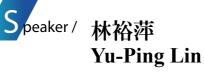
Marijuana is the most widely used illicit drug worldwide. Epidemiological studies indicate its increase in the risk of coronary artery disease. Adverse cardiovascular, cerebrovascular, and peripheral vascular effects have also been reported. In addition, three synthetic cannabis drugs have been approved by FDA for chemotherapy-induced nausea and vomiting. Synthetic cannabis drugs also show cardiovascular side effects. These results suggest that cardiovascular side effects exist in both recreational and medical use of marijuana. However, the underlying mechanisms remain poorly understood. We found that \triangle 9-tetrahydrocannabinol (\triangle 9-THC), the main mindaltering ingredient in marijuana, induced endothelial dysfunction in human endothelial cells and mice models via activation of cannabinoid CB1 receptor. Using high-throughput drug screening, we discovered genistein, a soybean isoflavone, was a new CB1 antagonist that attenuated marijuana-induced endothelial dysfunction and atherosclerosis, while preserving clinically useful effects such as sedation and analgesia. Cannabinoid CB1 receptor signaling is implicated in various diseases, including obesity, diabetes, cardiovascular disease, coronary artery disease, atherosclerosis, liver cirrhosis, and cancers. Although selective CB1 antagonists like rimonabant (Acomplia®) demonstrated therapeutic potential, their severe psychiatric side effects led to market withdrawal. Our recent work focuses on developing peripherally restricted CB1 antagonists to circumvent these side effects. In this presentation, I will report our latest findings on the role of CB1 receptor in cardiovascular disease. In addition, I will introduce our advancements in developing next-generation CB1 antagonists.



台灣毒物學學會 3/23 (Sun.) 09:00-09:45 2 樓,29 教室

th 2025 The 39th Joint Annual Conference of Biomedical Science 生物醫學聯合學術年會





Current Position

Assistant Professor of Department of Biotechnology and Bioindustry Sciences

Education/Training

2011 PhD, Department of Basic Science of National Cheng Kung University

Professional and Research Experience

2020-2024 Research Scientist of Oxford University2013-2019 Research Fellow of NIEHS

Ca²⁺ release-activated Ca²⁺ (CRAC) channels as a potential new therapy for treating environmental allergens-house dust mite

林裕萍 Yu-Ping Lin Assistant Professor of Department of Biotechnology and Bioindustry Sciences

House dust mite (HDM) allergens are major triggers of asthma worldwide. This study shows how HDM allergens, particularly the Der p3 protease activated by Der p1, stimulate protease-activated receptors, activating store-operated Ca²⁺ release-activated Ca²⁺ (CRAC) channels. These channels, regulated by STIM-Orai interactions, drive inflammatory responses through Ca²⁺-dependent transcription factors. Recent studies demonstrate that T cell-specific Orai1 deletion or pharmacological CRAC channel inhibition significantly reduces HDM-induced airway inflammation in mouse models. Combined partial inhibition of Der p3 and CRAC channels shows enhanced therapeutic efficacy compared to single-target approaches. The Der p3-PAR-CRAC channel axis represents a promising therapeutic target for allergen-induced asthma, with partial inhibition strategies potentially offering improved safety profiles while maintaining therapeutic efficacy.



台灣毒物學學會 3/23 (Sun.) 09:45-10:30 2 樓, 29 教室

th 2025 The 39th Joint Annual Conference of Biomedical Science 生物醫學聯合學術年會





Current Position

Professor, University of Pittsburgh School of Medicine, Department of Cell Biology & Clinical and Translational Science Institute University of Pittsburgh, Pittsburgh, PA Joint Appointment, Pittsburgh PA, USA

Past President, American Society for Cell Biology

Education/Training

- 1970 BS, University of Illinois, Chicago, IL
- 1973 MS, Texas Southern University, Houston, TX
- 1980 PhD, School of Medicine, University of Iowa, Iowa City, IA

Professional and Research Experience

- 1999-Present Professor, Depart. of Cell Biology, University of Pittsburgh, School of Medicine, Pittsburgh, PA.
- 1988-1999 Associate Professor, Department of Neurobiology, Anatomy and Cell Science, School of Medicine, Pittsburgh, PA.
- 1982-1988 Assistant Professor, School of Medicine, Department of Neurobiology, Anatomy and Cell Science, University of Pittsburgh, School of Medicine, Pittsburgh, PA.

Awards and Honors

- 2024 Elected President of the American Society for Cell Biology
- 2020 Awarded the Training and Experimentation in Computational Biology (TECBio) Outstanding Mentor of the Year Award, University of Pittsburgh, Department of Computational and Systems Biology, Computational Biology REU Program
- 2018 Inducted as a Lifetime Fellow of the American Society for Cell Biology

Visualizing Connexin Dynamics: Imaging-Based Insights into Cellular Communication and Trafficking

Sandra Murray

Professor, University of Pittsburgh School of Medicine, Department of Cell Biology & Clinical and Translational Science Institute University of Pittsburgh, Pittsburgh, PA Joint Appointment, Pittsburgh PA, USA, Past President, American Society for Cell Biology

Cell-cell communication is essential for maintaining tissue homeostasis, and gap junction channels play a pivotal role in facilitating this process by enabling the direct transfer of ions, metabolites, and signaling molecules between adjacent cells. Gap junction channels are composed of transmembrane proteins called connexins with connexin 43 (Cx43) being the most abundant isoform. Advances in imaging technologies have revolutionized our understanding of connexin dynamics, by shedding light on the complex processes governing gap junction channel assembly, internalization, and trafficking. In this talk, I will highlight how cutting-edge imaging approaches, including live-cell fluorescence microscopy, super-resolution techniques, and immunogold cytochemical transmission electron microscopy, have unveiled new insights into the life cycle of connexins. I will discuss the molecular mechanisms driving gap junction plaque internalization into annular gap junction vesicles, and their subsequent fate through degradation or recycling pathways. Furthermore, I will explore how connexin trafficking integrates with cellular organelles such as lysosomes and mitochondria, with implications for cellular signaling and energy homeostasis. By visualizing these dynamic processes, we have uncovered how connexins contribute to cellular communication in normal physiology and disease states. Our findings open new avenues for therapeutic interventions for developing novel strategies to modulate gap junctional communication in cancer, cardiovascular diseases, and metabolic disorders. This talk will highlight the power and beauty of imaging as a tool to understand the choreography of cellular communication and its potential in future research directions.



中華民國細胞及分子生物學學會 3/23 (Sun.) 09:30-10:30 3 樓, 30 教室

JACBS Joint Annual Conference of Biomedical Science

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th 2025 The 39th Joint Annual Conference of Biomedical Science 會





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th 2025 The 39th Joint Annual Conference of Biomedical Science 生物醫學聯合學術年會





Current Position

Professor, Department of Life Sciences, National Taiwan University

Education/Training

2017 Ph. D., Department of Biology, University of Houston2002 BS, Department of Zoology, National Taiwan University

Professional and Research Experience

2008-2012 Postdoc, Biology Department, John's Hopkins University

Awards and Honors

2018 傑出人才基金會年輕學者創新獎

2017 吳大猷先生紀念獎

Environmental Light modulates gut microbiota, social memory and circadian clock through intrinsically photosensitive retinal ganglion cells

陳示國 Shih-Kuo Chen Professor, Department of Life Sciences, National Taiwan University

In mammals, the retina at the back of the eye contains three types of photoreceptors. The classic photoreceptors, rod and cone cells, are essential for pattern vision, detecting light through visual opsins and relying on retinal ganglion cells to convey information to the visual cortex. However, a third type of photoreceptor, the intrinsically photosensitive retinal ganglion cells (ipRGCs), project to various nuclei in the hypothalamus and thalamus. These ipRGCs express the photopigment melanopsin, which has a peak absorption spectrum near 478 nm, enabling them to control non-image-forming functions such as circadian photoentrainment and the pupil light reflex. In our study, we discovered that light exposure can reduce social memory formation in mice. Through ipRGCs, light can regulate social memory by activating GABAergic neurons in the peri-supraoptic nucleus (pSON) and inhibiting oxytocin neurons in the supraoptic nucleus (SON). Furthermore, ipRGCs could influence gut microbiota oscillation and hair regeneration through sympathetic nerves, potentially mediated by the suprachiasmatic nucleus (SCN), the central oscillator for the circadian clock. Aberrant light dark cycle such as light exposure at night will impair gut microbe composition and dampen their daily oscillation. In summary, light information in mammals can modulate numerous physiological functions through a direct ipRGC-to-hypothalamus circuit, bypassing the visual cortex. This provides a neural pathway for mammals to respond to external light without "seeing" the light.



中國生理學會 3/22 (Sat.) 14:15-14:45 1 樓,第二教室





Current Position

Professor, Institute of Neuroscience, National Yang Ming Chiao Tung University, Taiwan

Education/Training

2006 PhD, Duke University2000 MD, National Taiwan University

Professional and Research Experience

2009-2017 Investigator, National Institutes on Aging, NIH, USA2017-2025 Professor, National Yang Ming Chiao Tung University

A common neural mechanism for selective attention across sensory modalities in the basal forebrain

林士傑 Shih-Chieh Lin Professor, Institute of Neuroscience, National Yang Ming Chiao Tung University, Taiwan

Selective attention enhances the processing of behaviorally relevant sensory inputs while filtering out distractions, leading to improved perception and behavioral responses specific to the attended modality. Despite the modality-specific manifestations of selective attention, here we identify a modality-common attention signal in the basal forebrain (BF), where attention signals from different sensory modalities converge onto the same population of noncholinergic BF neurons. Using a novel crossmodal selective attention task, in which auditory and visual stimuli were presented concurrently, rats were trained to rapidly switch attention between sensory modalities. Behavioral performance and BF activity were dictated solely by the currently attended modality, with minimal influence from perceptually salient inputs in the unattended modality. Remarkably, the same BF neurons exhibited highly similar responses to attended targets regardless of sensory modality, providing a modality-common signal for selective attention. This BF activity closely tracked behavioral performance on a trial-by-trial basis, including during task-related rapid attentional shifts and spontaneous, self-initiated switches. Furthermore, BF response amplitudes and latencies reliably decoded attentional engagement and the attended modality, respectively, in single trials. These findings suggest that selective attention across sensory modalities converges onto a shared mechanism in the BF, underscoring its role as a subcortical hub for integrating attention and promoting adaptive behavior.



中國生理學會 3/22 (Sat.) 14:45-15:15 1 樓,第二教室





Current Position

Professor, Kaohsiung Medical University, Kaohsiung, Taiwan

Education/Training

- PhD, Institute of Medicine, College of Medicine, Kaohsiung Medical University 1995
- MS, Institute of Medicine, College of Medicine, Kaohsiung Medical University 1990
- 1987 BS, School of Pharmacy, Kaohsiung Medical College

Professional and Research Experience

2005-Present	Professor, Department of Pharmacology, Kaohsiung Medical University
2018-2024	Prof. & Director, Graduate Institute of Medicine, Kaohsiung Medical University
2006-2012	Prof. & Chief, Department of Pharmacology, Kaohsiung Medical University

Awards and Honors

- 2002 Taiwan Pharmacological Society Young Investigator Award
- 2005 The 2005 Neuroplasticity Symposium and the 2nd TMU Neuroscience Symposium ---The **Distinguished Neuroscience Award**
- 2014 Associate Editor: The Kaohsiung Journal of Medical Sciences (KJMS)

Cornel iridoid glycosides improve peripheral nerve injury-induced neuropathic pain and associated neurogenic inflammation

吳炳男 Bin-Nan Wu Professor, Kaohsiung Medical University, Kaohsiung, Taiwan

Neuropathic pain remains the most frequent cause of suffering and disability throughout the world. Hyperalgesia and allodynia associated with neuropathic pain are the hallmarks of peripheral nerve injury. Since currently available treatments for neuropathic pain remain inadequate, it is imperative to continue the search for novel targets and improved therapies. We aimed to examine the inflammatory factors and pain-related ion channels in streptozotocin/ nicotinamide (STZ/NA)-induced rats and diabetic db/db mice and to explore the possible mechanisms of cornel iridoid glycosides (CIG) on peripheral nerve injury. Materials and Methods: Animals' blood glucose levels ≥200 mg/dl were used as diabetic models. STZ/NA-induced SD rats and db/db mice were performed to induce hyperalgesia and allodynia. SD rats were randomly divided into control, STZ/NA, control+CIG, and STZ/NA+CIG groups. Diabetic db/ db mice were separated into sham, sham+CIG, chronic constriction injury (CCI), and CCI+CIG groups. Intraperitoneal injection of the vehicle or drugs was performed once daily for 2 (rats) or 3 weeks (mice). Animals' body weight and blood glucose levels during the experimental period were measured. Next, we sacrificed the animal, and the sciatic nerve, dorsal root ganglia (DRG), and spinal cord were removed. Results and Discussion: Administration of CIG could effectively alleviate hyperalgesia and allodina in SD rats and db/db mice. CIG also reduced pain-associated channel protein CaV3.2 and calcitonin gene-related peptide (CGRP) in the surficial spinal dorsal horn of SD rats. CIG inhibited oxidative stress and NF-kB activation and decreased the levels of mRNA and protein of proinflammatory factors IL-1 β and TNF-alpha. In the group of db/db mice combined CCI, immunofluorescence staining results demonstrated that p-NF-KB increased in neurons and astrocytes, Cx43 increased in astrocytes, and P2X3R increased in neurons. Besides, the ATP content in the spinal cord was also significantly increased. All the effects were improved in the CCI + CIG group. Those data indicated that CIG attenuated Cx43-mediated ATP release, which bound to P2X3R and contributed to hindering the ERK/p38NF-kB activation. Conclusion: Those results suggested that CIG improved painful diabetic neuropathy (PDN)-mediated pain behaviors by inhibiting oxidative stress-provoked inflammation and pain-related channel proteins in the spinal cord to improve neuropathic pain. Our findings demonstrated that CIG might be a potential candidate for treating PDN. Keywords: Cornel iridoid glycosides, chronic constriction injury, neuropathic pain, neuroinflammation



3/22 (Sat.) 15:15-15:45 1樓,第二教室



Current Position

JACBS

Distinguished Research Fellow, Institute of Biomedical Sciences, Academia Sinica

Education/Training

1997 PhD, University College London

Professional and Research Experience

2023-Present Distinguished Research Feoolw, Institute of Biomedical Sciences, Academia Sinica

Awards and Honors

2023 NSTC Outstanding Research Award2017 NOST Outstanding Research Award

Roles of acid-sensing ion channels in sngception

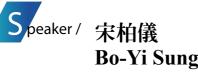
陳志成 Chih-Cheng Chen Distinguished Research Fellow, Institute of Biomedical Sciences, Academia Sinica

The perception of acid-sensation can be regarded as one of the most mysterious somatosensory functions. Traditionally, tissue acidosis which occurs in ischemia, inflammation, fatiguing exercise, etc., is a potent factor for activating proton-sensing ion channels/receptors to trigger pain, as has been demonstrated in humans and animal models. The location of the proton-sensing ion channels however, is more paradoxical being found on a wide range of somatosensory neurons. These, include not only nociceptors, but also pruriceptors, and nonnociceptive mechanoreceptors (e.g., proprioceptors). Thus, acidosis seems not only to be involved in nociception, but also in pruriception, proprioception, and anti-nociceptive signaling. For instance, the acid-sensing ion channel 3 (ASIC3) is arguably the most acid-sensitive of ion channels in somatosensory neurons and is involved in perception of acid-induced chronic pain in experimental animal models. Yet, intriguingly, ASIC3 is also expressed in proprioceptors where it behaves as a mechanically sensitive ion channel involved in tether-mode mechanotransduction. In addition, a recent study showed another acid-sensitive ion channel, ASIC1a, can mediate anti-nociceptive effects in dextrose prolotherapy. Therefore, the role of acid signaling in nonnociceptive somatosensory neurons is of great interest for understanding the neurobiology of pain associated with tissue acidosis, and a potential therapeutic target. To address the promiscuous nature of acid-sensation, we have coined the term "sngception (sng- ception)" for this specific somatosensory function, to distinguish it from the nociceptor neuron-specific sensation of painful stimuli (nociception). 'Sng' (pronounced as sa-ng) is derived from a linguistic phenomenon where both "sour taste" and muscle soreness are encoded in the same word in the Taiwanese language. In Chinese, such acid-like discomfort is often described as sng or sng-pain, again using the sng Taiwanese word that represents the state of feeling sore. In the pain clinic, soreness (or sng) sensation is seen as a distinct and characteristic sensory phenotype of various acute and chronic pain syndromes (e.g., delayed onset muscle soreness or DOMS, fibromyalgia, and radicular pain). It is also a sign of successful analgesia for acupuncture and many physical therapies. Here we show evidence that sng and pain can be segregated and distinguished separately in humans and mice. We also show in mouse models how sngception is transmitted and contributes to chronic hypersensitivity.



中國生理學會 3/22 (Sat.) 15:45-16:15 1 樓,第二教室





Current Position

Assistant professor, Department of Microbiology and Immunology, National Defense Medical Center

Education/Training

- 2010 MD, Department of Medicine, National Defense Medical Center, Taipei, Taiwan
- 2019 PhD, Pathobiology program, Johns Hopkins University School of Medicine, Baltimore, MD, USA

Professional and Research Experience

- 2022-Present Assistant professor, Department of Microbiology and Immunology, National Defense Medical Center, Taiwan
- 2020-2022 Assistant professor, Department of Biomedical Engineering, National Defense Medical Center, Taiwan
- 2017-2020 Instructor, Health Service Training Center, National Defense Medical Center, Taiwan

Awards and Honors

- 2018 Co-PI of Einstein Program, Ministry of Science & Technology (MOST), Taiwan
- 2016 Pathology Young Investigator Award, Johns Hopkins University, USA

Selected Publication

- Bo-Yi Sung, Yi-Hsin Lin, Qiongman Kong, Pali D. Shah, Joan Glick Bieler, Scott Palmer, Kent Weinhold, Hong-Ru Chang, Hailiang Huang, Robin K. Avery, Jonathan P. Schneck*, Yen-Ling Chiu* Wnt Activation-Induced PRMT1 Epigenetically Controls Memory T cell Polyfunctionality. J Clin Invest. 2022
- 2. Yen-Ling Chiu, Chung-Hao Lin, Bo-Yi Sung, Yi-Fang Chuang, Jonathan P. Schneck, Florian Kern, Graham Pawelec & George C. Wang. Cytotoxic polyfunctionality maturation of cytomegalovirus-pp65-specific CD4 + and CD8 + T-cell responses in older adults positively correlates with response size. Sci Rep. 2016.

A Systematic Approach to Spectral Cytometry Panel Design Incorporating Intracellular Staining with SCOPE (Spectral Cytometry Optimization and Panel Expansion)

宋柏儀 Bo-Yi Sung

Assistant professor, Department of Microbiology and Immunology, National Defense Medical Center

Spectral cytometry is a powerful tool, yet researchers often struggle with effectively designing high-dimensional panels. To tackle this challenge, we developed SCOPE (Spectral Cytometry Optimization and Panel Expansion)—a comprehensive strategy that enables users from any institute equipped with a spectral cytometer to quickly, conveniently, and flexibly design optimized panels, including intracellular staining. This approach overcomes the long-standing limitation where high-dimensional flow cytometry incorporating intracellular markers was primarily achievable only through CyTOF. I will first demonstrate how inverse matrix multiplication can be used to manually compute conventional flow cytometry compensation. I will then explain the data structure of spectral cytometry and how unmixing reconstructs the original signals. Key principles of panel design will be discussed, including fluorescence brightness, instrument configuration, antigen expression patterns, and antibody availability. By leveraging database searches, we selected 56 candidate fluorescent dyes. Single-stain experiments were conducted to calculate stain indices (SI) under normal and fix/perm-treated conditions. Unmixing analysis on the Thermo BigFoot spectral cytometer (U12V12B7Y12R5) at National Defense Medical Center revealed that over 44 dyes could be effectively separated. Using this information, we successfully designed a 35-color panel to comprehensively analyze tumorinfiltrating lymphocytes (TILs) populations in lung cancer. This study provides a systematic and scalable framework for spectral panel design, empowering researchers to maximize the potential of spectral cytometry for high-dimensional immunophenotyping, including the analysis of cytokines, transcription factors, and other intracellular proteins.



中華民國免疫學會 3/22 (Sat.) 13:30-14:00 1 樓,可勝廳





Current Position

Assistant Professor, Department of Life Science, National Taiwan University

Education/Training

- 2011 PhD, Graduate Institute of Immunology, National Taiwan University College of Medicine, Taiwan
- 2004 MS, Graduate Institute of Immunology, National Taiwan University College of Medicine, Taiwan
- 2001 BS, Department of Life Sciences, National Cheng Kung University, Taiwan

Professional and Research Experience

- 2023-2024 Instructor in Research, Department of Microbiology, Immunology & Molecular Genetics, UT Health San Antonio, USA
- 2018-2023 Postdoctoral Research Fellow, Department of Microbiology, Immunology & Molecular Genetics, UT Health San Antonio, USA
- 2014-2018 Postdoctoral Scholar, Department of Veterinary and Biomedical Sciences, The Pennsylvania State University, USA

Awards and Honors

- 2017 American Association of Immunologists (AAI) Trainee Abstract Awards, AAI annual meeting, Washington DC, USA
- 2013 The Postdoctoral Research Abroad Program Awards, Ministry of Science and Technology, Taiwan

A Novel Role for CCR10+ iNKT Cells in Skin Immunity: Regulating Iron Levels and Hair Follicle Morphogenesis in Early Life

王偉蓓 Wei-Bei Wang Assistant Professor, Department of Life Science, National Taiwan University

Invariant natural killer T (iNKT) cells are a unique subset of innate-like T cells that have diverse functions in the immune system. iNKT cells express restricted T cell receptors (TCR) to recognize self and foreign lipid antigens. Distinct iNKT subsets can quickly produce numerous cytokines to regulate immune responses in microbial infection, allergic disease, autoimmune disease, and cancer. These subsets have unique transcription factor profiles that determine their cytokineproducing abilities. However, the mechanisms that direct the tissue localization preference of different iNKT cell subsets are not well understood. Using CCR10 reporter mice, we found that the skin-homing chemokine receptor CCR10 is highly upregulated in iNKT cells during their thymic development stages in early life. Analysis of cytokine production in stimulated skin iNKT cells demonstrated that CCR10+ iNKT cells are unique iNKT2/1 subsets. In postnatal mice, iNKT cells are essential for immune equilibrium and skin morphogenesis. Further investigation revealed that skin-resident iNKT cells produce transferrin (Tf), a protein involved in iron metabolism. This finding suggested that iNKT cells might regulate iron levels in the skin, potentially influencing developmental processes. To explore this possibility, we conducted adoptive transfer experiments, introducing iNKT cells into hypotransferrinemic (hpx) mice that were deficient in transferrin. We observed a significant improvement in hair follicle development in these mice, with iNKT cells increasing iron levels in hair follicle stem cell progenitors. This process is crucial for hair follicle formation during early postnatal life. Overall, these studies enrich our understanding of the physiological roles played by iNKT cells in early skin development and may pave the way for novel therapeutic approaches targeting iNKT cells to promote skin health and regeneration.



中華民國免疫學會 3/22 (Sat.) 14:00-14:30 1 樓,可勝廳





Current Position

Associate Professor, Dept. of Microbiology & Immunology, Chang Gung University, Taiwan.

Education/Training

- 2009 PhD, Graduate Institute of Life Sciences, National Defense Medical Center, Taiwan.
- 2001 MS, Dept. of Public Health, National Yang-Ming University, Taiwan.
- 1999 BS, Dept. of Medical Biotechnology and Laboratory Science, Chang Gung University, Taiwan.

Professional and Research Experience

- 2016-2021 Assistant Professor, Dept. of Microbiology & Immunology, Chang Gung University, Taiwan.
- 2014-2016 Assistant Research Fellow, Molecular Medicine Research Center, Chang Gung University, Taiwan.
- 2010-2014 Postdoctoral Fellow, Immunology Research Center, National Health Research Institutes, Taiwan.

Selected Publication

- Wang LJ, Tsai CS, Chou WJ, Kuo HC, Huang YH, Lee SY, Dai HY, Yang CY, Li CJ, Yeh YT. Wang. Add-On Bifidobacterium Bifidum Supplement in Children with Attention-Deficit/ Hyperactivity Disorder: A 12-Week Randomized Double-Blind Placebo-Controlled Clinical Trial. Nutrients. 2024, 6(14):2260 (IF= 4.8, 18/114 in NUTRITION & DIETETICS)
- Chan XY, Chang KP, Yang CY, Liu CR, Hung CM, Huang CC, Liu HP, Wu CC. Upregulation of ENAH by a PI3K/AKT/β-catenin cascade promotes oral cancer cell migration and growth via an ITGB5/Src axis. Cell Mol Biol Lett 2024, 29:136 (IF= 9.2, 27/313 in BIOCHEMISTRY & MOLECULAR BIOLOGY)
- Chen KR*, Yang CY*, Shu SG*, Lo YC, Lee KW, Wang LC, Chen JB, Shih MC, Chang HC, Hsiao YJ, Wu CL, Tan TH, Ling P. Endosomes serve as signaling platforms for RIG-I ubiquitination and activation. Science Advances 2024, 10:45 (First author), (*These authors contributed equally to this study) (IF= 11.7, 11/134 in MULTIDISCIPLINARY SCIENCES)
- Lee SY, Li SC, Yang CY, Kuo HC, Chou WJ, Wang LJ. Gut leakage markers and cognitive functions in patients with Attention-Deficit/Hyperactivity Disorder. Children, 2023, 10:513, (IF= 2.835, 59/130 in PEDIATRICS)

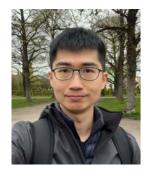
Functional roles of dual-specificity phosphatase 12 in T-cell survival

楊佳郁 Chia-Yu Yang Associate Professor, Dept. of Microbiology & Immunology, Chang Gung University, Taiwan.

Dual-specificity phosphatases (DUSPs) are a family of protein phosphatases, which dephosphorylate threonine and tyrosine residues on their substrates. DUSP12 is an atypical dual-specificity phosphatase that contains a phosphatase domain at the N-terminus and a zinc-binding domain at the C-terminus. DUSP12 mediates the regulation of Toll-like receptor signaling, cell cycle, hepatocyte metabolism, cardiac hypertrophy, and fibrosis. Moreover, a nonsynonymous mutation of DUSP12 has been identified in 2 patients with the T-cell-mediated autoimmune diseases. T cells play an important role in the adaptive immune response, and wellcontrolled T-cell signaling is essential for proper immune responses. However, the functional roles and molecular mechanisms of DUSP12 and its substrates/regulators in T cells and immune responses remain unclear. To study the DUSP12 functions in T cells, we have established T-cellspecific DUSP12 conditional knockout (cKO) mice by breeding DUSP12 floxed mice with CD4-Cre transgenic mice. Our data showed that DUSP12 cKO mice had severe T-cell lymphopenia in CD4+, CD8+, and regulatory T cells. Furthermore, the proportion of Annexin V-positive CD4+ and CD8+T cells was significantly increased in DUSP12 cKO mice compared with wild-type mice. These findings suggest that DUSP12 plays an important role in controlling T-cell survival. Using DUSP12 co-immunoprecipitation and liquid chromatography-mass spectrometry experiment, we have identified multiple potential DUSP12-binding proteins in T cells, which may regulate T-cell survival. We will further characterize the molecular mechanisms of DUSP12 in T-cell survival in this study.



中華民國免疫學會 3/22 (Sat.) 14:30-15:00 1 樓,可勝廳





Current Position

Assistant Professor, Department of Pharmacology, College of Medicine, National Cheng Kung University

Education/Training

- 2016 PhD, Department of Biomedical Engineering and Environmental Sciences, National Tsing Hua University
- 2011 BS, Department of Biomedical Engineering and Environmental Sciences, National Tsing Hua University

Professional and Research Experience

2019-2024 Postdoc, Institute of Biomedical Engineering & Nanomedicine, National Health Research Institute

Awards and Honors

- 2024 Third Place Award, IFMBE Young Investigator Competition, ICBHI 2024
- 2021 Summa Cum Laude Merit Award, ISMRM 2021

Selected Publication

Huang, S. M., Cho, K. H., Chang, K., Huang, P. H.*, and Kuo, L. W.* (2024) Altered thalamocortical tract trajectory growth with undisrupted thalamic parcellation pattern in human lissencephaly brain at mid-gestational stage. Neurobiology of Disease, p. 106577. doi: 10.1016/j.nbd.2024.106577

Diffusion MRI fiber-tractography of the developing human brain

黃聖閔 Sheng-Min Huang Assistant Professor, Department of Pharmacology, College of Medicine, National Cheng Kung University

Proper topographically organized neural connections are essential during brain development. We aim to disclose the developmental progress of brain connections by using diffusion MRI fiber-tractography. Specifically, the connections between the thalamus and the cerebral cortex are of importance in thalamus function. Thalamocortical (TC) fiber growth begins during the embryonic period and completes by the third trimester of gestation, so that human neonates at birth have a thalamus with a near-facsimile of adult functional parcellation. By using diffusion MRI fiber-tractography analysis of long-term formalin-fixed postmortem fetal brain, the thalamocortical tracts were reconstructed and the topological patterns of thalamic subregions were characterized. We found similar topological patterns of thalamic subregions and of internal capsule parcellated by TC fibers. On the contrary, the lissencephaly fetal brain showed less organized TC fibers and optic radiations, and much less cortical plate invasion by TC fibers. These results show the feasibility of diffusion MRI fiber tractography in postmortem long-term formalin-fixed fetal brains to disclose the developmental progress of TC tracts. Moreover, we further extend the fiber-tractography analyzing approach to investigate the major cerebellar fibers in developing human brain, trying to characterize the developing progress of cerebellar peduncles in different neonatal stages. Preliminary result reveals the developing changes of along tract diffusion MRI metrics, highlighting the capability of diffusion MRI in exploring the cerebellar connectome in developing human brain.



台灣分子生物影像學會 3/22 (Sat.) 13:00-14:00 2 樓, 20 教室





Current Position

Professor, Graduate Institute of Biomedical Engineering, National Taiwan University of Science and Technolog

Education/Training

2009 PhD, Department of Electrical Engineering in National Taiwan University

Professional and Research Experience

2009-2010 Postdoctoral Researcher, NTU Research Center for Medical Excellence – Division of Genomic Medicine

Awards and Honors

- 2017 Dr. Ta-You Wu Memorial Award
- 2018 Taiwan Innovation Award
- 2023 Taiwan Innovation Award

Selected Publication

- Ai-Ho Liao, Yu-Chen Chen, Chia-Yu Chen, Shun Cheng Chang, Ho-Chiao Chuang, Dao-Lung Lin, Chien-Ping Chiang, Chih-Hung Wang, Jehng-Kang Wang. Mechanisms of ultrasoundmicrobubble cavitation for inducing the permeability of human skin. Journal of Controlled Release, 349:388-400, 2022. (SCI) IF: 10.5, 12/354. (PHARMACOLOGY & PHARMACY)
- 2. Ai-Ho Liao*, Ying-Jui Lu, Yi-Chun Lin, Hang-Kang Chen, Huey-Kang Sytwu, Chih-Hung Wang*, "Effectiveness of a Layer-by-Layer Microbubbles-Based Delivery System for Applying Minoxidil to Enhance Hair Growth" Theranostics, 6(6), 817-827, 2016. (SCI) IF:12.4, 8/189. (MEDICINE, RESEARCH & EXPERIMENTAL)

Microbubble-assisted ultrasound for inner ear drug delivery

廖愛禾 Ai-Ho Liao Professor, Graduate Institute of Biomedical Engineering, National Taiwan University of Science and Technolog

Ultrasound-microbubbles (USMBs) can be applied for imaging, drug delivery, gene transfection, cancer therapy and blood-brain barrier opening. The inner ear is a highly specialized sense organ and lacks the capacity to regenerate hair cells which can be easily damaged by excessive stimulation of noise, ototoxic drugs and the effects of aging. In previous studies, USMBs has been demonstrated to enhance the permeation of round window membrane and local delivery of drug into the inner ear without hearing damage. In this presentation, we introduce the technique of USMBs in the inner ear drug delivery and illustrate the new challenge and insight. The cochlear blood-labyrinth barrier (BLB) and the blood-brain barrier (BBB) have many similarities and have blocking effects on many large and small molecules. However, some studies have confirmed that the cochlear blood-labyrinth barrier and the blood-brain barrier exist different mechanisms in drug delivery. When sudden deafness occurs due to damage to the inner ear, the blood flow in the tissue is reduced, causing ischemic damage and insufficient glucose and oxygen supply (Oxygen Glucose Deprivation, OGD). Hyperbaric oxygen therapy (HBOT) has been suggested as a viable option for treatment of sudden sensorineural hearing loss as it improves vascular dysfunction. However, the most common complication during HBOT is middle ear barotrauma, which can lead to permanent hearing loss and vertigo. Therefore, we prepared drug-coated or drug-loaded oxygenated albumin microbubbles (Met-OMB or MetOMB), and combined with ultrasound to improve the delivery efficiency of drug and oxygen through the round window membrane or cochlear blood-labyrinthine barrier, and treat inner ear damage. Moreover, the present study firstly explores the feasibility of combining siRNA-coated lysozyme-shelled microbubbles (LyzMBs) with ultrasound (US) to increase the knockdown effect of target genes on the cochlea as well as reducing the degradation of siRNA. The obtained results show that this approach can inhibit the expression of disease-causing gene and the generation of ROS in cells, and effectively reduce the ototoxicity induced by cisplatin.



台灣分子生物影像學會 3/22 (Sat.) 14:00-15:00 2 樓, 20 教室



Current Position

JACBS

Professor. Dept. of Biomedical Imaging and Radiological Sciences, National Yang Ming Chiao Tung University, Taiwan

Education/Training

2003 PhD, Pathology and Laboratory Medicine, School of Medicine, University of Rochester, NY, USA

Professional and Research Experience

2014-Present Professor, Dept. of Biomedical Imaging and Radiological Sciences, National Yang Ming Chiao Tung University, Taiwan

Awards and Honors

2024 2024 生物醫學年會之台灣分子生物影像學會傑出論文獎

- 國科會未來科技獎 2022
- 2022 JMBE 年度傑出論文獎

Integrating ultrahigh-brightness polymer dots and stereo NIR-II imaging to assess the angiogenesis with stemness of head and neck cancer and potent anti-angiogenic agents in vivo

李易展 Yi-Jang Lee

Professor. Dept. of Biomedical Imaging and Radiological Sciences, National Yang Ming Chiao Tung University, Taiwan

Head and neck cancer (HNC) is often diagnosed at an advanced stage with poor differentiation and prognosis. Late-stage tumors exhibit reduced proliferative fractions and increased cell loss, yet the remnant living cells remain poorly characterized. In vivo optical imaging of FaDu tumor-bearing mice revealed reduced tumor activity at advanced stages. However, remnant living FaDu cells isolated from these tumors exhibited accelerated growth, enhanced chemo-radioresistance, and antioxidant properties compared to pre-implanted cells. These cells demonstrated increased migration, invasion, and upregulation of epithelial-mesenchymal transition (EMT) markers. Moreover, they displayed cancer stem cells (CSC) associated characteristics, including high tumorigenicity, reduced side population, increased spheroid formation, and upregulation of TIC-associated biomarkers. Despite arsenic trioxide (ATO) treatment suppressing TIC-related biomarkers, Nrf2 was strongly induced, sustaining low oxidative stress. This suggests that the antioxidant potency of late-stage tumors could serve as a therapeutic target for advanced HNC. Given the critical role of angiogenesis in tumor progression and therapy resistance, we employed an ultrabright semiconducting polymer dots (Pdots)-based near-infrared-II (NIR-II) imaging platform to assess tumor vasculature and evaluate anti-angiogenic therapies. Stereo NIR-II imaging of xenograft tumors revealed that remnant living cells formed a denser vascular network than parental cells. To assess the efficacy of anti-angiogenic agents, we integrated Pdots-based NIR-II imaging with a 3D fluorescence imaging system in an oral squamous cell carcinoma (OSCC) model. Tumor-bearing mice implanted with MTCQ1 tongue cancer cells were treated with PX-478, a hypoxia-inducible factor-1 α (HIF-1 α) inhibitor, and BPR0C261, a microtubule-disrupting agent. Both agents significantly inhibited tumor growth, prolonged survival, and suppressed tumor vascularity without affecting body weight. Pdots-based NIR-II imaging demonstrated reduced tumor vascular density following treatment, consistent with ex vivo analysis showing decreased blood vessel formation. Immunohistochemical and Western blot analyses confirmed that PX-478 and BPR0C261 suppressed endothelial marker CD31 expression, while PX-478 additionally downregulated HIF-1a and VEGF-A, and BPR0C261 specifically reduced VEGF-A levels. These findings highlight the utility of Pdots-based stereo NIR-II imaging in evaluating angiogenesis and treatment response in aggressive tumor models. The identification of remnant living cells with CSC-like and antioxidant properties in late-stage HNC suggests that targeting oxidative stress pathways may enhance treatment efficacy. Additionally, the integration of advanced NIR-II imaging with biocompatible Pdots provides a powerful platform for real-time, non-invasive assessment of anti-angiogenic therapies, advancing personalized treatment strategies for aggressive head and neck cancers.



台灣分子生物影像學會 3/22 (Sat.) 15:20-16:20 2 樓, 20 教室





Current Position

Associate Professor, Department of Psychiatry, Taipei Veterans General Hospital and National Yang Ming Chiao Tung University, Taiwan

Education/Training

2017 PhD, Department of Clinical Neuroscience, Karolinska Institutet, Stockholm, Sweden2003 MD, School of Medicine, National Yang-Ming University, Taipei, Taiwan

Professional and Research Experience

2017-Present Attending Psychiatrist, Department of Psychiatry, Taipei Veterans General Hospital, Taipei, Taiwan

Awards and Honors

2011 Fellowship Award, 2nd AsCNP (Asian College of Neuropsychopharmacology), Seoul, Korea

Multimodal Neuroimaging to Investigate Cognitive Impairment in Neuropsychiatric Disorders

楊凱鈞 Kai-Chun Yang Associate Professor, Department of Psychiatry, Taipei Veterans General Hospital and National Yang Ming Chiao Tung University, Taiwan

Cognitive impairment is a critical factor in neuropsychiatric disorders, significantly impacting functional outcomes independent of other clinical variables and representing a major unmet therapeutic need. Neuroimaging offers a powerful means to investigate the in vivo relationships between brain structure, function, neurochemistry, and cognition. While neuroimaging research has yielded valuable insights, translating these findings into clinically useful biomarkers remains a challenge. This talk argues that moving beyond single-region analyses to examine brain networks/circuits, and integrating multiple modalities through multimodal neuroimaging, are crucial steps toward this goal. Specifically, we will explore the advantages of multimodal approaches, including network/circuit-based analyses and the unique opportunities afforded by combined PET/MR systems for simultaneously assessing diverse aspects of brain function and structure. We will discuss the potential of these techniques to elucidate the mechanisms underlying cognitive impairment in neuropsychiatric disorders, as well as the associated challenges and future directions. Ultimately, multimodal neuroimaging holds immense promise for advancing our understanding of these debilitating impairments and paving the way for more effective treatment strategies.



台灣分子生物影像學會 3/22 (Sat.) 16:20-17:20 2 樓, 20 教室



Speaker/ 董久源 Howard Doong

Current Position

臺灣來富可得生物科技股份有限公司董事長 天主教輔仁大學生命科學系兼任教授

Education/Training

OTHERS NIH, National Cancer Institute, Lab of Pathology, Clinical Research Fellow PhD The University of Chicago, the Department of Organismal Biology Anatomy OTHERS Program Harvard-MIT, Division of Health Sciences and Technology

Professional and Research Experience

執行長,美國輝景生物醫藥公司(ABVC BioPharma, Inc, NASDAQ上市公司) 董事長,美國 BioKey 醫藥品製造受託(CDMO)公司 助理教授,美國馬里蘭州州立大學醫學院和生物技術研究所

Awards and Honors

美國病理醫師學會 (CAP) 認證醫學實驗室主任 (Next Generation Sequencing) 臺灣首家在納斯達克資本市場上市的生物科技製藥公司 (ABVC) 執行長 美國國立衛生研究院國家癌症研究所最佳研究員獎 (Fellowship Award) 得獎主

Clinical Applications of Stem Cell-derived Exosomes

董久源 Howard Doong 臺灣來富可得生物科技股份有限公司 董事長 天主教輔仁大學生命科學系 兼任教授

Exosomes are biological nanoscale spherical lipid bilayer vesicles with a diameter of 40-200 nm secreted by cells. Exosomes act as intercellular messengers and have been regarded as miniature versions of their parental cells, partially because exosomes from a certain cell type provide cellspecific or unique sets of biomolecules (DNA, RNA & proteins). Exosomes are thought to be able to inherit similar therapeutic effects from their parent cells, such as embryonic and adult stem cells, through vertical delivery. Compared to stem cells, stem cell-derived exosomes possess numerous advantages, such as non-immunogenicity, non-infusion toxicity, easy access, effortless preservation, and freedom from tumorigenic potential and ethical issues. By reviewing relevant literature in recent years, this lecture is focusing on the applications and potential uses of stem cell-derived exosomes. Exosomes derived from mesenchymal stem cells are capable of treating numerous diseases encountered in orthopedics, neurology, plastic surgery, general surgery, thoracic surgery, cardiology, urology, head and neck surgery, ophthalmology, and obstetrics and gynecology. The diverse therapeutic effects of stem cell-derived exosomes are through a hierarchical translation of tissue-specific responses and cell-specific molecular signaling pathways. Future studies will combine insights from medical doctors, nanomedicine scientists and stem cell researchers in this intriguing area of research.



中華民國臨床生化學會 3/22 (Sat.) 14:20-15:00 3 樓, 31 教室





Current Position

Chief, Division of Endocrinology and Metabolism, Department of Internal Medicine, National Taiwan University Hospital

Professor, Faculty, Department of Internal Medicine, College of Medicine, National Taiwan University, Taiwan

Education/Training

- 1989 MD, Chung-Shan Medical University, Taiwan
- 2003 PhD, National Taiwan University, Taiwan (Physiology)
- 2007 OTHERS, Graduate Institute of Business Administration, National Taiwan University

Awards and Honors

- 2013 Professor Fan-Wu Chen's Outstanding Research Award from the Endocrinology
- 2010 Outstanding Publication Award of the Endocrinology Society of the Republic of China
- 2008 Excellent Publication Award in Journal of the Taiwan Internal Medicine Society

A Naïve Incident Biomarker Journey: Urinary Exosomal Peptides

王治元 Chih-Yuan Wang

Chief, Division of Endocrinology and Metabolism, Department of Internal Medicine, National Taiwan University Hospital, Professor, Faculty, Department of Internal Medicine, College of Medicine, National Taiwan University, Taiwan

Thyroid cancer, a common endocrine malignancy, remains a clinical challenge with recurrence rates as high as 30% even after thyroidectomy and radioactive iodine therapy. Traditional approaches relying on serum biomarkers, such as thyroglobulin, have limitations, particularly in cases complicated by anti-thyroglobulin antibodies or suboptimal sensitivity. Advances in molecular biology have brought urinary exosomal peptides into the spotlight as innovative, non-invasive alternatives for prognostics in thyroid cancer. These nano-sized vesicles, secreted by cells into bodily fluids like urine, serve as carriers of proteins, nucleic acids, and lipids, reflecting the state of their originating cells and offering a reliable window into disease progression. Studies have demonstrated a strong correlation between urinary exosomal peptides, such as thyroglobulin, tissue inhibitor of metalloproteinase (TIMP), and angiopoietin-1, with advanced thyroid cancer stages and lymph node metastasis. One study revealed that elevated preoperative levels of TIMP and angiopoietin-1 in urinary exosomes were significantly associated with lymph node metastasis, highlighting their value for identifying high-risk patients before surgery. Similarly, urinary exosomal thyroglobulin has shown potential in detecting recurrence post-thyroidectomy, even in cases where serum thyroglobulin levels fail to provide accurate results. Such findings underscore the clinical importance of these biomarkers in preoperative risk stratification and long-term surveillance. Longitudinal research has further validated the utility of urinary exosomal peptides in long-term monitoring. Another study tracked peptide levels in thyroid cancer patients over a decade and found minimal fluctuations among patients without recurrence, establishing their stability as reliable biomarkers. For high-risk individuals, consistent levels of urinary exosomal peptides within defined basal ranges correlated with a lower likelihood of recurrence, offering a non-invasive and reassuring monitoring tool for clinicians and patients alike. Urinary exosomal biomarkers hold several advantages over traditional methods. Urine collection is non-invasive, simple, and cost-effective, avoiding the need for expensive recombinant TSH stimulation or repeated imaging. Exosomes also protect their molecular cargo from enzymatic degradation, ensuring higher sensitivity and integrity of diagnostic data. Furthermore, they are unaffected by anti-thyroglobulin antibodies, a common limitation of serum thyroglobulin tests. Despite their promise, challenges such as standardizing methods for exosome isolation, peptide analysis, and large-scale validation remain. However, with ongoing advances in nanotechnology and bioinformatics, these obstacles are likely to be overcome. Although I hope urinary exosomal peptides could be a paradigm shift for thyroid cancer management in the future, offering a non-invasive, sensitive, and transformative approach to improving patient outcomes and quality of care. We still need more studies and research with ongoing program.

研討會演講 Symposia

> 中華民國臨床生化學會 3/22 (Sat.) 15:20-16:00 3 樓,31 教室





Speaker/ 楊崑德 Kuender D. Yang

Current Position

Vice Superintendent, MacKay Children's Hospital Professor, MacKay Medical College

Education/Training

- 1989 PhD, Immunology, National Defense Medical Center, Taiwan
- 1983 MD, Medicine, National Defense Medical Center, Taiwan

Professional and Research Experience

- 2016-Present Affiliated Professor, National Defense Medical Center, Taipei, Taiwan
- 2015-Present Professor, Department of Medical Research, Mackay Memorial Hospital; Institute of Biomedicine, Mackay Medical College, Taipei, Taiwan
- 2012-Present Affiliated Professor, Institute of Medical Sciences, National Yang Ming University, Taiwan

Awards and Honors

- 2023 20th Annual National Biotechnology Award
- 2022 The first place of the mentorship for medical student research, MacKay Medical School
- 2020 World top 2% Influential Scientist

臍帶間質幹細胞外泌體跨 3 代人機轉性臨床應用發展 Cross-generation mechanistic applications of exosomes from umbilical cord mesenchymal stem cells

楊崑德 Kuender D. Yang Vice Superintendent, MacKay Children's Hospital, Professor, MacKay Medical College

外泌體在細胞通訊中扮演關鍵角色,健康幹細胞的外泌體具再生與抗炎功能,而老化細胞或 癌細胞外泌體則可能促進老化與癌症。2013年,外泌體研究獲諾貝爾獎肯定。我們深耕間 質幹細胞及外泌體研究逾20年,利用醫療廢棄臍帶分離幹細胞,開發特色條件培養液與多 種外泌體製劑,並探索藥物載體應用。為推動再生醫療與節能減碳,我們建立多層次應用 模式。透過1)取得生產婦女同意後收集臍帶,2)分離與培養幹細胞,3)製備外泌體製劑,4) B2B 授權異體與自體應用,5) B2C 提供抗老、抗皺與抗肌少症產品,串聯學術、產業與醫療 機構,推動全民參與的再生醫療模式。外泌體為器官移植困境提供潛在解方。全球千萬人等 待移植,成功率低於5%。我們透過臍帶間質幹細胞(ucMSC)分離30-200nm 外泌體,發展 早期再生醫療,可能取代器官移植。外泌體無細胞核,免疫相容性高,具再生與免疫調節因 子,可經多種途徑給藥,優於細胞治療,並已獲專利技轉。其應用涵蓋三代人退化疾病,包 括:a) 早產兒腦缺氧與肺纖維化,b) 成人外傷與器官纖維化,c) 老年皺紋與退化疾病。此外, 外泌體具精準醫療價值。液態切片技術已應用於循環腫瘤細胞(CTC)監測,循環外泌體(CTE) 可進一步提升癌症與抗老治療精準度,為人類健康帶來突破。



中華民國臨床生化學會 3/22 (Sat.) 16:00-16:40 3 樓, 31 教室





Current Position

Associate Professor, Department of Biology and Anatomy National Defense Medical Center, Taipei, Taiwan, R.O.C.

Education/Training

- 2007 PhD, Department of Anatomy and Cell Biology, College of Medicine, National Taiwan University, Taiwan
- 1998 MS, Department of Anatomy and Cell Biology, College of Medicine, National Taiwan University, Taiwan
- 1996 BS, Department of Life Sciences, National Cheng Kung University, Taiwan

Professional and Research Experience

2015-Present Associate Professor, Department of Biology and Anatomy, National Defense Medical Center, Taipei, Taiwan, R.O.C.

Anti-NLRP3 inflammasome activation of GM1 ganglioside in microglia

黃雍協 Yuahn-Sieh Huang Associate Professor, Department of Biology and Anatomy National Defense Medical Center, Taipei, Taiwan, R.O.C.

Exogenous GM1 ganglioside has the potential to modulate innate immunity, suppressing LPSinduced activation of microglial cell lines and macrophages. The NLRP3 inflammasome, a critical protein in innate immunity, triggers robust inflammatory responses and is implicated in the progression of neurodegenerative diseases. The aim of this study was to investigate whether GM1 is involved in regulating NLRP3 inflammasome activation and the underlying mechanisms. We found that GM1 inhibits NLRP3 inflammasome activation in MG6 microglial cells in a dosedependent manner, as evidenced by decreased ASC puncta staining and NLRP3 and cleaved caspase-1 protein levels. LDH and ELISA assays indicated that GM1 decreased LPS/ATP-induced GSDMD-mediated pyroptosis and IL-1ß secretion, respectively. Mechanistically, GM1 inhibits LPS/ ATP-induced mtROS levels and reduces lysosomal cathepsin B release, both of which contribute to NLRP3 inflammasome activation. In LPS-primed MG6 cells, GM1 inhibited NF-B activation and suppressed the production of NLRP3 and pro-IL-1β. Furthermore, GM1 promoted autophagy/ mitophagy, which also contributes to the inhibition of NLRP3 inflammasome activation. In an animal study using LPS-treated mice, GM1 administration decreased the protein levels of NLRP3 and ASC in microglia. In conclusion, GM1 alleviates NLRP3 inflammasome activation and pyroptosis by modulating NF-B, mtROS and autophagy. GM1 can be a potential candidate for the treatment of NLRP3 inflammatory neurodegenerative diseases.



中華民國解剖學學會 3/22 (Sat.) 13:30-14:00 3 樓, 32 教室



Current Position

JACBS

Assistant Professor, Department of Anatomy, School of Medicine, China Medical University, Taiwan

Education/Training

2018 PhD, Graduate Institute of Life Sciences, National Defense Medical Center
2010 MS, Graduate Institute of Biology and Anatomy, National Defense Medical Center

Professional and Research Experience

- 2022-Present Assistant Professor, Department of Anatomy, School of Medicine, China Medical University, Taiwan
- 2018-2022 Postdoctoral fellow, Institute of Cellular and Organismic Biology, Academia Sinica, Taiwan

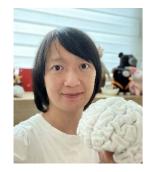
Hippocampal Development and Ventralization: The Role of COUP-TFI in Patterning

曾慶三 Ching-San Tseng Assistant Professor, Department of Anatomy, School of Medicine, China Medical University, Taiwan

As one of the most-studied brain regions, the hippocampus is renowned for its essential role in cognitive processes such as episodic memory and spatial learning; however, it also contributes to interoceptive emotions such as anxiety and depression. Along its longitudinal axis, the hippocampus is commonly divided into two halves: the dorsal and ventral hippocampi. Early studies with region-specific ablations demonstrate their functional specializations: the dorsal hippocampus is involved in spatial learning and memory processes, while the ventral hippocampus is implicated in motivational and emotional behaviors. These two functionally distinct domains differ in anatomy, histology, transcriptome, and disease susceptibilities. However, how these regions are established during hippocampal embryogenesis remains largely unknown. In our preliminary results, we found that the transcription factor COUP-TFI (chick ovalbumin upstream transcription factor I, or Nr2f1) is distributed in a low dorsal-to-high ventral gradient in the hippocampal epithelium, suggesting its role in the development of ventral populations. By comparing the hippocampal cytoarchitecture among wild-type, COUP-TFI conditional knockout (cKO), and conditional transgenic (cTG) mice, we showed that hippocampal volume was greatly reduced in the COUP-TFI-cKO but expanded in the COUP-TFI-cTG. Moreover, further analyses of CA1 pyramidal cell layer thickness, CA1 neuronal compositions, and hippocampal regional markers demonstrated that the hippocampus was dorsalized in COUP-TFI-cKO and ventralized in COUP-TFI-cTG. This process involves the antagonistic regulation of the Wnt and SHH signaling pathways, key players in hippocampal development. Furthermore, we are conducting behavioral analyses of COUP-TFI mutants with modified hippocampal structures to determine the functional outcomes of altered hippocampal patterning. These experiments aim to confirm the behavioral changes associated with altered COUP-TFI levels. In conclusion, our findings reveal a novel mechanism by which COUP-TFI modulates hippocampal ventralization, providing insights into the neural specialization that underlies disease susceptibilities, such as autism spectrum disorders and Alzheimer's disease.



3 樓,32 教室





Current Position

Brain Research Center, National Defense Medical Center, Taipei, Taiwan

Education/Training

- 2017 PhD, Department of Psychology, National Taiwan University, Taipei, Taiwan
- 2007 MS, Department of Psychology, National Taiwan University, Taipei, Taiwan
- 2004 BS, Department of Psychology, National Taiwan University, Taipei, Taiwan

Professional and Research Experience

- 2023-Present assistant professor, Brain Research Center, National Defense Medical Center, Taipei, Taiwan
- 2021-2023 post-doc researcher, Department of Psychology, National Taiwan University, Taipei, Taiwan

A preliminary MRI brain template for Taiwanese macaque

陳可欣 Ke-Hsin Chen Brain Research Center, National Defense Medical Center, Taipei, Taiwan

Non-human primates (NHPs) have long been critical models in biomedical research. Compared to other lab animals (e.g., fruit fly, rodents), NHPs are phylogenetically closer to humans, and thus provide better models of the health and diseases in terms of genetics, anatomy, physiology and behavior. For instance, in neuroscience, their large brain, high intelligence and sociability, make them especially suitable for the studies of higher cognitive functions and neuropsychiatric disorders. Following the COVID pandemic and the growing interests in brain-machine interfaces, there is a surge of the demand of NHP models. Nonetheless, the supply remains limited as among all the primate species, only a few are widely used as the animal model for research – namely, the rhesus macaque (Macaca mulatta), crab-eating macaque (Macaca fascicularis), Japanese macague (Macaca fuscata) and common marmosets (Callithrix jacchus). Formosan rock macague (Macaca cyclopis), also known as Taiwanese macaque, is the native primate living in Taiwan and is a close relative of the rhesus and Japanese macaques. However, the feasibility of using it in biomedical research, especially in neuroscience, has rarely been studied. To facilitate this species to be used in brain researches, a standard anatomical template is required for data analysis and comparison across subjects and studies. As a first step, in-vivo magnetic resonance images (MRI), including T1W, T2W, FGATIR and DTI, were collected from seven Taiwanese macagues (3) females). A preliminary MRI template with tissue segmentation maps was conducted to serve as a neuroimaging tool for analysis and visualization. To delineate cytoarchitecture using wholebrain sectioning and Nissl stain in the future, a high-resolution ex-vivo MRI scan of a perfused brain was acquired to achieve precise image registration between the MRI template and histological images. In conclusion, the present study provides a preliminary neuroimage tool for Taiwanese macaque, and henceforth a comprehensive anatomical brain template and atlas will be developed.



中華民國解剖學學會 3/22 (Sat.) 14:30-15:00 3 樓,32 教室





Current Position

Distinguished Professor, Department of Radiology, College of Medicine, Taipei Medical University Chief, Section of Neuroradiology, Department of Medical Imaging, Taipei Medical University Hospital

Education/Training

1985 MD, Medical degree in School of Medicine, National Defense Medical Center, Taipei

Professional and Research Experience

- 2019-2023 Vice president, Taipei Medical University
- 1990-2011 Attending Neuroradiologist, Department of Radiology, Tri-Service General Hospital, Taipei
- 1992-1993 Clinical Researcher, Department of Radiology, The Children Hospital's of Philadelphia, USA

Awards and Honors

- 2024 National Science and Technology Council Academic Research Award, Taiwan
- 2024 ASNR Honorary Member Award
- 2024 Outstanding Contribution Award, Wang Ming-Ning Memorial Foundation, Taiwan

CNS Lymphatic-Glymphatic System from Neuroimaging Perspectives

陳震宇 Cheng-Yu Chen

Distinguished Professor, Department of Radiology, College of Medicine, Taipei Medical University, Chief, Section of Neuroradiology, Department of Medical Imaging, Taipei Medical University Hospital

The discovery of the meningeal lymphatic vessels and the glymphatic system has revolutionized our understanding of CNS fluid balance, immune surveillance, and waste clearance. The meningeal lymphatic vessels, located parallel to the dural venous sinuses and middle meningeal arteries, drain immune cells, small molecules, and excess fluid from the CNS into the deep cervical lymph nodes. These vessels function downstream of the glymphatic system, a brain-wide network of perivascular spaces that facilitates the clearance of metabolic waste products, particularly during sleep. Dysfunction of these systems has been implicated in various neurological disorders, including neurodegenerative diseases, stroke, and head trauma. Evaluating the glymphatic system in humans remains challenging due to the lack of approved fluorescent tracers and the invasive nature of intrathecal gadolinium-based contrast agents (GBCA). Non-invasive neuroimaging techniques have emerged as promising alternatives, with the Diffusion Tensor Image Analysis along the Perivascular Space (DTI-ALPS) method gaining attention for its ability to indirectly evaluate glymphatic function through the ALPS-index. However, recent critiques have guestioned its reliability due to sensitivity to imaging conditions and issues like fiber crossing. Other techniques, such as choroid plexus volume assessment, perivascular space volume measurement, and evaluations of blood-brain barrier or venous wall permeability using GBCA, offer complementary insights into glymphatic function. Additionally, clearance-specific techniques like diffusion-weighted arterial spin labeling (DW-ASL) have shown promise in imaging aquaporin-4, a key water channel involved in glymphatic transport. This talk will address the limitations of individual techniques and introduce a multimodal imaging approach integrating structural imaging, dynamic assessment, and clearance-specific techniques. By advancing our knowledge of glymphatic function in health and disease through multimodal neuroimaging, we can ultimately develop improved diagnostic and therapeutic strategies for neurological disorders.



1樓,第一教室

th 2025 The 39th Joint Annual Conference of Biomedical Science 生物醫學聯合學術年會



p^{eaker /} 蔡欣熹 Hsin-Hsi Tsai

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Current Position 台灣大學醫學院神經科臨床助理教授 台大醫院神經部主治醫師

Education/Training 2021 PhD, 台灣大學臨床醫學研究所 2012 MD, 台灣大學醫學系

Professional and Research Experience

2021-2023 主任,台大醫院北護分院教學研究部
2018-2022 兼任講師,台灣大學醫學院神經
2013-2023 主治醫師,台大醫院北護分院神經內科

Awards and Honors

- 2024 國科會吳大猷先生紀念獎
- 2023 Paul Dudley White International Scholar(International Stroke Conference)
- 2023 腦血管疾病防治基金會高明見教授優秀論文獎

Meningeal Lymphatic System—A Potential Treatment Target for Stroke Patients

蔡欣熹 Hsin-Hsi Tsai 台灣大學醫學院神經科臨床助理教授 台大醫院神經部主治醫師

Lymphatic drainage is essential for maintaining overall tissue fluid and solute balance, proper metabolic function, and macromolecule clearance. The newly discovered meningeal lymphatic system within the dura mater carries macromolecules away from the brain parenchyma and transports cerebral spinal fluid to the cervical lymph nodes in the periphery. This system has been considered to play a major role in neurodegenerative diseases and other central nervous system disorders, including stroke. In this talk, I will briefly introduce current advances in the understanding of meningeal lymphatic system in different stroke subtypes, including ischemic stroke, subarachnoid hemorrhage and intracerebral hemorrhage. We recently performed a pilot study which investigated the contribution of the meningeal lymphatic system to intracerebral hemorrhage pathologies using animal models. we observed that meningeal lymphangiogenesis and increased lymphatic drainage occurred until late phase after stroke, suggesting a potential role in the recovery phase. The impairment of meningeal lymphatic function impeded intraparenchymal hematoma resolution, whereas its enhancement reduced hematoma volume and ameliorated neurological deficits. Based on the results from current literature and hypothesis, meningeal lymphatics has been considered to have major implications after strokes, and yet its pathophysiology and translational potential remain to be tested in future studies.



台灣藥理學會 3/23 (Sun.) 09:30-10:00 1 樓,第一教室





Current Position

Professor & Director, Institute of Clinical Medicine, National Yang Ming Chiao Tung University, Taipei, Taiwan

Chief, Division of Translational Research, Department of Medical Research & Attending Neurologist, Department of Neurology, Taipei Veterans General Hospital, Taipei, Taiwan

Education/Training

OTHERS, Neurovascular Research Lab, Massachusetts General Hospital, Harvard Medical School PhD, Institute of Clinical Medicine, National Yang-Ming University, Taipei, Taiwan MD, School of Medicine, National Yang-Ming University, Taipei, Taiwan

Professional and Research Experience

- 2021-Present Professor, Institute of Clinical Medicine, National Yang Ming Chiao Tung University, Taipei, Taiwan
- 2017-Present Attending Physician, Division of Translational Research, Department of Medical Research, Taipei Veterans General Hospital
- 2008-2017 Attending Physician, Department of Neurology, Neurological Institute, Taipei Veterans General Hospital

Awards and Honors

- 2022 Outstanding Research Award, Ministry of Science and Technology, Taiwan (科技部 110 年度傑出研究獎)
- 2021 Tien Te Lee Biomedical & Technology Award (李天德青年醫藥科技獎)
- 2019 Ta-You Wu Memorial Award, Ministry of Science and Technology, Taiwan (科技部吳大猷 先生紀念獎)

Glymphatics and Meningeal Lymphatics in Complex Neurovascular Disorders

陳世彬 Shih-Pin Chen

Professor & Director, Institute of Clinical Medicine, National Yang Ming Chiao Tung University, Taipei, Taiwan, Chief, Division of Translational Research, Department of Medical Research & Attending Neurologist, Department of Neurology, Taipei Veterans General Hospital, Taipei, Taiwan

The glymphatic system and meningeal lymphatics have emerged as critical components in brain homeostasis, waste clearance, and neuroimmune regulation. The glymphatic system facilitates the perivascular transport of cerebrospinal fluid and interstitial solutes, while meningeal lymphatic vessels provide an essential drainage route for immune cells and macromolecules from the central nervous system to peripheral circulation. Dysfunction in these systems has been linked to neuroinflammation, impaired cerebrovascular reactivity, and the accumulation of neurotoxic proteins, all of which may contribute to the pathogenesis of complex neurovascular disorders. To investigate these processes, we have developed non-invasive imaging techniques to visualize human glymphatic and meningeal lymphatic dynamics, enabling their exploration in translational research. Our studies have examined the roles of glymphatic and meningeal lymphatic dysfunction in neurovascular disorders such as migraine, reversible cerebral vasoconstriction syndrome, and cerebral small vessel disease. These findings highlight the importance of preserving glymphatic and meningeal lymphatic function for the prevention and treatment of neurovascular diseases. Further research into the mechanisms underlying their dysfunction may pave the way for novel therapeutic strategies targeting these clearance pathways.



台灣藥理學會 3/23 (Sun.) 10:00-10:30 1 樓,第一教室

th 2025 The 39th Joint Annual Conference of Biomedical Science 生物醫學聯合學術年會



JACBS



Current Position

Associate Professor, College of Medicine, National Taiwan University, Taiwan Director, Department of Physical Medicine and Rehabilitation, National Taiwan University Hospital Hsin-Chu Branch, Taiwan

Education/Training

2005 MD, Medicine, National Taiwan University, Taipei, Taiwan
2020 PhD, Institute of Biomedical Engineering, National Taiwan University, Taipei, Taiwan

Professional and Research Experience

- 2012-Present Attending physician, Department of Physical Medicine and Rehabilitation, National Taiwan University Hospital, Taipei, Taiwan
- 2020-2020 Director, Department of General Medicine, National Taiwan University Hospital Biomedical Park Branch, Hsinchu, Taiwan
- 2017-2021 Assistant professor, Department of Physical Medicine and Rehabilitation, College of Medicine, National Taiwan University, Taipei, Taiwan

Awards and Honors

- 2024 World's Top 2% Scientists (Elsevier Data Repository)
- 2024 Taiwan Academy of Physical Medicine and Rehabilitation Excellent Research Award
- 2024 Professor Chen Xiyao's Outstanding Ultrasound Paper Award

Enhancing Glymphatic Function via Ultrasound: Therapeutic Potential for Stroke and ALS

吳爵宏 Chueh-Hung Wu

Associate Professor, College of Medicine, National Taiwan University, Taiwan, Director, Department of Physical Medicine and Rehabilitation, National Taiwan University Hospital Hsin-Chu Branch, Taiwan

The glymphatic system plays a crucial role in maintaining brain homeostasis by facilitating the clearance of metabolic waste and toxins through cerebrospinal fluid and interstitial fluid exchange. Dysfunction of this system has been implicated in neurological disorders, including stroke and amyotrophic lateral sclerosis (ALS). Recent advancements in ultrasound technology, particularly very low-intensity ultrasound (VLIUS), have shown promising potential in modulating glymphatic function. This presentation explores the mechanisms by which VLIUS enhances glymphatic activity, focusing on its ability to influence the TRPV4-AQP4 pathway. Preclinical studies showed that ultrasound stimulation can improve waste clearance and promote functional recovery in stroke models. Similarly, in ALS, VLIUS holds potential to slow disease progression. By highlighting the therapeutic implications of ultrasound in enhancing glymphatic function, this talk aims to provide insights into this novel, non-invasive strategy for treating these debilitating conditions.



台灣藥理學會 3/23 (Sun.) 10:30-10:50 1 樓,第一教室



Current Position

JACBS

Head, Department of Microbiology and Immunology, College of Medicine, Chang Gung University, Taiwan.

Adjunct Researcher, Division of Rheumatology, Allergy, and Immunology, Chang Gung Memorial Hospital-Keelung, Keelung, Taiwan

Education/Training

- 1997 PhD, Oak Ridge Graduate School of Biomedical Sciences, University of Tennessee -Knoxville TN, U.S.A.
- 1989 MS, Institute of Biochemistry, College of Medicine, National Taiwan University, Taipei, Taiwan
- 1987 BS, Department of Biology, National Cheng Kung University, Tainan, Taiwan

Professional and Research Experience

- 2013-2022 Director, Graduate Program of Molecular Medicine, College of Medicine, Chang Gung University, Taiwan.
- 2016-Present Adjunct Researcher, Department of Anatomic Pathology, Chang Gung Memorial Hospital-Linkou, Taoyuan, Taiwan
- 2016-2017 Visiting Professor, The Kennedy Institute of Rheumatology, University of Oxford, UK

Awards and Honors

- 2023 Elected Full Member of Sigma Xi, The Scientific Research Honor Society
- 2023 Outstanding Research Award, The Chinese Society of Immunology
- 2012 Board Member, Adhesion-GPCR Consortium

The role of GPR97-induced PAR2 transactivation in neutrophil-driven inflammatory responses

林錫賢 Hsi-Hsien Lin

Head, Department of Microbiology and Immunology, College of Medicine, Chang Gung University, Taiwan., Adjunct Researcher, Division of Rheumatology, Allergy, and Immunology, Chang Gung Memorial Hospital-Keelung, Keelung, Taiwan

Neutrophils play a vital role in the innate immune system, contributing significantly to antimicrobial defense and inflammatory responses. Abnormal neutrophil dysfunction usually results in harmful inflammatory or autoimmune diseases, highlighting the need for stringent regulation of their immune effector activities. Neutrophils harbor various intracellular proteinases, including proteinase 3 (PR3) and myeloperoxidase, which are essential for effective microbial killing. Interestingly, these two proteins are also the primary targets of autoantibodies responsible for rare autoimmune diseases, specifically granulomatosis with polyangiitis and microscopic polyangiitis. Our recent research has uncovered a novel allosteric activation mechanism for membrane PR3 (mPR3), involving the formation of a unique PR3/CD177/GPR97/PAR2/CD16b protein complex on the neutrophil surface. This receptor complex enables GPR97 to enhance the proteolytic activity of mPR3, which subsequently cleaves and transactivates PAR2, leading to robust neutrophil activation. The molecular mechanism underlying mPR3-mediated GPR97-PAR2 transactivation in neutrophils will be discussed here. We propose that the CD177/GPR97/ PAR2/CD16b receptorsome constitutes a multi-target complex with significant potential for developing therapeutics aimed at modulating human neutrophil-driven inflammatory diseases.







Current Position

Research Fellow, Institute of Biomedical Sciences, Academia Sinica, Taiwan

Education/Training

- 2003 PhD, National Cheng Kung University
- 1999 MD, National Taiwan University

Professional and Research Experience

- 2019-2020 Professor, Institute of Epidemiology and Preventive Medicine, National Taiwan University, Taiwan
- 2014-2019 Associate Professor, Institute of Epidemiology and Preventive Medicine, National Taiwan University, Taiwan
- 2009-2014 Assistant Professor, Institute of Epidemiology and Preventive Medicine, National Taiwan University, Taiwan

Awards and Honors

- 2023 Chief in Biotechnology, Taiwan Bio-develop Foundation, Taiwan
- 2021 18th National Innovation Award in Academic Research, Taiwan
- 2018 Outstanding Research Award, National Science and Technology Council, Taiwan

Developing novel nanoimmuno-drugs targeting dendritic cells for cancer therapy

李永凌 Yungling Leo Lee Research Fellow, Institute of Biomedical Sciences, Academia Sinica, Taiwan

Immune checkpoint inhibitor therapy and adoptive cell transfer immunotherapy harness components of the immune system to fight tumor cells. Dendritic cells (DCs), a critical linker between innate and adaptive immunity, are important targets for PD-1 axis blockade, indicating that developing DC-targeting drugs could benefit cancer therapy. Our previous research revealed that tumor growth was profoundly restricted in AhR DC-conditional knockout (AhRf/ f CD11cCRE) mice. Therefore, we discovered and inserted synthetic peptide 65 (SP65) via phage display onto surface of liposomal CH223191 (SP65-lipo-CH), having considerable affinity with DCs. In non-tumor models, SP65-lipo-CH applied on DCs would induce IL-12 production which resulted in IFN- γ production from NK cells. Additionally, it should also be emphasized that AhR inhibition on DCs reduced PD-L1 expression on surface. In a tumor xenograft model, SP65-lipo-CH demonstrated moderate efficacy against MC38 through NK cells activation and degranulation. Furthermore, the majority of tumors were eradicated and became undetectable when mice were co-administrated with SP65-lipo-CH and anti-PD-1. In an orthotopic and metastatic model, SP65-lipo-CH application two days prior to tumor inoculation effectively suppressed LLC growth in lungs, which could stem from NK cells activation via IL-12 from DCs. Our findings suggest that SP65 is a powerful ligand to target DCs and enhance drug delivery into DCs. SP65-lipo-CH illustrates future "off-the-shelf" products and holds substantial promise for cancer immunotherapy.







Current Position

Associate Professor, Institute of Clinical Medicine National Yang-Ming University

Education/Training

- 2009 PhD, National Yang-Ming University, Taiwan
- 2021 MS, National Taiwan University, Taiwan
- 1998 BS, National Taiwan University, Taiwan

Professional and Research Experience

- 2021-Present Adjunct Associate Professor, Biomedical Industry Ph.D. Program
- 2020-Present Adjunct Associate Professor, Institute of Emergency and critical care medicine, National Yang Ming Chiao Tung University
- 2015-2020 Assistant Professor, Institute of Clinical Medicine, National Yang-Ming University Taiwan

Awards and Honors

- 2023 Wu Ho-Su TBF Taiwan Bio-development Foundation Medical Award
- 2023 Travel Grant Winner, 15TH International Congress on Systemic Lupus Erythematosus
- 1970 Outstanding Research Scholar Award, Chinese Society of Immunology

NLRP12: An Innate Immune Checkpoint Managing Health and Pathology through the Regulation of Type I IFN Production

陳斯婷 Szu-Ting Chen Associate Professor, Institute of Clinical Medicine National Yang-Ming University

Innate immunity serves as the first line of host defense against infections. It also maintains physiological balance, influences the composition of the microbiota, and plays crucial roles in contexts of disease progression. NLRP12, a member of the nucleotide-binding oligomerization domain (NOD)-like receptors (NLRs) superfamily, is primarily expressed by cells of the myeloid lineage and serves as an innate immune checkpoint to regulate the activation of signaling pathways driven by innate immune receptors. NLRP12 limits DSS-induced colon inflammation and tumorigenesis through the negative regulation of canonical and noncanonical NF- κ B signaling in an experimental colitis model. NLRP12 suppresses NLRP3 inflammasome activation by physically interacting with NLRP3, thereby nonsense mutations in NLRP12 increase NLRP3 inflammasome activity and spontaneous release of IL-1 β in patients with autoinflammatory diseases due to the loss of confinement between NLRP12 and NLRP3. Additionally, NLRP12 suppresses virus and nucleic acid-induced type I IFN (IFN-I) production. This suppression occurs through the downregulation of NLRP12 expression, which releases the confinement within the type I IFN receptor signaling during virus infection. Consequently, the host regulates innate immune signaling by modulating the expression levels of NLRP12, leading to an anti-viral response through increased IFN-I production. However, prolonged low NLRP12 expression results in excessive IFN-I production, facilitating the progression of inflammatory diseases, such as systemic lupus erythematosus (SLE). The ability of NLRP12 to limit IFN-I production is linked to its role in suppressing neutrophil hyper-responsiveness to bacterial infections and stimulation by nucleic acid-containing immune complexes derived from SLE patients. By constraining excessive neutrophil activation, NLRP12 functions as an innate immune checkpoint, shaping host defense mechanisms and maintaining immune homeostasis.



台灣藥理學會 3/23 (Sun.) 14:00-14:30 1 樓,第一教室





Current Position

Assistant Research Fellow, Department of Medical Research, E-Da Hospital, Taiwan

Education/Training

- 2006 PhD, Graduate Institute of Life Sciences, National Defense Medical Center and Academia Sinica, Taipei, Taiwan
- 2003 MS, Department of Medical Biotechnology and Laboratory Science, Chang Gung University, Taoyuan, Taiwan
- 1999 BS, Department of Biomedical Sciences, Chung Shan Medical University, Taichung, Taiwan

Professional and Research Experience

- 2018-2021 Assistant Professor, Department of Cosmetic Science, Providence University, Taichung, Taiwan
- 2014-2018 Postdoctoral Fellow, Graduate Institute of Natural Products, Chang Gung University, Taoyuan, Taiwan
- 2012-2014 Postdoctoral Fellow, Genomics Research Center, Academia Sinica, Taipei, Taiwan

Awards and Honors

- 2023 Outstanding Alumni Award, Department of Biomedical Sciences, Chung Shan Medical University
- 2022 Dr. Tsungming Tu Young Investigator Award, The Pharmacological Society
- 2019 Junior Research Award, Society of Chinese Natural Medicine

Advancing the development of drug candidates for neutrophilic inflammatory diseases

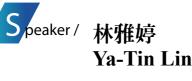
陳柏任 Po-Jen Chen Assistant Research Fellow, Department of Medical Research, E-Da Hospital, Taiwan

Neutrophilic inflammation, characterized by dysregulated neutrophil activation, triggers various inflammatory responses, including chemotactic infiltration, oxidative bursts, degranulation, and the formation of neutrophil extracellular traps (NETs). This type of inflammation is central to the pathogenesis of many inflammatory diseases, particularly acute respiratory distress syndrome (ARDS). Despite current treatments, managing neutrophil-associated inflammatory symptoms remains a significant challenge. To advance the development of drug candidates targeting neutrophilic inflammatory diseases, we focused on repurposed clinical drugs and natural products. First, we demonstrated that ribociclib, a cyclin-dependent kinase 4 and 6 (CDK4/6) inhibitor clinically used in cancer treatment, serves as a novel phosphodiesterase 4 (PDE4) inhibitor, effectively mitigating inflammatory responses in activated human neutrophils and alleviating ARDS symptoms in mice. Second, we showed for the first time that Bletinib derived from Bletilla formosana, a native medicinal plant in Taiwan, acts as a novel Src family kinases (SFKs) inhibitor to reduce neutrophilic inflammation-mediated lung damage in human neutrophils and mice. Together, the repurposing of ribociclib and the discovery of naturally occurring Bletinib highlight their potential as lead drug candidates for neutrophilic ARDS. Targeting neutrophilic PDE4 and SFKs offers promising off-label alternatives for treating lung lesions and other inflammatory conditions.



1樓,第一教室





Current Position

Assistant Professor, Graduate Institute of Metabolism and Obesity Sciences, Taipei Medical University, Taiwan

Education/Training

- 2016 PhD, Graduate Institute of Biomedical Sciences, Division of Physiology and Pharmacology, Chang Gung University, Taiwan
- 2009 MS, Graduate Institute of Basic Medical Sciences, Division of Physiology and Pharmacology, Chang Gung University, Taiwan

Professional and Research Experience

- 2021-Present Assistant Professor, Graduate Institute of Metabolism and Obesity Sciences, Taipei Medical University, Taiwan
- 2018-2019 Visiting Scholar, Institute of Neurobiology & Institute of Comparative Molecular Endocrinology, Ulm University, Germany
- 2016-2021 Postdoctoral Fellow, Graduate Institute of Biomedical Sciences, Division of Physiology and Pharmacology& Healthy Aging Research Center, Chang Gung University, Taiwan

Awards and Honors

2024 IUPS International Early Faculty Prize, The International Union of Physiological Sciences (IUPS)

Hypothalamic Insulin Resistance and Energy Balance: A Neuropeptide's Novel Contribution

林雅婷 Ya-Tin Lin Assistant Professor, Graduate Institute of Metabolism and Obesity Sciences, Taipei Medical University, Taiwan

The hypothalamus is a critical brain region that regulates peripheral metabolic functions through insulin signaling. Hypothalamic insulin signals act via multiple neuronal circuits and anabolic/catabolic pathways, ultimately converging on the vagus nerve and sympathetic fibers to coordinate energy metabolism across peripheral organs. Insulin resistance in the hypothalamus leads to dysregulated energy balance, characterized by increased food intake, enhanced lipolysis, elevated hepatic glucose production, reduced thermogenesis in brown adipose tissue, and impaired browning of white adipose tissue. These disruptions are key contributors to the onset and progression of metabolic disorders such as obesity and diabetes. In recent years, neuropeptide FF (NPFF) has emerged as a significant regulator of energy homeostasis. Our research focuses on elucidating the mechanisms by which NPFF influences metabolic disorders through its actions in the central nervous system. We have demonstrated that NPFF exacerbates obesity- and diabetes-related metabolic abnormalities, primarily through the activation of its type 2 receptor (NPFFR2) in the hypothalamic arcuate nucleus. Deletion of NPFFR2 in mice alleviated both central and peripheral metabolic disturbances associated with metabolic disorders. Additionally, NPFFR2 activation was found to impair hypothalamic insulin sensitivity while simultaneously enhancing feeding behavior. The role of NPFFR2 in promoting central insulin resistance is likely mediated by its induction of neuroinflammation. These findings provide valuable insights into the pathophysiological role of NPFF signaling and highlight NPFFR2 as a potential therapeutic target for metabolic disorders.







Current Position

Assistant Professor, Department of Life Sciences, National Chung Hsing University, Taiwan

Education/Training

2018 PhD, Graduate Institute of Physiology, College of Medicine, National Taiwan University, Taiwan

Professional and Research Experience

2019-2023 Post-doctoral researcher, Graduate Institute of Anatomy and Cell Biology, College of Medicine, National Taiwan University, Taiwan

Synergistic Effects of Particulate Matter and Hyperglycemia on Endothelial Inflammation: Oxidative Stress, Inflammation, and Potential Therapeutic Interventions

賴財春 Tsai-Chun Lai Assistant Professor, Department of Life Sciences, National Chung Hsing University, Taiwan

Cardiovascular diseases (CVDs) are associated with particulate matter (PM) exposure and diabetes, while the molecular mechanisms underlying their combined effects on endothelial damage remain unclear. Our previous study investigated the synergistic impact of high glucose (HG) and PM2.5 on endothelial inflammation and explores potential protective interventions. Human umbilical vein endothelial cells (HUVECs) and endothelial cells (ECs) were treated with 30 mM HG and 10 or 50 μ g/mL PM to simulate hyperglycemia and air pollution exposure. Cellular damage, apoptosis, and oxidative stress were assessed via reactive oxygen species (ROS) production, mitochondrial function assays, and Western blot analysis of autophagy-related proteins, mitophagy-related protein, and inflammation markers, including p62, LC3B, BNIP3, intercellular adhesion molecule-1 (ICAM-1), and vascular cell adhesion molecule-1 (VCAM-1). In vivo, streptozotocin (STZ)-induced diabetic mice were exposed to PM via intratracheal injection to evaluate endothelial inflammation. Potential protective effects of vitamin D and nanocurcumin (NCur) were investigated. Co-exposure to HG and PM significantly increased endothelial cell damage, apoptosis, and mitochondrial ROS production while reducing mitochondrial membrane potential. This exposure also promoted mitochondrial fission, autophagy, and mitophagy by upregulating DRP1, Fis1, p62, LC3B, and BNIP3. In vivo, PM exposure exacerbated oxidative stress, mitochondrial dysfunction, and endothelial inflammation in diabetic mice. Vitamin D and NCur effectively alleviated these effects by improving cell viability, reducing mitochondrial ROS levels, and modulating mitophagy and inflammation. Therefore, simultaneous exposure to PM and HG induces endothelial inflammation through oxidative stress, mitochondrial impairment, and inflammatory signaling. Vitamin D and NCur offer protective effects by reducing ROS, improving mitochondrial function, and modulating key inflammatory pathways. These findings suggest that Vitmain D and NCur may be promising therapeutic strategies for mitigating the impact of diabetes and air pollution on CVD progression.



中國生理學會 3/23 (Sun.) 14:54-15:18 1 樓,第二教室





Current Position

Assistant Professor, Institute of Physiology, National Yang Ming Chiao Tung University

Education/Training

2014 PhD, National Yang-Ming University

Professional and Research Experience

- 2020-2022 Postdoctoral Fellow, Department of Medical Research, Taipei Veterans General Hospital
- 2017-2020 Postdoctoral Fellow, Institute for Engineering in Medicine, University of California, San Diego

Awards and Honors

- 2022 Albert Ly-Young Shen Research Award
- 2018 Oversea Outstanding Youth Award, R.O.C.

RNA modifications in Cardiovascular Development and Disease

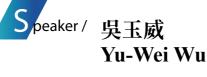
簡千栩 Chian-Shiu Chien Assistant Professor, Institute of Physiology, National Yang Ming Chiao Tung University

With the rapid increase in the aging population worldwide, cardiovascular diseases (CVDs) have become a major health threat to elderly individuals, inflicting a significant burden on healthcare systems. Therefore, elucidating the molecular mechanisms underlying cardiovascular development and disease progression is crucial for disease prevention and elderly health management. Recent studies have demonstrated that RNA modifications regulate gene expression and participate in various physiological processes, including cardiovascular development and pathology. However, the precise role of RNA modifications in vascular and cardiac development and diseases remains unclear. Our team mainly employs vascular and heart organoids as model systems to investigate the functional roles and mechanisms of RNA modifications in cardiovascular development and disease progression. In vascular research, we have identified that RNA modifications regulate vascular inflammation-related genes, thereby influencing the progression of atherosclerosis. Additionally, we have integrated imaging analysis with artificial intelligence (AI) technologies to identify the accurate vascular organoid differentiation assessment and further reveal a critical regulatory role of RNA methylation in vascular development. In cardiac research, we discovered that losing mitochondrial RNA methylation may promote chemo-drug-induced cardiotoxicity. Moreover, we established heart organoids to investigate the impact of RNA modifications on cardiac development and disease progression. Our future research will focus on elucidating the molecular mechanisms by which RNA modifications regulate vascular and cardiac development and contribute to disease. We will also develop RNA modification-based therapies to identify novel diagnostic biomarkers and therapeutic targets for cardiovascular diseases.



中國生理學會 3/23 (Sun.) 15:18-15:42 1 樓,第二教室





Current Position

Assistant Research Fellow, Institute of Molecular Biology, Academia Sinica, Taiwan

Education/Training

- 2012 PhD, Institute of Neurology (IoN), University College London UCL, London, UK
- 2007 MS, Institute of Zoology, National Taiwan University, Taipei, Taiwan
- 2003 BS, Department of Zoology, National Taiwan University, Taipei, Taiwan

Professional and Research Experience

- 2013-2019 Postdoctoral Research Fellow,, Department of Neurosurgery, Stanford University School of Medicine, Palo Alto, CA
- 2012-2013 Postdoctoral Research Fellow, RIKEN Brain Science Institute, Wako, Japan

Awards and Honors

- 2021 Career Development Award, Academia Sinica, Taiwan
- 2019 Academia Sinica Young Investigator Fellowship, Academia Sinica, Taiwan
- 2015 Postdoctoral Research Fellowship, Parkinson's Disease Foundation, USA

Mixed selectivity of subthalamic nucleus neurons in encoding motor and reward behaviors

吳玉威 Yu-Wei Wu Assistant Research Fellow, Institute of Molecular Biology, Academia Sinica, Taiwan

The subthalamic nucleus (STN) plays a critical role in modulating motor and cognitive functions within the basal ganglia, and its involvement in Parkinson's disease (PD) and deep brain stimulation (DBS) is well established. However, the behavioral representations of individual STN neurons remain poorly understood. Using in vivo calcium imaging in behaving mice, we tracked single-cell STN activity across multiple behaviors, including locomotion, licking, and rewarddriven actions. Our results reveal that STN neurons exhibit mixed selectivity, encoding multiple behaviors with distinct temporal dynamics through both excitatory and inhibitory responses. These findings suggest a more complex functional role for the STN beyond simple motor control. Furthermore, population-level analyses demonstrate that STN activity robustly encodes motor parameters such as locomotion speed and licking intensity, potentially reflecting computational principles underlying behavioral modulation. We also compared neural representations in the STN to those in the adjacent zona incerta (ZI). While neurons in both regions encode locomotionrelated variables, ZI neurons exhibit more diverse calcium activity patterns, including longer event durations and weaker correlations with movement parameters. In contrast, STN neurons more faithfully encode motor states and display stronger contextual interactions across different behaviors. These findings highlight the overlapping yet distinct contributions of the STN and ZI in regulating motor and reward-related behaviors, offering new insights into their respective roles in basal ganglia circuits and their broader implications for motor control and reinforcement learning.







Current Position

Associate Professor, Department of Physiology, National Cheng Kung University

Education/Training

- 2015 PhD, Institute of Clinical Medicine, National Cheng Kung University
- 2010 MS, Institute of Clinical Medicine, National Cheng Kung University
- 2003 MD, College of Medicine, National Cheng Kung University

Professional and Research Experience

- 2021-2025 Clinical Associate Professor, Department of Plastic Surgery, National Cheng Kung University Hospital
- 2017-2019 Visiting Assistant Professor, Department of Bioengineering, UCLA
- 2003-2025 Physician, Department of Plastic Surgery, National Cheng Kung University Hospital

Awards and Honors

- 2024 國際傑出發明家 學術國光獎章
- 2023 未來科技獎
- 2022 國家新創獎

Modulating neuromuscular interface with electroceuticals: Feeding on demand

薛元毓 Yuan-Yu Hsueh Associate Professor, Department of Physiology, National Cheng Kung University

Neuromuscular junction (NMJ) dysfunction can occur after nerve injury, particularly injuries that affect the peripheral nervous system. When the motor nerves that innervate skeletal muscle are damaged, it can result in muscle weakness, atrophy, and even paralysis. Following nerve injury, the NMJ undergoes a series of changes that can contribute to dysfunction, including loss of synaptic architecture and neurotransmitters and maintaining the mechanism of the postsynaptic microenvironment of denervated skeletal muscle.

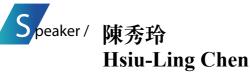
Electroceuticals, also known as bioelectronic medicine or neural engineering, refer to the use of electrical stimulation to modulate the function of the body's neural system for therapeutic purposes. Electroceuticals aim to treat various health conditions by interfacing with the body's nervous system, including the brain, spinal cord, and peripheral nerves, to regulate physiological processes such as pain perception, inflammation, and organ function. Electroceuticals can potentially play a role in promoting NMJ regeneration by modulating the activity of the motor neurons that innervate skeletal muscle. In this talk, I will briefly introduce our recent electroceutical strategy for NMJ regeneration. NMJ degradation is ameliorated with decreased muscle atrophy via direct distal nerve electrical stimulation. In addition, the skeletal muscle injury-associated genes are downregulated under feeding distal nerve electrical stimulation. Long-term functional improvement is achieved with increased nerve reinnervation and NMJ regeneration. Furthermore, electroceuticals also facilitate direct muscle neurotization in terms of NMJ regeneration at the denervated muscle. The strategy of electroceuticals provides promising benefits for improving neuromuscular interface regeneration via enhancing distal axon reinnervation per se.



中國生理學會 3/23 (Sun.) 16:06-16:30 1 樓,第二教室







Current Position

Department of Food Safety/Hygiene and Risk Management, College of Medicine, National Cheng Kung University

Education/Training

2004 PhD, Department of Basic Medicine, National Cheng Kuang University

Professional and Research Experience

- 2017-Present Vice-director, Research Center of Environmental Trace Toxic Substances
 2017 Director/Professor, Department of Food Safety/Hygiene and Risk Management, National Cheng Kung University
- 1998-2004 President, Taiwan Society of Indoor Environmental Quality (TSIEQ)

暴露農藥對於腸道微生物群及代謝體與腎臟功能下降之影響探討 Effects of exposure to pesticides on renal function, gut microbiota, and kidney function decline

陳秀玲 Hsiu-Ling Chen

Department of Food Safety/Hygiene and Risk Management, College of Medicine, National Cheng Kung University

Abstract Chronic kidney disease (CKD) and diabetic kidney disease (DKD) are major global health challenges, with nearly 50% of CKD patients also diagnosed with diabetes. Compared to CKD patients, those with DKD face a higher risk of progressing to dialysis or kidney transplantation, significantly reducing their quality of life and imposing substantial economic burdens on healthcare systems. Research suggests that short-term, high-level exposure to organophosphate and carbamate pesticides can induce glucose production by gut microbiota, leading to hyperglycemia. Pesticide exposure may also accelerate CKD progression by disrupting gut microbiota balance and exacerbating kidney damage. However, evidence on the underlying mechanisms of pesticide exposure in CKD patients remains limited. Therefore, CKD patients were enrolled, with their dietary patterns and lifestyle habits surveyed. Blood and urine samples were analyzed using targeted and non-targeted methods to measure metabolomics and pesticide levels as indicators. Then, we focused on investigating the correlations between pesticide exposure, gut microbiota composition, and metabolomics in CKD patients. Machine learning techniques was also applied to evaluate the predictive power of pesticide exposure, gut microbiota, and metabolomics for forecasting kidney function decline in CKD patients. The current study utilized UHPLC-Orbitrap-MS for untargeted metabolomics analysis, genomics analysis to investigate gut microbiota and LC-MS/MS to analyze targeted pesticides and oxidative damage biomarkers (8-OH-dG), aiming to identify integrated biomarkers for predicting kidney function decline in CKD patients. In the 98 CKD patients, enabling analysis of the relationship between pesticide exposure and metabolite concentrations. Additionally, potential biomarkers for pesticide exposure were identified, demonstrating the robust analytical capabilities in metabolomics analysis and implementing AI technology, bio-informatics in high-precision medicine of our team.



台灣毒物學學會 3/23 (Sun.) 13:00-13:30 2 樓, 29 教室





Current Position

Professor of the Institute of Toxicology, National Taiwan University, Taiwan

Education/Training

- PhD, Analytical and Environmental Sciences, King's College London, UK. 2011
- MS, Department of Chemistry, National Tsing Hua University, Taiwan. 2005
- 2002 BS, Department of Chemistry, National Taiwan Normal University, Taiwan.

Professional and Research Experience

- 2022-Present Professor, Institute of Toxicology, National Taiwan University, Taiwan
- 2019-2022 Associate professor, Institute of Toxicology, National Taiwan University, Taiwan
- 2016-2019 Associate professor, Institute of Forensic Medicine, National Taiwan University, Taiwan

Awards and Honors

- 2024 Young Scholar Award by the Taiwan Society for Mass Spectrometry.
- 2016 Supervisor of the Taiwan Academy of Forensic Sciences.
- 2020 Secretary-General of the Taiwan Academy of Forensic Sciences.

Wastewater-Based Epidemiology for Monitoring the Use of 68 NPS and Conventional Drugs in the Taipei Metropolitan Area, Taiwan, **During and After the COVID-19 Pandemic**

陳珮珊 Pai-Shan Chen Professor of the Institute of Toxicology, National Taiwan University, Taiwan

Amid the profound impacts of COVID-19 and associated social restrictions, this study applied wastewaterbased epidemiology (WBE) to monitor the use of 38 conventional drugs and 30 new psychoactive substances (NPS) in northern Taiwan. Daily wastewater samples were collected from four treatment plants in Taipei between September 2021 and January 2024. The timeline encompassed various phases, including nightclub reopenings, holidays, Lunar New Year, a localized COVID-19 outbreak, and regular periods, providing a comprehensive perspective on drug use patterns during and after the pandemic. In total, 31 drugs were identified, including five NPS. Notably, tramadol, zolpidem tartrate, CMA, and MDPV were detected in Taiwanese sewage for the first time, with detection frequencies ranging from 1.4% to 89.0%. Among conventional drugs, methamphetamine exhibited a detection frequency of 100%, indicating consistent daily consumption despite the restrictions imposed during the pandemic. This finding highlights the resilience of methamphetamine use, even under conditions that severely disrupted social and economic activities. Drug consumption patterns varied across the timeline. For example, methamphetamine and morphine usage declined during periods of nightclub closures but surged following their reopening, suggesting that access to these substances may have been limited during social restrictions. The consumption trend of methadone appeared to compensate for reduced morphine use, hinting at a substitution effect among opioid users. Meanwhile, ketamine and NPS displayed consistent usage patterns throughout the study period, reflecting the stable demand for these substances among certain user groups. NPS, often associated with party settings, were particularly affected by supply chain disruptions and enforcement complexities during the pandemic. Despite these challenges, their use persisted, although at fluctuating levels. Benzodiazepines, commonly coabused with synthetic cathinones in Taiwan, exhibited a contrasting trend to NPS. Their consumption aligned more closely with acetaminophen, potentially reflecting increased stress and anxiety levels during the pandemic. This correlation underscores the psychological toll of COVID-19 and the role of certain pharmaceuticals in coping with these effects. Another notable finding was the lack of significant differences in drug consumption between weekdays and weekends. Traditionally, recreational drug use spikes during weekends, driven by social gatherings and nightlife activities. However, the pandemic blurred these distinctions, with lockdowns and social restrictions disrupting conventional social rhythms. This shift suggests that the behavioral patterns of drug users adapted to the new normal imposed by the pandemic. This study underscores the utility of WBE as a real-time surveillance tool for monitoring drug use trends. By capturing a broad spectrum of substances, including emerging NPS, WBE provides valuable insights into the evolving landscape of drug consumption. The findings reveal not only the persistence of drug use despite social and economic disruptions but also the complex interplay between access, supply chain dynamics, and user behavior during and after the COVID-19 pandemic. Such data are crucial for informing public health strategies and tailoring interventions to address substance abuse in the postpandemic era.



台灣毒物學學會 3/23 (Sun.) 13:30-14:00 2 樓 · 29 教室





Current Position

Professor and Director, the Ph.D. program for Cancer Biology and Drug Discovery, China Medical University, Taiwan.

Education/Training

- 2007 OTHERS, Department of Molecular and Cellular Oncology, UT. M.D. Anderson Cancer Center, Houston, TX, USA
- 2006 OTHERS, Department of Pharmacology, National Taiwan University, Taipei, Taiwan
- 2004 PhD, Department of Pharmacology, National Taiwan University, Taipei, Taiwan

Professional and Research Experience

- 2024 -Present Chairman, Program for Cancer Biology and Drug Discovery, China Medical University, Taichung, Taiwan
- 2019-Present Associate Director, Graduate Institute of Biomedical Sciences, China Medical University, Taichung, Taiwan
- 2017-2022 Associate Dean, the Department of Research & Development, China Medical University, Taichung, Taiwan

Awards and Honors

- 2024 Potential Team of New Drug Development, Pitch Day, National Biotechnology Research Park.
- 2012 Teacher Award for Outstanding Teaching Performance, College of Medicine, China Medical University
- 2007 Young Scholar Award for Medical Research, Professor C. Y. Lee Foundation

The Impact of Environmental Pollutants on Tumorigenesis and Therapeutic Efficacy of Anti-Cancer Drugs

黃偉謙 Wei-Chien Huang Professor and Director, the Ph.D. program for Cancer Biology and Drug Discovery, China Medical University, Taiwan.

Environmental pollutants have emerged as critical factors influencing both the initiation and progression of cancer, as well as the efficacy of anti-cancer therapies. This talk will highlight recent findings on the impact of key environmental pollutants, including cigarette smoke, incense smoke, and particulate matter (PM2.5), on oncogenic pathways and therapeutic resistance in non-small cell lung cancer (NSCLC). These pollutants have been shown to activate pro-oncogenic signaling cascades, alter the tumor microenvironment, and reduce the effectiveness of standard anti-cancer drugs, posing significant challenges for treatment. In addition to lung cancer, our research explores the role of plasticizer exposure in the early onset of breast cancer, focusing on its effects on metabolic and immune dysregulation. Through a comprehensive analysis of these pathways, we have identified potential therapeutic targets that could pave the way for the development of novel and more effective treatment strategies for both NSCLC and breast cancer. By addressing the molecular underpinnings of pollutant-induced tumorigenesis and drug resistance, this presentation aims to shed light on innovative approaches to combat the dual threat posed by environmental toxins and cancer.



台灣毒物學學會 3/23 (Sun.) 14:00-14:30 2 樓,29 教室





Current Position

Director of Master Program in Clinical Genomics and Proteomics, College of Pharmacy, Taipei Medical University, Taiwan

Education/Training

- 2008 PhD, Department of Chemistry, National Taiwan Normal University
- 2002 BS, Department of Chemistry, Tunghai University

Professional and Research Experience

- 2021-Present Associate Professor, Master Program in Clinical Genomics and Proteomics, College of Pharmacy, Taipei Medical University, Taiwan
- 2021-Present Adjunct Associate Professor, Department of Pharmaceutical Sciences, Taipei Medical University, Taiwan
- 2021-Present Adjunct Associate Professor, Ph.D. Program in Biotechnology Research and Development, Taipei Medical University, Taiwan

Awards and Honors

- 2022 Young Scholarship Research Award, Taiwan Mass Spectrometry Society, Taiwan
- 2017 C-HPP Young Investigator Award, the 16th Human Proteome Organization World Congress, Dublin, Ireland
- 2015 Poster Award, Third Prize, 2015 International Conference on Advanced Translational Research in Food Science, Environmental Toxicology and Cancer Biology, Taipei, Taiwan

Differential proteomic profiles of lung injury in rat models upon pulmonary exposure to air pollution

韓嘉莉 Chia-Li Han

Director of Master Program in Clinical Genomics and Proteomics, College of Pharmacy, Taipei Medical University, Taiwan

Chronic obstructive pulmonary disease (COPD) is one of the major causes of morbidity and mortality globally. Inhalation of particulate matter (PM) air pollution has been studied to closely associate with COPD. However, the pathogenesis mechanisms underlying PM2.5-induced lung injury is largely unknown, leading to the poor stratification and treatment of the disease. Thus, we aim to explore the underlying molecular mechanisms associated with PM-mediated lung injury by quantitative proteomics analysis of lung tissues from ageing and young rat models with whole body exposure to traffic-related PM pollutants and compared it with that in rat models exposed to high-efficiency particulate air-filtered gaseous pollutants. Our data showed that before lung function decline the 0.5-yr rats had exhibited differential dysregulation of proteins involved in oxidative stress, cellular metabolism, calcium signalling, inflammatory responses, and actin dynamics under exposures to PM and gaseous pollutants. On the contrary, more significant and consistent molecular effects were observed in 1.5-yr rats exposed to PM and gaseous pollutants, of which the malignancy-related ERB signalling pathways were activated additionally in PM-exposed ageing rats. Based on our data, we proposed a detailed pathogenic mechanism to depict temporal and dynamic molecular regulations associated with PM- and gaseous pollutants-induced lung injury. We expect that our findings would provide valuable information towards progression of air pollution-caused lung injury and serve as a repository to search for potential draggable targets.



研討曾演講 Symposia





Current Position

Associate Professor, School of Food Safety, College of Nutrition, Taipei Medical University

Education/Training

- 2015 PhD, Department of Biomedical Engineering and Environmental Sciences, National Tsing Hua University, Taiwan
- 2009 MS, Department of Biomedical Engineering and Environmental Sciences, National Tsing Hua University, Taiwan.
- 2007 BS, Department of Chemistry, National Taiwan Normal University, Taiwan.

Professional and Research Experience

- 2016-2017 Postdoctoral research, Institute of Toxicology and Genetics, Karlsruhe Institute of Technology, Germany
- 2013-2014 Visiting PhD student, Department of chemicals and product safety, Federal Institute for Risk Assessment (BfR), Germany

Awards and Honors

Postdoctoral Research Abroad Program, Ministry of Science and Technology (MOST), Taiwan Xin Tian Temple long term scholarship, Taiwan

German Academic Exchange Service (DAAD)-MOST sandwich program for PhD candidates

Detecting fluorescent-labeled nanoplastics in digestive fluids and tissue using Nano-tracking analysis and near-infrared fluorescence imaging

蕭伊倫 I-Lun Hsiao Associate Professor, School of Food Safety, College of Nutrition, Taipei Medical University

Abstract Humans may inevitably be exposed to nanoplastics (NPIs) through ingestion. The aggregation state of NPIs significantly influences their absorption efficiency, so understanding behaviors of NPIs during digestion, both in the presence or absence of food matrix is vital for risk assessment. On the other hand, in order to obtain results for different time points in toxicokinetics and tissue distribution studies, previous research has typically required the use of large numbers of animals. In accordance with the 3R principle, a novel methodology that minimizes animals use is imperative. In our recent studies, commercial fluorescent-labeled NPIs were employed to characterize the size in both artificial and real digestive fluids using a Nano-tracking analysis fluorescence model, and proved that NPI sizes in artificial digestive fluids were underestimated. A near-infrared (NIR) fluorescence contrast agent was labeled in a polyethylene terephthalate (PET) NPI and utilized for real-time in vivo tracking of the NPIs. This presentation will demonstrate how accurate tracking of fluorescent-labeled NPIs in complex biological matrices can be achieved by avoiding autofluorescence of proteins and scattering of solid matrices. Reference: 1. Lee, G., Jhang, Y.J., Jhang, Y.T., Chang, Y.C., Chang, H.W., Chuang, C.Y., Chuang, Y.K., Lin, C.W., Hsiao, I.L.* (2024) Artificial digestion represents the worst-case scenario for studying nanoplastic fate in gastrointestinal tract. Journal of Hazardous Materials, In Press.



台灣毒物學學會 3/23 (Sun.) 15:00-15:30 2 樓, 29 教室





Current Position

Charles Howard Candler Professor, Emory University, USA Director, Emory Vaccine Center, Emory University School of Medicine, USA

Education/Training

- 1972 BS, Idaho State University, Pocatello, ID
- 1974 MS, Idaho State University, Pocatello, ID
- 1981 PhD, Harvard University, Cambridge, MA

Professional and Research Experience

1995Present Georgia Research Alliance Eminent Scholar in Vaccine Research

- 1995Present Professor, Microbiology and Immunology, Emory University School of Medicine, Atlanta, Georgia,
- 1992-1995 Professor, Department of Microbiology & Immunology, UCLA School of Medicine, Los Angeles, California,

Awards and Honors

- 2022 Class of Fellows of the Academy of Immuno-Oncology (SITC)
- 2021 Member of American Academy of Arts and Sciences
- 2020 Distinguished Fellow of American Association of Immunologists (AAI)

What is T cell exhaustion

Rafi Ahmed Director, Emory Vaccine Center, Emory University

T-cell exhaustion is a phenomenon characterized by stepwise and progressive loss of T-cell functions that arises from chronic antigen exposure. T cell exhaustion was first defined in the mouse model of chronic lymphocytic choriomeningitis virus (LCMV) infection. During chronic antigen stimulation, exhausted T cells fail to differentiate into functional memory cells, possess poor effector function, reduced proliferation and sustained expression of several inhibitory receptors. These T cells acquire a transcriptional and epigenetic state that is distinct from functional effector or memory T cells. Exhaustion prevents optimal tumor control and adequate immune response to infections. High levels of programmed death- 1 (PD-1) expression is one of the hallmarks of exhausted T cells. PD-1 targeting therapy reinvigorates the exhausted CD8 T cells which is instrumental in controlling virus and tumor burden. In the last two decades, therapeutics targeting the PD-1 signaling pathway has been highly successful in the treatment of people living with cancer.

A subset of "exhausted" CD8 T cells possess high proliferative capacity and is identified as **PD-1**⁺**TCF-1**⁺**TOX**⁺ **stem-like CD8 T cells**. These cells play a major role in sustaining CD8 T cell responses during chronic viral infection and cancer. These quiescent stem-like CD8 T cells can be generated as early as day 5 after LCMV infection regardless of acute or chronic infection and serve as the precursors of exhausted CD8 T cells. **Stem-like CD8 T cells provides the proliferative burst after PD-1 targeted therapy and is critical for the reinvigoration of exhausted CD8 T cells**. Better understanding of the biology of stem-like CD8 T cells will lead to the development of novel therapeutics and have significant implications in immunotherapy; particularly in the optimizing checkpoint blockade strategies to reinvigorate exhausted T cells.



免疫學會X細分學會合辦 3/23 (Sun.) 13:10-14:10 3 樓, 30 教室 免疫學會X細分學會合辦 3/23 (Sun.) 13:10-14:10 3 樓, 30 教室

何謂T細胞耗竭

JACBS

Rafi Ahmed 美國艾莫瑞 (Emory) 大學疫苗中心主任

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當CD8 T細胞因病毒感染後長期處於抗原暴露(antigen exposure),漸次失去清除這些受感 染細胞的能力,稱為T細胞耗竭(T cell exhaustion)。此現象最早發現於脈絡叢腦膜炎病毒 (lymphocytic choriomeningitis virus, LCMV)感染小鼠實驗,當小鼠受抗原的長期刺激, 導致T細胞無法分化至具完整免疫功能的「記憶型T細胞」(memory T cells), 而成為免疫功 能低下(poor effector function)與細胞增生力降(reduced proliferation)的耗竭T細胞 (exhausted T cells) •

為何這些 T 細胞會耗竭?仔細分析耗竭 T 細胞膜上的受體 (receptors),發現有一些受體會 抑制 T 細胞分化,也恰是免疫系統查核點,例如T 細胞上的 CTLA-4、PD-1、LAG-3、TIM-3 等受體。原來是這些細胞進入耗竭狀態時,由 DNA 走向 RNA 的轉錄狀態 (transcriptional state)和表觀遺傳狀態 (epigenetic state)[即基因的功能改變], 導致耗竭 T細胞已有別於 「效能型T細胞」(effector T cells)或「記憶型 T細胞」。事實上,耗竭 T細胞的這些抑制 受體會致該細胞無法辨識抗原,猶如視而不見,導致免疫武功驟降而患者病況加重。科學 發現其機轉是耗竭 T細胞高度表現如「程式死亡分子 -1」(programmed cell death protein-1, PD-1) [註 1] 的標誌。此後,針對 PD-1 的治療研究可重振 CD8+T 細胞毒殺病毒感染細胞與 癌細胞的能力;即以 PD-1 的訊息途徑已掀起免疫治療的新曙光 [註 2]。

有趣的是近來研究發現有一群 T 細胞被稱為 PD1⁺TCF-1⁺Tox⁺ CD8 T 幹細胞, 在病毒持續 感染和癌症中扮演著維持 CD8 T 細胞功能的極重要的角色,這一些靜止的 CD8 T 幹細胞 無論是在 LCMV 急性或慢性 [註 3] 的感染第 5 天即出現, 作為耗竭 T 細胞的前驅細胞 (precursor cells)。因此,耗竭 CD8T 細胞的幹細胞 (Stem-like CD8 T cells) 在 PD-1 免疫治療 後,提供爆發式的 CD8 T 細胞增殖,極關鍵地扭轉原已一蹶不振的 CD8+T 細胞恢復並維持 其原有的免疫功能。

未來更深入明瞭耗竭 CD8T 細胞的幹細胞生物特性,如妥用不同免疫查核點的最佳化治療 **策略**,將導引**免疫治療**的新里程碑。

[註1]:日本京都大學特聘教授本庶佑 (Tasuku Honjo) 的研究團隊,在 1992 年著手研究「程 序性細胞死亡」(programmed cell death)的機制,這是一種細胞自殺以維持體內恆定。他 們發現 T 和 B 細胞在走向死亡時,會誘發一蛋白 PD-1 (programmed cell death-1)。後又發 現缺乏 PD-1 基因時,小鼠會表現許多發炎症狀,但卻對病毒有較強的抵抗力,因此思考 PD-1 是

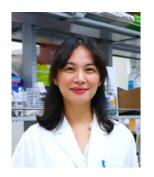
否和免疫機制有關。本庶佑和艾利森 (James Allison) 榮獲臺灣 2014 年唐獎之生技醫藥獎和 2018 年諾貝爾生理醫學獎,表彰他們各發現 T 細胞表面的兩免疫查核點抑制因子 CTLA-4 和 **PD-1**的卓越貢獻。

[註 2]: 在發現 PD-1 的七年後,也發現 PD-1 的配體 (ligand) PD-L1。即 PD-1 是 T 細胞的一「煞 車鍵」,而 PD-L1 是啟動煞車的開關。當 PD-L1 與 T 細胞表面的 PD-1 結合,會抑制 T 細胞 的活化。自此發展免疫抑制劑或單株抗體(如 anti-PD-1 antibody),免疫治療(immunotherapy) 可有很大的臨床應用,如應用於愛滋病毒(HIV-1)、B型與C型肝炎病毒(HBV and HCV)所造 成慢性發炎、癌症的 T 細胞衰弱,均可經由這些免疫抑制劑或單株抗體達到臨床治療效果。 因此若經由 anti-PD-1 抗體的幫助,能調整 T 細胞分化方向,重新活化找回具有正常功能的 T 細胞,提供治療的全新視野,極具潛力以**免疫治療**的新作法,以面對不同疾病所帶來 T 細胞 問題,這二十多年來也有很多成功案例與研究,為治療帶來新曙光。

[註 3]:LCMV 急性感染與慢性感染是由不同的病毒株感染所造成的兩種結果: LCMV 阿姆斯壯病毒株 (Armstrong strain) 感染實驗小鼠會造成急性感染 LCMV 科降 13 病毒株 (Clone 13 strain) 感染實驗小鼠會造成慢性感染









Current Position Professor, Institute of Microbiology and Immunology, NYCU, Taiwan

Education/Training 2007 PhD, Duke University, U.S.A.

Professional and Research Experience

2025-Present Professor, Institute of Microbiology and Immunology, NYCU, Taiwan
2022-Present Deputy Director, Laboratory Animal Center, NYCU, Taiwan
2022-2023 Vice Secretary General, Chinese Society of Immunology, Taiwan

Awards and Honors

- 2023 Fellow of Higher Education Accreditation (FHEA)
- 2022 Chinese Society of Immunology Outstanding Research Award
- 2019 Ta-You Wu Memorial Award

The Contribution of Lysosomal Metabolite Transporter, ENT3, to the Immune Responses

徐嘉琳 Chia-Lin Hsu Professor, Institute of Microbiology and Immunology, NYCU, Taiwan

Equilibrative nucleoside transporter 3 (ENT3) is a lysosomal metabolite transporter that facilitates intracellular nucleoside translocation. This talk will discuss its role in immune cells and potential involvement in disease settings.



中華民國細胞分子生物學學會 3/23 (Sun.) 15:20-15:50 3 樓, 30 教室





Current Position

Associate Investigator, Immunology Research Center, National Health Research Institutes, Taiwan

Education/Training

- 2008 PhD, Institute of Basic Medical Sciences, National Cheng-Kung University, Taiwan
- 2003 MS, Graduate Institute of Pathology, National Taiwan University, Taiwan
- 2001 BS, Department of Botany, National Taiwan University, Taiwan

Professional and Research Experience

- 2022-Present Associate Investigator, Immunology Research Center, National Health Research Institutes, Taiwan
- 2015-2022 Assistant Investigator, Immunology Research Center, National Health Research Institutes, Taiwan
- 2008-2014 Postdoctoral Fellow, Immunology Research Center, National Health Research Institutes, Taiwan

Awards and Honors

- 2019 57th Ten Outstanding Young Persons (Taiwan) 第 57 屆十大傑出青年獎 醫學研究類
- 2018 Ta-You Wu Memorial Award from Ministry of Science and Technology (科技部吳大猷先 生紀念獎)
- 2017 President Rey-Shyong Tsai Outstanding Paper Award in Metabolism and Nephrology (第一屆蔡瑞熊校長優秀研究論文獎)

MAP4K3/GLK in Inflammation and Aging

莊懷佳 Huai-Chia Chuang Associate Investigator, Immunology Research Center, National Health Research Institutes, Taiwan

MAP4K3 (also named GLK) belongs to the mammalian Ste20-like kinase family. GLKoverexpressing T cells are correlated with multiple human autoimmune diseases including systemic lupus erythematosus (SLE). GLK directly phosphorylates and activates PKC θ , leading to activation of IKK/NF-κB in T cells. GLK-deficient mice display impaired T-cell-mediated immune responses or autoimmune diseases. GLK signaling selectively stimulates IL-17A production in T cells by inducing AhR-RORyt complex and their nuclear translocation. In contrast, GLK signaling inhibits Foxp3 transcription by blocking the function of FoxO1. Collectively, GLK signaling induces IL-17A transcription and inhibits Foxp3 transcription, leading to induction of Th17 differentiation and reduction of Treg differentiation. Thus, GLK inhibitors could be more effective than IL-17A blockade for treatment of autoimmune diseases. Furthermore, we found that 39% SLE patients harbor GLK germline or somatic variants, which cause increased of GLK mRNA/protein levels. Recently, we identified a novel protein-coding gene, UHRF1P, as a SLEspecific transcript by three machine learning (AI) statistical methods. Remarkably, UHRF1P induction blocked the interaction between GLK and its E3 ubiguitin ligases (MKRN4 and UHRF1), leading to GLK overproduction. Besides T cells, we found that GLK is induced in epithelial cells and macrophages of human COVID-19 patients, as well as tissues of lung cancer and liver cancer. GLK directly phosphorylates and stabilizes ACE2 proteins, and GLK-induced ACE2containing exosomes are important pathogenic factors for COVID-19. In cancer cells, GLK directly phosphorylates and activates IQGAP1, resulting in induction of Cdc42-mediated cell migration and cancer metastasis. Taken together, GLK is a therapeutic target for inflammatory/autoimmune diseases and cancer recurrence. Interestingly, GLK also regulates animal lifespan. GLK deficiency in Caenorhabditis elegans results in an expansion of the worm lifespan. Similarly, GLK-deficient mice show a significant extension of lifespan. The serum levels of proinflammatory cytokines are increased in aged wild-type mice, but are decreased in aged GLK-deficient mice. Chronic inflammation plays a critical role in the aging process. Thus, expanded lifespan of GLK-deficient mice may be due to decreased inflammatory responses (inflamm-aging). These findings suggest that GLK inhibitors may be used as prophylactic agents to suppress inflamm-aging.



中華民國細胞分子生物學學會 3/23 (Sun.) 15:20-16:20 3 樓, 30 教室





Current Position

Associate Research Fellow, Institute of Molecular Biology, Academia Sinica, Taiwan

Education/Training

- 2008 PhD, Division of Biological Sciences, University of California, San Diego
- 2001 MS, School of Cognitive and Computer Sciences, University of Sussex
- 1998 BS, Department of Zoology, National Taiwan University

Professional and Research Experience

- 2016-2024 Assistant Research Fellow, Institute of Molecular Biology, Academia Sinica
- 2011-2016 Postdoc, Department of Systems Biology, Harvard Medical School
- 2010-2011 Postdoc, Department of Cellular and Molecular Pharmacology University of California, San Francisco

Awards and Honors

- 2020 傑出人才基金會積極留任國內優秀學者獎
- 2013 Ruth L. Kirschstein National Research Service Award NIGMS
- 2001 Distinction M.Sc. honor degree, University of Sussex

Nature as a great sculptor — a lesson from ferroptotic trigger waves

陳昇宏 Sheng-Hong Chen Associate Research Fellow, Institute of Molecular Biology, Academia Sinica, Taiwan

Large-scale cell death is commonly observed during organismal development and in human pathologies1,2,3. These cell death events extend over great distances to eliminate large populations of cells, raising the question of how cell death can be coordinated in space and time. One mechanism that enables long-range signal transmission is trigger waves6, but how this mechanism might be used for death events in cell populations remains unclear. Here we demonstrate that ferroptosis, an iron- and lipid-peroxidation-dependent form of cell death, can propagate across human cells over long distances (\geq 5 mm) at constant speeds (around 5.5 μ m/min) through trigger waves of reactive oxygen species (ROS). Chemical and genetic perturbations indicate a primary role of ROS feedback loops (Fenton reaction, NADPH oxidase signalling and glutathione synthesis) in controlling the progression of ferroptotic trigger waves. We show that introducing ferroptotic stress through suppression of cystine uptake activates these ROS feedback loops, converting cellular redox systems from being monostable to being bistable and thereby priming cell populations to become bistable media over which ROS propagate. Furthermore, we demonstrate that ferroptosis and its propagation accompany the massive, yet spatially restricted, cell death events during muscle remodelling of the embryonic avian limb, substantiating its use as a tissue-sculpting strategy during embryogenesis. Our findings highlight the role of ferroptosis in coordinating global cell death events, providing a paradigm for investigating large-scale cell death in embryonic development and human pathologies.



中華民國細胞分子生物學學會 3/23 (Sun.) 16:20-16:50 3 樓, 30 教室





Current Position

Assistant Professor, Institute of Anatomy and Cell Biology, College of Medicine, National Yang Ming Chiao Tung University, Taiwan

Education/Training

PhD, Biomedical Engineering, National Yang Ming University, Taipei, Taiwan

Professional and Research Experience

- 2024-Present Assistant Professor, Institute of Anatomy and Cell Biology, College of Medicine, National Yang Ming Chiao Tung University, Taipei, Taiwan
- 2022-2024 Assistant Professor, Department of Anatomy and Cell Biology, School of Medicine, National Yang Ming Chiao Tung University, Taipei, Taiwan
- 2016-2022 Postdoctoral Fellow, Institute of Cellular and System Medicine, National Health Research Institutes, Zhunan, Taiwan

Awards and Honors

- 2022 財團法人沈力揚教授醫學教育獎學紀念基金會講師級研究與進修獎助
- 2021 Postdoctoral Researcher Academic Research Award, Ministry of Science and Technology (MOST)
- 2018 Excellent Paper (Oral Presentation) Award, 2018 National Health Research Institutes Research Day

3D 列印技術在解剖學教學之應用

蔣偉程 Wei-Cheng Jiang Assistant Professor, Institute of Anatomy and Cell Biology, College of Medicine, National Yang Ming Chiao Tung University, Taiwan

Advancements in 3D printing technology have revolutionized educational methodologies across disciplines. In anatomy education, traditional approaches relying on cadaver dissection and 2D illustrations often present challenges in accessibility, ethical concerns, and comprehension of complex structures. In contrast, customized 3D-printed anatomical models provide visual and tactile representations of human structures, creating a more interactive and inclusive learning experience. These models offer accurate replications of organs and systems, enabling students to examine spatial relationships and intricate details that are difficult to visualize using conventional methods. Moreover, 3D printing facilitates the creation of pathology-specific models, aiding in the contextualization of clinical scenarios and bridging the gap between theory and practice. Additionally, these tools are cost-effective and reusable, making them suitable for institutions with limited access to cadaveric specimens. The integration of 3D bioprinting technologies holds the potential to simulate physiological functions, further advancing the scope of anatomy education and enhancing its future relevance to clinical practice.



中華民國解剖學學會 3/23 (Sun.) 13:00-13:30 3 樓, 32 教室





Current Position

Assistant Professor, Anatomy, School of Medicine, Fu-Jen Catholic University

Education/Training

- 2006 PhD, Department of Anatomy and Cell Biology, College of Medicine, National Taiwan University, Taiwan
- 1998 MS, Department of Anatomy and Cell Biology, College of Medicine, National Taiwan University, Taiwan
- 1992 BS, Biology, Fu Jen University, New Taipei City, Taiwan

Professional and Research Experience

2006-Present Assistant Professor, Fu Jen University

- 2001-2005 Teaching Assistant, National Taiwan University
- 2000-2001 Anatomy Lecturer, Chang Gung University

3D printing in Anatomy Education

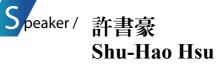
鍾敦輝 Tun Hui Chung Assistant Professor, Anatomy, School of Medicine, Fu-Jen Catholic University

3D printing technology has been making much progress and is actively applied at all levels recently. DICOM (Digital Imaging and Communications in Medicine) data donated by the cadavers are used to build a 3D brain blood vessel database, and then print the 3D blood vessel structures to the medical student in gross anatomical teaching at Fu Jen Catholic University. By using the 3D software to analyze and create the gross brain blood vessel data and STL (STereo Lithography) output for we using a 3D printer to print blood vessel structures and applying them to the teaching of medical gross anatomy experiments. We used a 3D scanner for the anatomical models and create the 3D files to upload them to the Sketchfab website for online browsing. Based on these online materials, brain slices teaching video, we design an online laboratory for teaching neuroanatomy and gross anatomy experiments of the School of Medicine. We conduct questionnaires to evaluate effectiveness of the neuroanatomy online laboratory we designed to learn. The questionnaire shows that students generally agree that 3D software, 3D scanning or 3D printing are helpful for anatomy courses. We hope that we can increase the amount of 3D anatomy database using a 3D scanner, and continue to optimize the database and the anatomy teaching website. Students can even design, construct and print their own anatomical models to learn and can continuously develop, add 3D printing model to assist and increase students' interest in learning. In the future, we will try to cooperate with the Computer Tomography Machine of the Institute of Forensic Medicine, National Taiwan University School of Medicine. We will establish a computer tomography database for the anatomy teacher, and provide DICOM support for human anatomy images in the Fu Jen Catholic University and Hospital.



中華民國解剖學學會 3/23 (Sun.) 13:30-14:00 3 樓,32 教室





Current Position

副教授國立臺灣大學醫學院解剖所暨細胞生物學(科)研究所

Education/Training

- PhD, Molecular, Cellular and Developmental Biology, THE OHIO STATE UNIVERSITY 2012
- 2003 MS, Department of Anatomy and Cell Biology, College of Medicine, National Taiwan University, Taiwan
- BS, Zoology, National Taiwan University, Taiwan 2001

Professional and Research Experience

- 2023-Present Associate Professor, Dept. of Anatomy and Cell Biology, National Taiwan University, Taipei, Taiwan.
- 2016-2023 Assistant Professor, Dept. of Anatomy and Cell Biology, National Taiwan University, Taipei, Taiwan.
- Postdoctoral Associate, Dept. of Pathology, University of Pittsburgh, Pittsburgh, PA. 2013-2015

Decoding the Body: The Advantages and Limitations of Virtual Reality in Anatomy Education

許書豪 Shu-Hao Hsu 副教授國立臺灣大學醫學院解剖所暨細胞生物學(科)研究所

Virtual reality (VR) anatomy software offers numerous advantages for anatomy education, enabling students to achieve a deeper and more intuitive comprehension of human anatomy. VR technology overcomes the spatial and resource limitations of traditional anatomy education, enabling students to engage in anatomical studies anytime and anywhere without needing a physical laboratory or special equipment. Over the past year, VR anatomy equipment has been integrated with various approaches into the Gross Anatomy Lab course. With real-time projecting and recording functions, students collaboratively created and recorded instructional videos on anatomical structures. Also, students were guided to use VR equipment to perform cross-sections of the human body and match the structures in plastinated cross-sectional cadaveric specimens. This 'slicing' function is a significant advantage of VR software; compared to traditional physical dissections, VR software allows students to explore various cross-sections at any time. In addition to exploring gross anatomy, instructors can utilize the software's exam feature to conduct virtual "station rounds" for identifying specific structures. Through cloud-based data analysis, teachers can track students' test scores and understand their learning progress. However, several drawbacks of VR anatomy need to be addressed to apply this new technology in anatomy education continuously. First, the overall pricing of most systems is too expensive to increase the headset-to-student ratio, which is critical for students to practice virtual dissections frequently. Second, it is challenging to fine-tune or troubleshoot the VR settings without the manufacturer's help. Third, new course design ideas generated from the teaching experience are difficult for the manufacturer to produce due to the cost, which the academy possibly underestimates. In conclusion, a mutually beneficial collaboration between the manufacturer and the school is urgently needed to overcome these issues and turn VR anatomy into an indispensable tool in gross anatomy education in the future.



3/23 (Sun.) 14:00-14:30 3 樓,32 教室





Current Position

Associate Professor, Department of Anatomy and Cell Biology, School of Medicine, College of Medicine, Taipei Medical University

Education/Training

PhD, Department of Anatomy and Cell Biology, College of Medicine, National Taiwan University, Taiwan

MS, Department of Anatomy and Cell Biology, College of Medicine, National Taiwan University, Taiwan

BS, Department of Nutrition, College of Health Care and Management, Chung Shan Medical University

Professional and Research Experience

- 2022-Present Associate Professor, Department of Anatomy and Cell Biology, School of Medicine, College of Medicine, Taipei Medical University
- 2012-2022 Assistant Professor, Department of Anatomy and Cell Biology, School of Medicine, College of Medicine, Taipei Medical University
- 1999-2012 Instructor, Department of Anatomy and Cell Biology, School of Medicine, College of Medicine, Taipei Medical University

Awards and Honors

- 2024 教學實踐研究計畫傳習教師
- 2022 年度教學表現優異獎第一名
- 2022 傑出優良教師

Redesigning a Flipped Classroom Course and Evaluating Effectiveness in Medical Education: Case Study of the Course of "Anatomy"

陳淑華 Seu-Hwa Chen Associate Professor, Department of Anatomy Medicine, Taipei Medical University

Virtual reality (VR) technology has been used in medical education and anatomy learning. First, we took students taking anatomy courses at medical universities as subjects to explore the correlation between the application of VR technology and students' learning achievements. The results showed that participants' learning performance after using VR had a significantly positive correlation with the frequency of VR control and the degree of engagement when using VR. Then the final spatial ability and anatomy laboratory scores increased significantly under the intervention of VR in anatomy learning, but it did not affect the anatomy lecture score. Therefore, besides integrating virtual reality tools into classroom instruction for the past five years, increasing opportunities for students to use the 3D organon anatomy app after class to improve learning performance in the anatomy lecture. Furthermore, the exam pass rate of participants in the School of Medicine was studied in the flipped classrooms and VR innovative courses "Skeleton-Muscular System". Compared with participants in lecture-based teaching, the pass rate was significantly increased in the "remember," "analyze," and "apply" types of questions. Moreover, in the middle-scoring group and low-scoring group, the pass rate of participants in the types of "analyze" and "apply" questions has been significantly improved. According to the analysis of Spearman correlation coefficients, the pass rate in "remember," "understand, "analyze," and "apply" questions has a moderate positive correlation with the bell-ringer lab exam scores of the gross anatomy laboratory. Further analysis of the pass rates of the high-, middle-, and lowscoring groups on guestions of different difficulty (level 1: easy, level 2: medium, level 3: difficult). Results show the pass rate of the high- and middle-scoring groups in the midterm exam has no statistical significance, but they are both significantly higher than those in the low-scoring group. In the final exam, pass rates on level 1 and level 3 guestions of the middle- and low-scoring groups have significantly increased than the midterm exam. The better pass rate in the highscoring group is the level 2 and 3 questions. Based on the above research results, integrating virtual reality tools into anatomy instruction may increase students' spatial abilities to affect their learning achievements in anatomy lectures and laboratories, and improve retention learning.



中華民國解剖學學會 3/23 (Sun.) 14:30-15:00 3 樓,32 教室

Associate Professor, Department of Anatomy and Cell Biology, School of Medicine, College of





Current Position

Director, Biomedical Translation Research Center, Academia Sinica, Taiwan Distinguished Research Fellow, Institute of Cellular and Organismic Biology, Academia Sinica

Education/Training

1993 PhD, Institute of Pathology, College of Medicine, National Taiwan University

Professional and Research Experience

2020-Present Distinguished Research Fellow, Institute of Cellular and Organismic Biology, Academia Sinica

2019-Present Director, Biomedical Translation Research Center, Academia Sinica, Taiwan

Awards and Honors

- 2011 NSC Outstanding Research Award, National Science Council, Taiwan (twice, in 2011-2014 and 2015-2018.)
- 2018 The Executive Yuan Award for Outstanding Science and Technology Contribution
- 2020 The 17th National Innovation Award- Excelsior Award

Epithelial cell adhesion molecule-targeted niche therapy attenuates Wnt signaling to suppress colorectal cancer stemness

吳漢忠 Han-Chung Wu

Director, Biomedical Translation Research Center, Academia Sinica, Taiwan, Distinguished Research Fellow, Institute of Cellular and Organismic Biology, Academia Sinica

Cancer stem cells (CSC) are widely implicated in tumorigenesis and cancer re-occurrence, but the development of therapeutics to target CSCs remains a challenge due to their plasticity. Nevertheless, CSCs in colorectal cancer (CRC) highly express epithelial cell adhesion molecule (EpCAM) and are dependent on Wnt signaling for their function. To simultaneously target EpCAM and Wnt signaling, we combined our EpCAM-neutralizing antibody, EpAb2-6 (NCT05687682), with a porcupine inhibitor (LGK974) in a clinically feasible 'niche therapy' for the treatment of CRC. Patient-derived tumor-organoids (PDTOs), xenografts (PDX), CSC-derived models and tissue arrays obtained from patients were utilized. Therapy-induced gene expression changes were studied by RNAseg analysis. CSC-related mechanisms and niche-factor inhibition were assessed using stemness assays, analysis of tumor interstitial fluid, and super resolution microscopy. Therapeutic efficacy was tested in patient/CSC-derived animal models. The combination therapy attenuated Wnt signaling and targeted CSC properties, even in KRAS-mutant patient samples. At a molecular level, cleaved extracellular domain of EpCAM (EpEX) was enriched in the tumor microenvironment and mimicked natural Wnt ligands by directly interacting with Wnt receptors to induce signaling. Activated Wnt signaling induced ADAM17/TACE, augmenting shedding of EpEX in a positive feedback-loop. Ultimately, the therapy depleted EpEX enrichment and consequent Wnt-related activity, inhibiting cancer stemness. When tested in multiple patient/CSC-derived, metastatic and orthotopic models, the combined therapy halted cancer progression and prolonged animal survival. In conclusions, EpCAM promotes cancer stemness by stimulating Wnt signaling via the action of EpEX as niche factor. Therefore, EpAb2-6-based niche therapy may target CSCs and prove beneficial for treatment of CRC, including KRAS mutant disease.



台灣生物化學及分子生物學學會 3/23 (Sun.) 13:30-14:30 3 樓, 33 教室





Current Position

Director, Biotechnology Center, National Chung Hsing University, Taiwan Distinguished Professor, Institute of Genomics and Bioinformatics, National Chung Hsing University, Taiwan

Education/Training

- 2003 PhD, Institute of Biochemical Sciences, National Taiwan University
- 1999 MS, Graduate Institute of Biochemistry, National Chung Hsing University, Taiwan
- 1997 BS, Department of Food Science, Fu Jen Catholic University, Taiwan

Professional and Research Experience

- 2022-2023 Associate Dean, College of Life Sciences, National Chung Hsing University, Taiwan
- 2014-2020 Director, Institute of Genomics and Bioinformatics, National Chung Hsing University, Taiwan
- 2013-Present Professor, Institute of Genomics and Bioinformatics, National Chung Hsing University, Taiwan

Awards and Honors

- 2020 17th National Innovation Award for development of broad spectrum antiviral drug against coronaviruses
- 2020 Ministry of Science and Technology (MOST) Outstanding Research Award
- 2015 Young Scientist Research Award, Tien-Te Lee Biomedical Foundation

New Strategies for Targeting Functional DNAs with Small Molecules in Cancer Therapy

侯明宏 Ming-Hon Hou

Director, Biotechnology Center, National Chung Hsing University, Taiwan, Distinguished Professor, Institute of Genomics and Bioinformatics, National Chung Hsing University, Taiwan

Cancer remains one of the leading causes of mortality worldwide, highlighting the urgent need for innovative and targeted therapeutic strategies. Traditional chemotherapy is often constrained by severe off-target effects, drug resistance, and dose-dependent toxicity. Recent advances in DNA-targeting small molecules provide new opportunities to selectively modulate key genomic elements involved in oncogenesis. In this study, we present two complementary DNA intercalation strategies that enhance anticancer specificity and efficacy by exploiting distinct structural features of DNA. The first approach employs a dual-binding site intercalation strategy, in which actinomycin D (ActD) and doxorubicin (Dox) exhibit synergistic binding to consecutive GCCG motifs within GC-rich promoters, such as the epidermal growth factor receptor (EGFR) promoter. High-resolution X-ray crystallography reveals that ActD intercalates at 5'-GC sites, inducing local conformational changes that optimize Dox binding at adjacent 5'-CG sites via stacking interactions, hydrogen bonding, and drug-drug cooperativity. This cooperative binding mode stabilizes GCCG-rich DNA sequences, enhancing sequence selectivity and reducing offtarget interactions. Functional studies in breast cancer models confirm that this combination effectively downregulates EGFR expression, leading to significant tumor suppression. The second strategy focuses on bis-intercalators, a class of DNA-targeting agents that induce topological alterations by bridging adjacent DNA duplexes. Using a tetraplex base-pair junction model, we demonstrate that bis-intercalators DA4 and DA5 selectively cross-link DNA at CpGrich junctions, transforming B-DNA into an overwound A-DNA-like conformation, which disrupts topoisomerase II function. Structural analysis reveals that DA5, with its optimized flexible linker, aligns its chromophores with CpG sites, facilitating continuous stacking and water-mediated hydrogen bonding. This structural perturbation enhances DNA stabilization and anticancer efficacy, as demonstrated in SW620 xenograft models. By integrating these two mechanistically distinct yet complementary strategies including dual-site intercalation for sequence-specific targeting and bis-intercalator-induced structural modulation, this study provides a structural and mechanistic foundation for designing highly selective DNA-binding chemotherapeutics. These findings highlight the potential of structure-guided drug design in developing precision anticancer therapies with enhanced specificity, reduced toxicity, and improved clinical efficacy.



台灣生物化學及分子生物學學會 3/23 (Sun.) 13:30-14:30 3 樓, 33 教室





Current Position

Professor, Department and Graduate Institute of Pharmacology, National Taiwan University, Taiwan

Attending physician, Division of Cardiology, Department of Internal Medicine, NTU Hospital, Taiwan

Education/Training

- 2000 MD, National Taiwan University College of Medicine
- 2012 PhD, Washington University in St Louis, USA

Professional and Research Experience

2012-2014 Post-Doc, University of Illinois at Chicago/Brown University
2000-2005 Resident/Clinical Fellow, Department of Internal Medicine, NTU hospital

Awards and Honors

- 2021 Outstanding Research Award, NSTC
- 2024 Taiwan Bio-development Foundation (TBF) Chair Professor Award
- 2022 The 18th Tien Te Lee Biomedical Awards

Targeting aberrant TXNDC5 expression in stromal fibroblasts resolves tumor desmoplasia and resistance to immune checkpoint blockade in colorectal cancer with mesenchymal traits

楊鎧鍵 Kai-Chien Yang

Professor, Department and Graduate Institute of Pharmacology, National Taiwan University, Taiwan, Attending physician, Division of Cardiology, Department of Internal Medicine, NTU Hospital, Taiwan

Mesenchymal-type colorectal cancer (CRC), characterized by strong stromal infiltration and immune tolerance, resists immune checkpoint blockade and has poor outcomes. Cancer-associated fibroblasts (CAFs), abundant in tumor stroma, actively remodel the extracellular matrix (ECM), aid immune evasion, and drive tumor progression. We have recently identified thioredoxin domaincontaining protein 5 (TXNDC5), a protein disulfide isomerase (PDI), as a critical mediator of fibroblast activation and ECM remodeling in organ fibrosis. We hypothesized that TXNDC5 could also contribute to fibroblast activation, stroma formation and tumor progression in cancer, especially in the stroma-enriched fibrogenic mesenchymal-type CRC. Methods: Transcriptome databases of CRC were re-analyzed to determine the clinical relevance of TXNDC5. Experimentally, CRC was induced in mouse lines by azoxymethane (AOM) and dextran sulfate sodium (DSS) stimuli, a model sharing multiple characteristics with human mesenchymal-type CRC. Human colonic fibroblast line CCD-18co was used to investigate the molecular mechanisms by which TXNDC5 regulates colonic fibroblast activities. Fibroblast-specific TXNDC5 knockout (Col1a2-Cre/ERT2*TXNDC5fl/fl, cKO) mice were generated, combining with single-cell RNA sequencing analyses on AOM/DSS-induced CRC tumors in these animals, to clarify how fibroblast TXNDC5 impact tumor microenvironment, CRC progression and response to immune checkpoint blockade. Findings: TXNDC5 was predominantly expressed in stromal fibroblasts of human and mouse CRC. Fibroblast-specific deletion of Txndc5 lessened CAF activation, attenuated tumor fibrosis and reduced tumor burden in AOM/DSS-induced CRC. Mechanistically, increased TXNDC5 levels augments TGF signaling in CAF by post-translational stabilization of TGFBR1 through its PDI activity. In addition, deletion of Txndc5 in CAFs led to less tumor desmoplasia, decompressed tumor vessels and attenuated intratumoral hypoxia, thereby easing immune tolerance and increasing cytotoxic T cell infiltration in CRC. Single-cell transcriptome analysis revealed a marked change of intratumoral immune cell populations upon fibroblastspecific deletion of TXNDC5, shifting from myeloid-derived suppressive cells to cytotoxic tumorinfiltrating lymphocytes. Importantly, depletion of TXNDC5 in CAFs potentiated the anti-tumor effects of immune checkpoint blockade with anti-PD1 therapy in CRC. Conclusions: Our data suggest an important yet previously unrecognized role of fibroblast TXNDC5 in CRC progression, through enhancing CAF activation, stroma formation and immune escape. Combining immune checkpoint blockade with TXNDC5 deletion synergistically improved anti-tumor effects in CRC. Targeting TXNDC5, therefore, can be a novel therapeutic approach for CRC patients.



台灣生物化學及分子生物學學會 3/23 (Sun.) 14:50-15:50 3 樓, 33 教室





Current Position

Distinguished Professor, Department of Biotechnology and Bioindustry Sciences, National Cheng Kung University, Taiwan

Dean, College of Bioscience and Biotechnology, National Cheng Kung University, Taiwan

Education/Training 1999 PhD, Institute of Life Science, National Defense Medical Center, Taipei, TW

Professional and Research Experience

2024-Present President, The Taiwan Society for Biochemistry and Molecular Biology, Taiwan
2022-2023 Chairman, Life Sciences Research Promotion Center, Taiwan
2019-2022 Vice President, Academic Affairs, NCKU, Taiwan

Awards and Honors

- 2024 K. T. Li Honorary Scholar Award
- 2023 K. T. Li Gold Medal Award
- 2021 MOST Outstanding Research Award

Disruption of the pentraxin 3/CD44 interaction can be an efficient strategy for disease therapy

王育民 Ju-Ming Wang

Distinguished Professor, Department of Biotechnology and Bioindustry Sciences, National Cheng Kung University, Taiwan, Dean, College of Bioscience and Biotechnology, National Cheng Kung University, Taiwan

Fibroblasts, as key structural components of all organs, play a pivotal role in immune-mediated inflammatory diseases, including cancer. Pentraxin 3 (PTX3), a secretory factor induced by proinflammatory cytokines and various stresses, is primarily expressed by fibroblasts and monocytes/ macrophages in injured tissues and is elevated in the serum of patients with inflammatory diseases. Beyond its well-established role in promoting cancer migration, invasion, stemness, and drug resistance, our study reveals that PTX3 also contributes to immunosuppression by activating M2 macrophages and inhibiting cytotoxic CD8+ T cells. Furthermore, in vitro and in vivo studies showed that PTX3 plays a crucial role in tissue fibrosis, with its interaction with CD44 significantly driving fibrotic disease progression. We further found that PTX3 regulate the activation of TGF β signaling and extracellular matrix and epithelial-mesenchymal transition genes in epithelial cells and fibroblasts. Recognizing the pathological significance of PTX3, we developed WHC-001, a PTX3specific neutralizing antibody, to explore its therapeutic potential in chronic diseases, including cancer and fibrosis. Our findings demonstrate that WHC-001 effectively suppresses tumor progression in colon cancer and triple-negative breast cancer (TNBC) while also mitigating tissue fibrosis. These results suggest that targeting the PTX3/CD44 axis with WHC-001 represents a promising therapeutic strategy for cancer and fibrotic diseases.



台灣生物化學及分子生物學學會 3/23 (Sun.) 14:50-15:50 3 樓,33 教室



2025 The 39th Joint Annual Conference of Biomedical Science 合學 術年



科技新知研討會 **Technology Symposium**



■ 2025 The 39th Joint Annuel Conference of Biomedical Science 生物醫學聯合學術年會

時間:3月22日(Sat.)12:00-12:30 地點:3樓,31教室 單位:莫德納台灣股份有限公司

Speaker/黃立民 台灣大學特聘教授 台灣大學醫學院小兒科暨台大公衛學院流行病學與預防醫學研究所教授 感染症醫學會名譽理事長 兒科醫學會副理事長 台灣病毒暨疫苗學會理事長

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Moderator/司徒惠康

中華民國免疫學會理事長

mRNA 科學:從新冠抗疫到未來的無限可能

mRNA 技術的發展為疫苗和治療領域帶來了革命性的變革,開啟了精準醫療的新時代。從 COVID-19 疫苗的快速研發,到未來在感染性疾病、癌症免疫治療和罕見病治療上的應用,mRNA 技術展現了廣泛的潛力。

有關 mRNA 技術的核心優勢, mRNA 疫苗的研發建立在數位序列設計、mRNA 合成與脂質納米 顆粒(LNP) 遞送技術之上,具備以下優勢:快速開發與靈活製造、多功能應用、強效免疫應 答、細胞無需進入核內等等。因此在 COVID-19 疫苗的突破上, mRNA 技術在 COVID-19 疫情中 證明了其極高的效率和可行性。例如, Moderna 的 mRNA-1273 疫苗從病毒基因測序到獲得緊急 使用授權(EUA)僅耗時 11 個月,遠超傳統疫苗的研發速度。此外,針對 COVID-19 變異株(如 XBB1.5)的新一代疫苗 mRNA-1283,已經展現出更好的免疫原性與更長的冷藏保存期限。

在 mRNA 技術的未來應用上,也將提供許多例子供聽眾參考,包括在多重疫苗開發上有流感與 COVID-19 聯合疫苗(mRNA-1083)、呼吸道合胞病毒(RSV)疫苗(mRNA-1345):適用於老年 人和嬰幼兒,降低住院與死亡風險等,以及不論在癌症免疫治療或是罕見病與慢性疾病治療的 各項成功例子。

mRNA 技術不僅改變了疫苗的開發方式,也為癌症、罕見病、免疫治療等領域帶來了新希望。未來,隨著遞送系統的優化與抗原設計的進步,mRNA 技術將在更多疾病領域實現突破,為全球公共衛生帶來深遠影響,充分展示了 mRNA 技術如何從 COVID-19 疫苗開始,進一步拓展到更廣泛的醫療應用,並強調了該技術在精準醫療時代的無限潛力。

時間:3月22日(Sat.)10:30-11:00 地點:1樓,中庭 單位:龐德生技有限公司

Speaker/ Jonathan Yang Applications Specialist, Leica Biosystems

Troubleshooting Routine Histology: A Guide on How to Avoid Common Mistakes 常規組織學疑難排解:如何避免常見錯誤的指南

Understanding the routine histology workflow is essential for producing high-quality slides and accurate diagnoses. This session will cover the complexity of a typical histology process, highlighting why troubleshooting can be challenging and provides practical insights and proper techniques on how to avoid common mistakes in key steps, including: Grossing, Fixation, Processing, Embedding, Microtomy, Staining, Coverslipping, Storage and Archiving.



時間:3月22日(Sat.)15:00-15:30 地點:1樓,中庭、 單位:美商伯瑞股份有限公司台灣分公司

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Speaker/ 呂秋瑩 Bio-Rad 美商伯瑞專案經理 台灣大學生化科技學系碩士

JACBS

Enhancing CAR-T Manufacturing Quality with Droplet Digital PCR 利用微滴數字 PCR 提升 CAR-T 的製程品質

th 2025 The 39th Joint Annual Conference

合學術年

在當今的細胞與基因治療領域, CAR-T 細胞療法已成為癌症治療的重要突破。然而, 確保 CAR-T 細胞製造的穩定性和一致性仍然是一項重大挑戰。微滴數字 PCR(Droplet Digital PCR, ddPCR) 作為一種高靈敏度、高精確度的基因定量技術,為CAR-T製造流程提供了強大的品質控制工具。 本次講座將介紹 Bio-Rad ddPCR 技術,包括其原理、優勢及如何克服傳統 qPCR 方法的局限性。 ddPCR 透過數位化樣本分割,可提供更準確的 病毒載體滴度測定、CAR 基因拷貝數分析,以及 殘餘 AAV 檢測,確保基因改造的 T 細胞品質。

此外,我們將探討 AAV(腺相關病毒)相關試劑套組 在 CAR-T 製造中的應用。透過 ddPCR 技術, 研究人員和製造商能夠精確量化基因表達、監測製造變異,並提升 CAR-T 細胞治療的一致性與安 全性。本講座將深入探討這些技術如何優化 CAR-T 生產流程,提升整體治療品質。







th 2025 The 39th Joint Annuel Conference of Biomedical Science 生物醫學聯合學術年會

中國生理學會

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編號	論文題目
PY001	Examinations of environmental enrichment in morphine-induced rewarding conditioned place preference: analysis of brain-derived neurotrophic factor and neuroinflammation responses 潘靖怡 , 黃智偉
PY002	Elucidations of environmental enrichment on methamphetamine-induced behavioral sensitization in behavior and brain mechanisms 鄭凱恩 , 吳少傑 , 黃智偉
PY003	Examinations of chronic mild stress altering phosphorylated extracellular signal- regulated kinase to increase neuronal apoptosis in the brain 馬琬珺 , 黃智偉
PY004	Investigating the Pathway of PKC a in the Disruption of Endothelial Tight Junctions Induced by Blue Light 鍾孝庭 , 溫宏諾 , 李青澔
PY005	The Association of Peripheral Blood Inflammation Indices with Disease Severity and Mortality Caused by Coronavirus Disease 2019 (COVID-19) 張智鈞 , 詹鈞任 , 魏止善 , 朱芳業
PY006	Novel KIF20B Insertion Mutation Associates with Male Infertility and Impaired Spermatogenesis in Taiwanese Population 汪雅雲 , 林盈宏
PY007	Investigating the Role of Anti-aging Klotho in Dentate Gyrus Network Dynamics and Behavioral Correlates 歐諾亞 , 連正章
PY008	Effects of oxytocin in posttrumatic stress disorder affecting rewarding and aversive effects induced by alcohol 洪沛濬 , 劉人瑄 , 蔡羽柔 , 吳承恩 , 林宇晨 , 黃智偉
PY009	Footshock stress induces freezing behavior and interlukin-1beta expression in the medial prefrontal cortex, amygdala, and hippocampus during situational reminder: a posttraumatic stress animal model test 宋昀臻,洪沛濬,黃智偉
PY010	社會支持對於憂鬱症患者在憂鬱行為反應之研究 王崇美 , 黃智偉
PY011	Exploring How CCR5 in Brown Adipose Tissue Affects Lipid Metabolism in the Liver 羅祐安 , 邱威誠 , 郁兆蘭
PY012	Aryl Hydrocarbon Receptor Defect Attenuates Mitogen-Activated Signaling Through Leucine-Rich Repeats and Immunoglobulin-Like Domains 1 (LRIG1)- Dependent EGFR Degradation 李青澔 , 許翰琳 , 陳竑愷 , 詹燕茹

編號	a de la companya de l
PY013	Sex Differences in Neural Circuits Und 黃貽珺,陳榆涵,夏子涵,陳純娟,黃
PY014	The Role of Spinal BAF in Epigenetic S Neuropathic Pain 謝明君 , 賴政遠 , 林則彬 , 王學孝 , 鄭
PY015	CtBP1-LSD1 Complex-Mediated Epige Transcription in the Dorsal Root Gang Neuropathic Pain 謝明君,賴政遠,周迪侖,倪曉彤,陳 彭賢祐
PY016	The Role of Mitochondrial Methyltran Cardiotoxicity 鍾昕叡 , 簡千栩
PY017	Elevated HIF-1 α –NKCC1 Signaling Ur Therapeutic Potential of 2-Methoxyes 陳子漠 , 林維星 , 呂睿傑 , 陳易群 , 吳
PY018	Exploring the Influence of Neuropept Resistance and Metabolic Dysregulati 賴苡捷 , 林雅婷
PY019	Exosomes Derived From miR-5004-3p Treat Gouty Arthritis by Inhibiting the 黃渝珊 , 陳冬生
PY020	Green tea epigallocatechin gallate inh microRNA-let-7a/HMGA2 signaling pa 許紫媞
PY021	Exosomes Derived from Ohwia Cauda Cells Enhanced Treatment of Gout by Modulating Mitochondrial Dynamic. 巫靖妤,陳冬生
PY022	Nostoc commune Polysaccharide Extr Therapeutic Efficacy in Ameliorating S Mitochondrial Dynamics. 周秀咪,陳冬生
PY023	Improvement of Dyspnea in Long CO 謝雨珊 , 陳姚向
PY024	A Patient-Derived Xenograft Model Bi 王瑞鈴 , 蕭麗如 , 羅昀琪 , 馬文輝 , 謝



論文題目

nderlying Observational Fear Learning 黃佳瑜

Silencing of the Mu-Opioid Receptor Gene in

鄭仁坤 , 楊博勝 , 許介謙 , 周迪侖 , 彭賢祐 genetic Modulation of ErbB2 Gene

nglion Contributes to Paclitaxel-Induced

陳安旂,陳美錡,許耕綸,許介謙,林則彬,

ansferase Mettl15 in Doxorubicin-induced

Underlies Juvenile Stress-Induced Anxiety: estradiol 尼京訓 相亦珍 日団枝

吳宗訓 , 楊奕玲 , 呂國棟

ptide FF on Palmitate-Induced Leptin ation

3p-Overexpressing Mesenchymal Stem Cells to ne ROS/Inflammasome/Pyroptosis Pathway

nhibits X9 beige preadipocyte growth via the pathways

data Extract Treated-Mesenchymal Stem by Regulating Inflammasome Activation and c.

ktract Enhances Wharton's Jelly Stem Cell g Senescent Cardiac Tissue via Modulation of

OVID Patients Using an Incentive Spirometer

Biobank for Cancer Study and Drug Discovery 謝曉君,包玉蘋,楊乃潔,陳姿伶,秦咸靜

th 2025 The 39th Joint Annual Conference of Biomedical Science 生物醫學聯合學術年會

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PY035	Vaspin Alleviates Atherosclerotic Plaque Instability by Regulating Smooth Muscle Cell Phenotypic Switching 陳懿 , 蔡旻倩
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ata extract on neuroprotection

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ibrosis Development in Scleroderma

ial: A Herbal Solution for Hypertension and

, 王一舟 , 林嘉祥 , 謝佩坊 , 林銘炫 , 陳品勳 ,

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scavenging capacity is a crucial mechanism for g tolerance in mung bean seedlings

nents enhance drought tolerance in mung oncentration and antioxidant activity

and Cold Tolerance in Mung Bean Seedlings nt

n Dephosphorylation with Phosphorylation

II Molecule Inhibitors that Interfere with the T5H

ol to Identify Drug Leads from Natural Products 邱顯泰

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valuation against Cholestatic Liver Injury 黃瑋琪 , 陳文英

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ects Chondrocytes against Sodium rough Activating PI3K/Akt Pathway 邱溥容 , 胡祐甄 , 張基隆

n (CLP) Regulates Low-Molecular-Weight rium carotovorum subsp. Carotovorum

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rine Senescence Mediated by Exosomes from ent Cells in Skin Aging

Required for Nuclear Envelope Integrity

l Differentially Methylated RNA m5C Landscape nisms in Mice

ous Lactobacillus Species Restored DSSrs and Neurotransmitter Imbalances in Mice

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BC115	Investigating the Regulation of PTX3 to Glycosylation of PD-L1 in Lung Cancer 洪凯琳 工会员 林佩際			

BCT15 洪凱琳,王育民,林佩瑩

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Thermodynamic Analysis of the Role of D/CR Function

3 (PTX3) and Fibroblast Growth Factor 2 (FGF2)

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IMM2	C9-Based CART Cells Prolong Survival in Pancreatic Cancer by Targeting Oncofetal Chondroitin Sulfate 賴巨虎,姜珂,李啟賓, Preeta Ananthanarayanan,陳雅萍,董爽, Serena Tondi, 王奕霖, Berina Šabanović,張穎宜,沈柏用, Agerbæk MØ, Thomas M. Clausen, Tobias Gustavsson, Thor G. Theander, Ali Salanti, Christopher Heeschen, Alexandra Aicher
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IMM4	Rhodiosin Alleviates Inflammatory Responses in Activated Macrophages by Suppressing NLRP3 Inflammasome Activation and STAT3 Phosphorylation 俞麗人 , 郭敏玲
IMM5	Lung Microbiota-derived SCFAs Coordinate Alveolar Macrophages and γδT Cells Activation through FFAR2 黃鼎捷 , 邵正玄 , 張雅貞
IMM6	IL-10 Therapy Strengthened the Immune Responses in Metastatic Liver Tumors. 顏孟玄 , 林佳儀 , 許瑜欣 , 王妙容 , 莊雅惠
IMM7	Deciphering Sex-Dimorphic Mechanisms of SAA3 in Macrophage Polarization and Activation via Simultaneous scRNA-seq and scATAC-seq 王睿君 , 周姿吟 , 林建達 , 葉育真
IMM8	Exploring Microbiome-Independent Mechanisms of Helminth Infections in Modulating Atherosclerosis and Immune Responses 陳克華 , 趙重洵 , 張世宗 , 李昆達 , 林建達
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en of the imiquimod (IMQ) stimulated lupus

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d Treg Instability in IBD and the Restorative

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PH001	Oxytocin Treatment Rescues Irritability-like Behavior in Cc2d1a Conditional Knockout Mice 程冠翔 , 洪毓傑 , 凌斌 , 許桂森
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PH028	Deubiquitinase USP24 Activated by IL-6/STAT3 Enhances PD-1 Protein Stability and Suppresses T cell Anti-tumor Response 謝宏嘉 , 洪建中 , 王憶卿
PH029	CASK promotes non-small cell lung cancer growth via regulating constitutive EGFR activation and ERK- and Akt-dependent p21 expression 賴允涵 , 張晏瑜 , 黃婷茵 , 林琬琬
PH030	Neuroprotective potency of natural product TMUN003 against neurodegenerative disease via inhibition of DYRK1A 彭兆翔,洪紹旂,杜皇儒,曾彥慈,林偉德,李政忠,劉逸軒,許凱程,潘秀玲, 皇甫維君
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PH047	Discovery of A Novel Formyl Peptide I Neutrophilic Inflammation 陳柏任,陳舜華,洪欣儀,李宜臻
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AN066	Osteoclasts stimulated by calcitonin enhance Slit3 secretion which promotes angiogenesis 朱慈暉 , 羅友志 , 姚文杰 , 黃宸鏞 , 徐昕妤 , 賴昕霖 , 廖敏宏 , 徐佳福
AN067	Investigation of Osteogenesis and Angiogenesis Coupling in 3D-Printed Vascularized Bone Organoids Under Mechanical and Fluidic Stimulations 徐佳福 , 羅友志 , 姚文杰 , 徐昕妤 , 黃宸鏞 , 廖敏宏 , 賴昕霖
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AN069	Investigating The Correlation of Noise-Induced Hearing Loss with Neuroinflammation and Neurodegenerative Diseases Using Positron Emission Tomography 鍾凱鈞,陳智遠,陳芊汗,鄭澄意,馬國興,

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CM001	Orphan nuclear receptors drive Alte development 維娜斯 , 沈依伶 , 黃玫潔 , 曾奕儒 ,
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, 丁品喬 , 簡裕峰 , 陳偉武 , 陳律佑 dance Sensing to Monitor Stem Cell Activity in

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'β-Catenin Signaling Promotes the Progression llicular Thyroid Cancer. Bo-Rong Chen, Michael Hsiao, Ming-Hsun Wu,

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Circular RNAs and Host Genes Act in Regulating in Skeletal Myogenesis

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eneration Circulating Tumor Cell(CTC) Detection

ential of Nobiletin in Non-Small Cell Lung Pathogenesis of NSCLC.

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CM068	Stress Induced Crosstalks of Musashi-1 Stress Granules and β -Destruction Complex via LLPS in CRC Organoids 謝霖翔 , 謝采穎 , 江奕勳 , 劉彥汶 , 許賀俊 , 梁振威 , 鐘育志 , 邱光裕
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TX020	Developing an Integrated Platform with Yeast and Human Tumor Cell Models to Characterize Anticancer Drugs Targeting the Mitochondrial Respiratory Complexes 鞠采霓 , 張欣蕙 , 李立璿 , 黃薇玲 , 張壯榮 , 郭靜娟
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In Vitro High-Throughput and High-Content ve Toxicity Testing and ToxPi Ranking of using on Liver and Endocrine Disruptions

^f Geniposidic Acid on Necroptosis-Induced d In Vivo.

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y Recombinant SODTMP-Latex Clearing Fusion 4 Heterologously Expressed in Escherichia coli (全福 A

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MI012	Vapor-Phase Fabrication of Porous Parylene Coatings for an Interstitial Fluid Filtration Device 張育銘 , 黃啟裕 , 林奕維 , 陳賢燁

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MI013	Targeting the LIMK/CFL Pathway with Treatment 林旻穎 , 呂志得 , 吳駿一 , 陳亮丞 , 李
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MI018	Personalized Targeted Radionuclide Tl Consistency Model Networks 王佳柔 , 林可瀚 , 楊邦宏 , 施政廷 , 吳
MI019	The Upregulation of Cofilin-1 in Senes Telomere Function 高佳偉 , 李易展
MI020	Outcome Prediction in HER2-positive Clinical Feature Analysis of DCE-MRI 洪采妮,李佳芬,吳文沛,盧家鋒
MI021	Utilizing Diffusion Tensor Imaging to I Repetitive Mild Traumatic Brain Injury 郭品慧 , 黃琡惠 , 宋映葦 , 高瑀絜
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MI024	In Vivo SPECT/CT Imaging of Radiolab Solid Tumor Model 陳亮丞 , 羅瑋霖



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sion and Protection Against Noise-Induced cranial Ultrasound Combined with Intravenous d Oxygenated Microbubbles 王智弘, 廖愛禾

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MI025	ALDOC-mediated neurotransmitter reprogramming and PPAR-γ signaling to treat glioblastoma progression 林又妤 , 何兆璟 , 郭翰錫 , 張御展
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Characteristics in a Rodent Model with arametric MRI 謝永雋,官振翔,徐子琹,楊啟裕,郭立威 dicines with Oxygen Vacancies for Imagingver Tumors Ain-Chiao Liao, Chia-Hao Su

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MI060	SNR Enhancement Using a Single-Channel Phased Array Coil for 3T MRI 吳政哲 , 陳名傑 , 何伯勳 , 江宣翰 , 郭立威 , 卓冠宏
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,鄭凱鴻,楊浚泓,何宗澧,詹振勳,陳怡珊,

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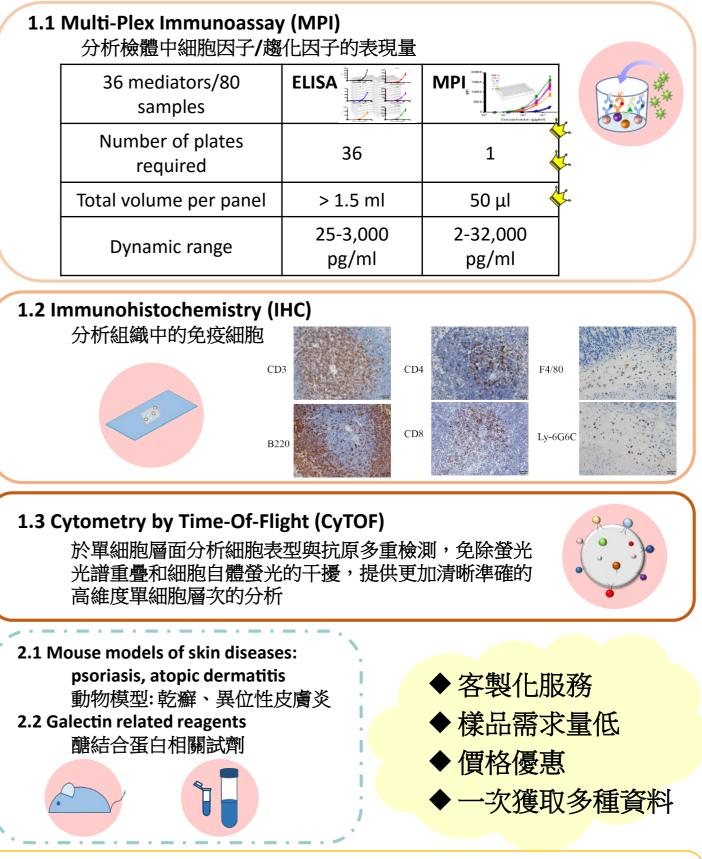
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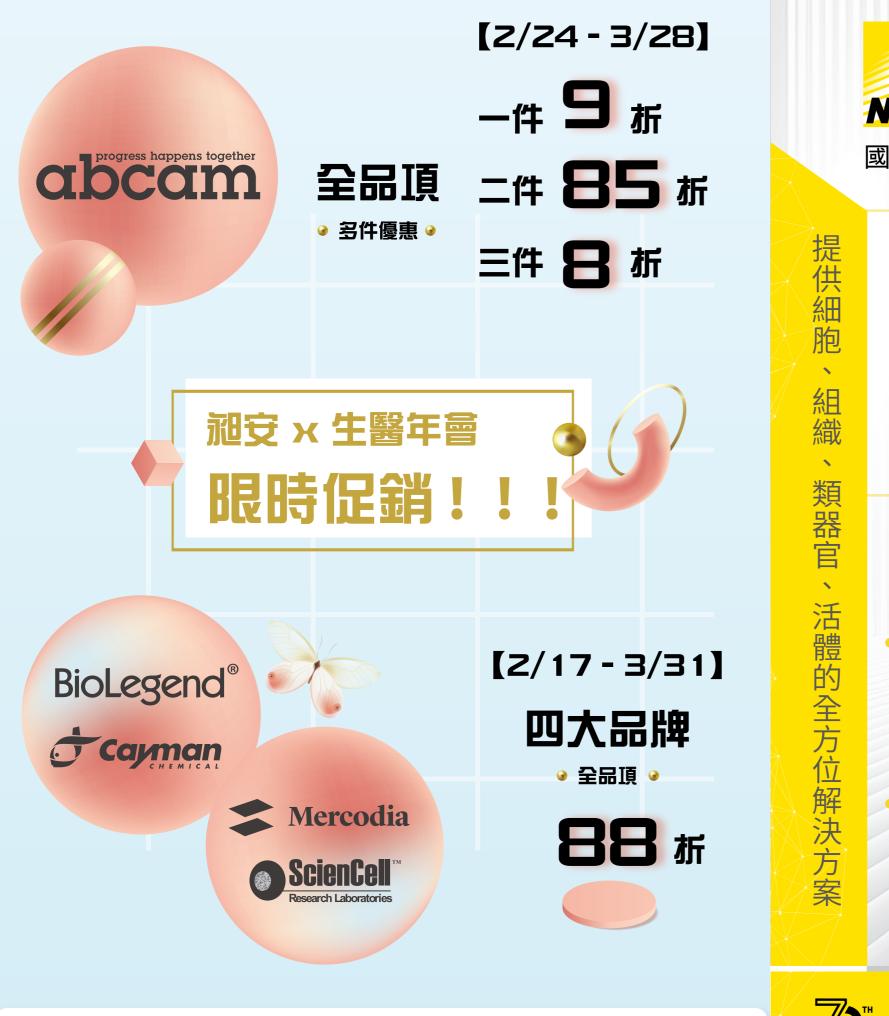
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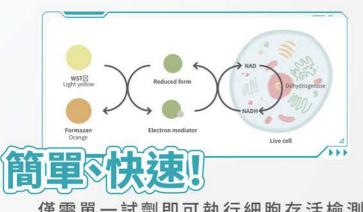
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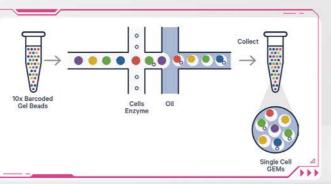


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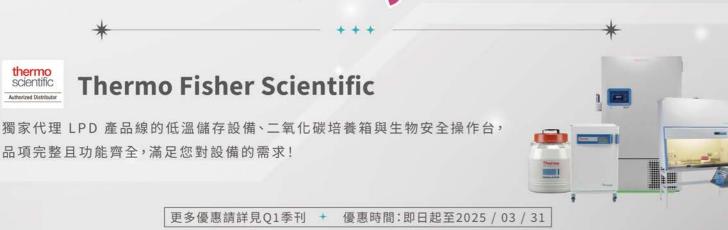
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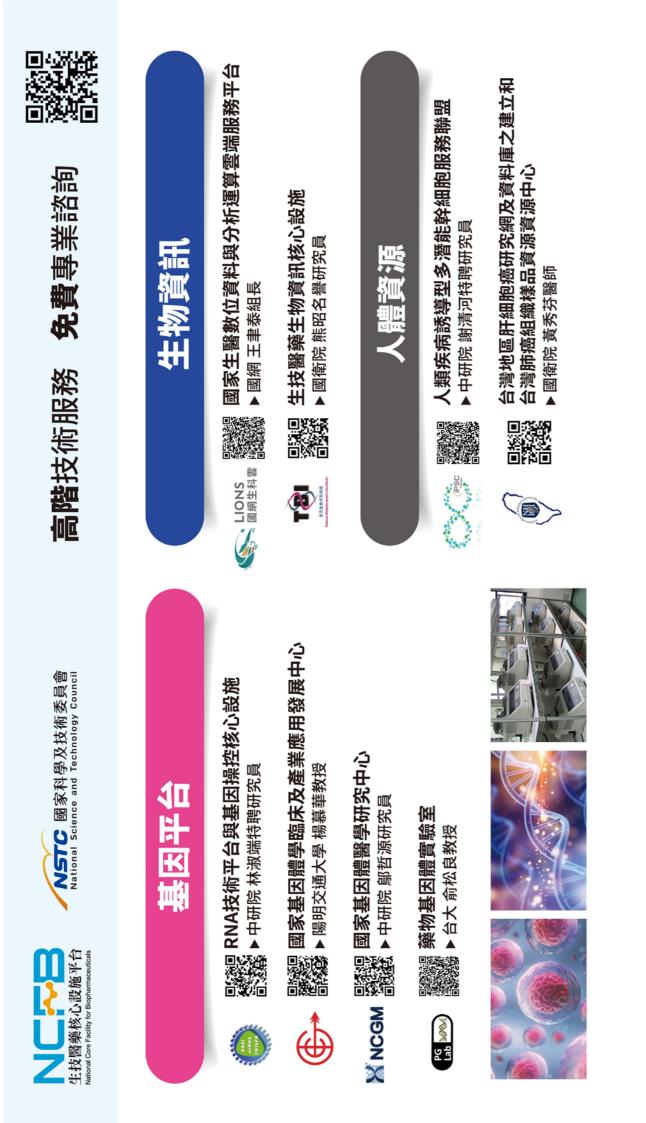










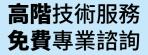




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生物資源



模式生物資源中心 ▶臺大丁照棣教授



台灣水稻突變種原庫及基因資料庫 之管理與加值利用 ▶興大 賀端華院士

次世代藥物



一站式藥物早期研究 / 臨床前服務平台 ▶國衛院 洪明秀研究員



回察回·次世代核酸藥物平台 高效效 ▶清大 孫玉珠教授 次世代核酸藥物平台



● 核酸藥物材料核心設施服務平台
 ● 國衛院劉士任研究員

