國 科 會 生 命 科 學 研 究 推 動 中 心

# 研討(習)會 結案報告

一、基本資料

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| 中文名稱 | 2025 生物醫學新知研討會 |
| 英文名稱 | 2025 Joint Symposium on Recent Advances in Biomedical Sciences |
| 中文摘要報告 | 本國際會議最後有超過百人參加。其中有 11 位來自中研院生醫所、3 位來自國立清華大學、2 位來自美國、2 位來自英國、1 位來自法國及 1 位來自德國的知名醫學教授，各自發表他們在神經科學的研究議題上的最新研究成果。同時有 71 篇的海報論文，從事神經科學的醫學研究學者與許多新竹地區醫學相關從業學者、醫師與會，透過不同的論文發表形式（研究報告、口頭報告、海報報告等），擴大本所與新竹學術界、醫界的交流與接觸，交流最新的神經科學與生物醫學的新知。<br>遠道而來的 6 位國際知名學者，是自從疫情爆發以來，首次親自造訪台灣，與現場的與會人員，面對面的交流，一面提高研究視野並與相關領域學者共享研究成果；另一方面也拓展學者的研究人脈，同時提高學校、醫院和研究機構的知名度及國際上的能見度。<br>感謝大家一起腦力激盪，面對面討論重要的基礎與臨床醫學課題，促成團隊間合作，以利轉譯醫學研究之推展，共同為國人的健康努力。 |
| 英文摘要報告 | In the end, this international conference had more than a hundred participants. Among them, there are 11 well-known medical professors from the Institute of Biomedical Sciences of Academia Sinica, 3 from National Tsing Hua University, 2 from the United States, 2 from the United Kingdom, 1 from France and 1 from Germany, each of whom published their latest research results on neuroscience research topics. There were also 71 poster papers. Medical researchers engaged in neuroscience and many Hsinchu area medical practitioners and physicians attended the conference. Through different paper presentation forms (research reports, oral reports, poster reports, etc.), the institute expanded its exchanges and contacts with the academic and medical communities in Hsinchu and exchanged the latest knowledge in neuroscience and biomedicine.<br>The six internationally renowned scholars who came from afar visited Taiwan in person for the first time since the outbreak of the epidemic. They had face-to-face exchanges with the participants on site, which not only improved their research horizons and shared their research results with scholars in related fields, but also expanded the scholars' research network, while also increasing the reputation and international visibility of schools, hospitals and research institutions. |

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| | Thank you all for brainstorming together, discussing important basic and clinical medical topics face to face, promoting collaboration among teams, promoting translational medical research, and working together for the health of our people. |
| 研討(習)會目的 | 中央研究院生物醫學研究所與國立清華大學於 2025 年 3 月 10 日至 12 日共同舉辦 2025 生物醫學新知研討會。此次之新知研討會將著重於神經科學領域的學術探討。<br>我們國家正逐漸邁入高齡社會，神經科學研究將為我國和先進國家重點發展領域之一，其應用性研究則對我國醫療、社福及經濟的各項均有極大的影響，如能有效偵測、預防與治療這些神經的病變，對達到治療的目的有非常大的助益。神經醫學的重要性以及所涉的龐大醫療市場，使得世界各國皆大力投入研發，為了使我國神經醫學和相關領域之研究基於原有之良好基礎上，能作更進一步之研究及突破，以提升神經醫學相關領域之研究水準，因此借召開此次 2025 生物醫學新知研討會之便，期望能有更多頂尖優秀人才投入進行較深入、創新及具長遠規劃之神經醫學科學領域研究，俾使基礎與臨床神經醫學研究突破瓶頸，開拓新研究思維及領域，以期能讓我國在具有發展優勢的神經醫學科學在特定研究主題之研究水平達到國際一流標準，並在將來於國際上居於領先地位。<br>本研討會的主要目的是以神經的基礎研究治療新策略為主旨，包括從分子到功能學科的最新進展的主要議題，並從基礎到臨床研究。並探討慢各種神經的生理與病理機制，以及在相關臨床醫學上有關神經的治療發展現況和未來的新趨勢，並希望經由邀請專家學者的研究成果交流，提昇國內學術界之研發能量，以及促進基礎與臨床實務與技術交流的機會。有鑑於此，此次會議邀請世界先進的神經醫學的研究學者與國內神經科學研究學者共同在一個自由分享與合作氣氛下的國際會議中做學術交流。<br>有鑒於神經的醫學是國家基礎研究中的重點項目。為了提升年輕學者與學生的視野，及增加與醫學界的交流，以達到基礎與醫學相互結合的效果，也為了鼓勵國內學者參與研討會並發表論文，在中央研院生物醫學研究所與國立清華大學聯合舉辦此次的國際學術會議，研討會預定 2025 年 3 月 10 日於中研院生醫所，舉辦 1 場大型生物醫學研討會。之後前往新竹，於 3 月 10-12 日舉辦三場有關神經科學的學術交流討論會及二場生物醫學壁報研討會。預期可藉大型開放式演講與壁報活動，吸引大量新竹地區醫學相關從業學者與會，擴大本所與其他地區醫界的交流與接觸。本所同仁亦可藉學術交流討論會報告研究進展，達到與醫界充分交流並促成規畫合作內容的目的。大家一起腦力激盪，面對面討論重要的基礎與臨床醫學課題，促成團隊間合作，以利轉譯醫學研究之推展，共同為國人的健康努力。 |
| 參加對象(含人數) | 神經科學的醫學研究學者、學生 |
| 預期效益達成狀況 | （1）瞭解目前神經的生理和病理機制在不同模型的研究議題， 並獲得最新研究成果，預期對國內研究水準具有相當大的助益。<br>（2）瞭解臨床領域的研究現況與神經的治療的成果，以提供國內基礎研究的參考依據。 |

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| | （3）藉由參與國際研討會，一方便提高研究視野並與相關領域學者共享研究成果，這是提升國內研究能力的方式之一；另一方面更期許拓展學者的研究人脈，亦能提高學校、 醫院和研究機構 的知名度及國際上的能見度。<br>(4) 本國際會議預計約有 100 名從事神經的科學基礎和臨床研究之生物醫學研究學者，透過不同的論文發表形式（研究報告、口頭報告、海報報告等），交流最新的神經科學與生物醫學的新知。<br>(5) 此次舉辦地點在國立清華大學，預計將吸引許多新竹地區醫學相關從業學者與會，擴大本所與其他地區醫界的交流與接觸。本所同仁亦可藉學術交流討論會報告研究進展，達到與醫界充分交流並促成規畫合作內容的目的。 |

活動照片和影片

- 照片(至少 10 張，請填寫照片說明)

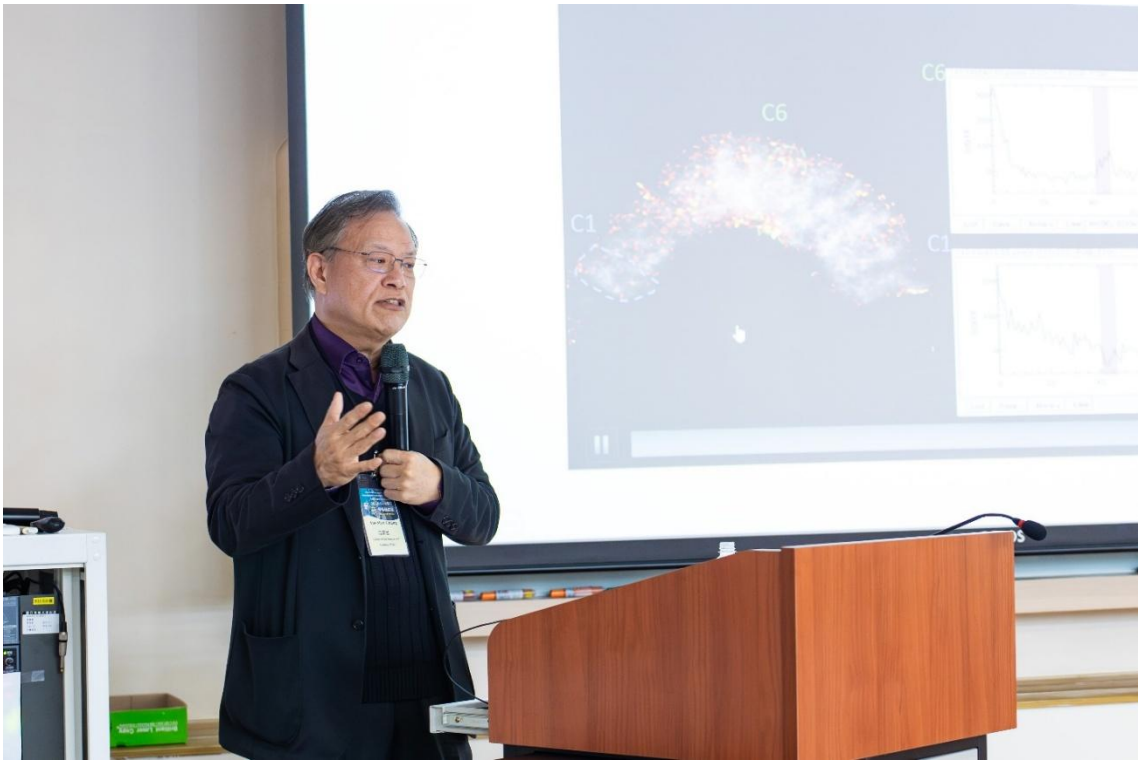
全體大合照

中研院生醫所所長(Dr.  陳儀莊)提問


Dr. Florian Lesage  上台發表

Dr. Eric Huang 提問

Dr. Peter McNaughton 提問




Dr. 江安世上台演講

Dr. Michael Hausser 提問

Dr. Gary Lewin 提問

Dr. Ying-Hui Fu 上台演講

海報展互動

# 2025 IBMS-NTHU Joint Symposium on Recent Advances in Biomedical Sciences

# 2025年中研院生醫所/清大生醫學院聯合生物醫學嶄新學術研討會

**Mar. 10-12, 2025**

## Program & Abstracts

## 議程與摘要

**中央研究院生物醫學科學研究所B1C演講廳、**

**國立清華大學生命科學二館黃秉乾講堂**

**Organizers:**

Institute of Biomedical Sciences, Academia Sinica

(中央研究院 生物醫學科學研究所)

College of Life Sciences and Medicine, National Tsing Hua University

(國立清華大學生命科學暨醫學院)

**Sponsor:**

國家科學及技術委員會補助

生命科學研究推動中心

Life Sciences Research Promotion Center (LSRPC)

# Symposium Program
# March 10-12, 2025

## Day 1 (Monday, March 10)

B1C Auditorium, Institute of Biomedical Sciences, Academia Sinica

| Time | Speaker | Title |
|---|---|---|
| 09:00-09:25 | Registration | |
| Moderator: Dr. Huang, Yi-Shuian (黃怡萱), IBMS | | |
| 09:30-09:40 | Opening Remarks<br>**Dr. Yijuang Chern (陳儀莊) -** Director, Institute of Biomedical Sciences, Academia Sinica | |
| 09:40-10:25 | Dr. Gary Lewin | *Exploring neuronal proteomes to understand touch and pain* |
| 10:25-11:10 | Dr. Michael Hausser | *Illuminating causal links between neural circuit activity and behaviour* |
| 11:10-11:20 | Closing Remarks | |

**Day 2 (Tuesday, March 11)**

Pien-Chien Lecture Hall, Life Science Building II, NTHU

國立清華大學生命科學二館黃秉乾講堂

| **Time** | **Speaker** | **Title** |
|---|---|---|
| 08:30-09:00 | Registration | |
| Moderator: Dr. Lee, Eminy H.Y. (李小媛), IBMS | | |
| 09:00-09:10 | Opening Remarks<br>**Dr. Yijuang Chern (陳儀莊) -** Director, Institute of Biomedical Sciences, AS<br>**Dr. Linyi Chen (陳令儀)-** Acting Dean for College of Life Sciences and Medicine, National Tsing Hua University | |
| 09:10-9:55 | Dr. Ann-Shyn Chiang<br>江安世 | *Dynamic Memory Retrieval and Individualized Decision-Making in Drosophila* |
| 9:55-10:40 | Dr. Peter McNaughton | *The role of HCN2 ion channels in chronic pain, tinnitus and opioid addiction* |
| 10:40-11:00 | Group Photo & Coffee Break | |
| Moderator: Dr. Lee, Eminy H.Y. (李小媛), IBMS | | |
| 11:00-11:45 | Dr. Eric Huang | *Disentangling immune-vascular interface in the prenatal human brain* |
| 11:45-12:05 | Dr. Chen, Chih-Cheng<br>陳志成 | *A role for proprioceptors in sngception* |
| 12:05-13:20 | Lunch | |
| Moderator: Dr. Lee, Eminy H.Y. (李小媛), IBMS | | |
| 13:25-13:45 | Dr. Pan, Ming-Kai<br>潘明楷 | *Neural dynamic approaches to cerebellar motor control* |
| 13:45-14:05 | Dr. Cheng, Sin-Jhong<br>鄭信忠 | *Management and services of the Neuroscience core facility* |
| 14:05-14:25 | Dr. Lo, Chung-Chuan<br>羅中泉 | *From Neural Stalemates to Robust Decisions: Insights into Goal-Directed Control in Drosophila Brains* |
| 14:25-14:45 | Dr. Hsu, Ching-Lung<br>徐經倫 | *Biophysics-Informed One-Shot Synaptic Algorithms for Learning Place Fields in Precise Memory-Dependent Spatial Behavior* |
| 14:45-15:05 | Dr. Hwang, Dennis W<br>黃聖言 | *Advances in Dynamic Imaging Techniques in Neuroscience: A Multifaceted Analysis of Metabolism and Vascular Function* |

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| 15:05-16:25 | Poster Session Group A & Coffee Break<br>at B1, Life Science Building II, NTHU<br>國立清華大學生命科學二館 B1 交誼廳 | |
| Moderator: Dr. Lee, Eminy H.Y. (李小媛), IBMS | | |
| 16:25-16:45 | Dr. Lee, Kuo-Sheng<br>李國昇 | *Exploring the Fundamental Circuit Mechanisms Underlying Somatosensation* |
| 16:45-17:05 | Dr. Chang, Chun-hui<br>張鈞惠 | *Aberrant Activation of the Orbitofrontal Cortex on Fear Regulation: A Systems Neuroscience Study* |
| 17:05-17:25 | Dr. Wang, Guey-Shin<br>王桂馨 | *Investigating the neural pathogenesis of myotonic dystrophy: from neurodevelopmental disorders to neurodegeneration* |
| 17:25-17:45 | Dr. Yang, Shi-Bing<br>楊世斌 | *Neural circuit basis of social investigative behaviors* |
| 18:00 | Dinner at B1, Life Science Building II, NTHU<br>國立清華大學生命科學二館 B1 交誼廳 | |

**Day 3 (Wednesday, March 12)**

Pien-Chien Lecture Hall, Life Science Building II, NTHU

國立清華大學生命科學二館黃秉乾講堂

| **Time** | **Speaker** | **Title** |
|---|---|---|
| Moderator: Dr. Lee, Eminy H.Y. (李小媛), IBMS | | |
| 9:00-9:45 | Dr. Florian Lesage | *From potassium selectivity to sodium permeability, a property specific to two pore-domain potassium channels* |
| 9:45-10:30 | Dr. Ying-Hui Fu<br>傅瑩惠 | *Piecing together the SLEEP puzzle: genes, molecules, and circuits* |
| 10:30-11:45 | Poster Session Group B & Coffee Break & Reviewed PI & Reviewers 1-1 meeting<br>at B1, Life Science Building II, NTHU<br>國立清華大學生命科學二館 B1 交誼廳 | |
| 11:45-12:05 | Dr. Lin, Wan-Chen<br>林宛蓁 | *Engineering high-performance optogenetic tools for specialized neural inhibition* |
| 12:05-12:25 | Dr. Huang, Yi-Shuian<br>黃怡萱 | *CMTR1-catalyzed mRNA capping in neurogenesis and brain development* |
| 12:25-12:35 | Closing Remarks<br>**Dr. Yijuang Chern (陳儀莊) -** Director, Institute of Biomedical Sciences, AS<br>**Dr. Linyi Chen (陳令儀)-** Acting Dean for College of Life Sciences and Medicine, National Tsing Hua University | |

**Gary R. Lewin Ph.D.**
Molecular Physiology of Somatic Sensation Laboratory, Max Delbrück Center for Molecular Medicine, Berlin, Germany

Website: https://www.mdc-berlin.de/lewin

## Education

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| 1986 | B.Sc. | University of Sheffield, Sheffield |
| 1990 | Ph.D. | University of London, London |

## Awards and Honors

2022 Honorary Fellow of the Physiological Society of the United Kingdom, Physiological Society (London)
2021 Bill Willis Prize Lecture, International Society for the Study of Pain
2019 Ernst Jung Prize for Medicine, Ernst Jung Stiftung
2008 Elected member of European Molecular Biology Organization, EMBO
1996 Young Investigator Prize of the International Society for the Study of Pain , IASP

## Ten most important career citations until 2017

1. Wetzel C, Pifferi S, Picci C, Gök C, Hoffmann D, Bali KK, Lampe A, Lapatsina L, Fleischer R, Smith ES, Bégay V, Moroni M, Estebanez L, Kühnemund J, Walcher J, Specker E, Neuenschwander M, von Kries JP, Haucke V, Kuner R, Poulet JF, Schmoranzer J, Poole K, **Lewin GR**. Small-molecule inhibition of STOML3 oligomerization reverses pathological mechanical hypersensitivity**. Nat Neurosci**. 2017 Feb;20(2):209-218. PubMed PMID: 27941788.

2. Park TJ, Reznick J, Peterson BL, Blass G, Omerbašić D, Bennett NC, Kuich PHJL, Zasada C, Browe BM, Hamann W, Applegate DT, Radke MH, Kosten T, Lutermann H, Gavaghan V, Eigenbrod O, Bégay V, Amoroso VG, Govind V, Minshall RD, Smith ESJ, Larson J, Gotthardt M, Kempa S, **Lewin GR**. Fructose-driven glycolysis supports anoxia resistance in the naked mole-rat. **Science**. 2017 Apr 21;356(6335):307-311. PubMed PMID: 28428423.

3. Poole K, Herget R, Lapatsina L, Ngo HD, **Lewin GR**. Tuning Piezo ion channels to detect molecular-scale movements relevant for fine touch. **Nat Commun**. 2014 Mar 24;5:3520. PubMed Central PMCID: PMC3973071.

4. Ranade SS, Woo SH, Dubin AE, Moshourab RA, Wetzel C, Petrus M, Mathur J, Bégay V, Coste B, Mainquist J, Wilson AJ, Francisco AG, Reddy K, Qiu Z, Wood JN, **Lewin GR,** Patapoutian A. Piezo2 is the major transducer of mechanical forces for touch sensation in mice. **Nature**. 2014 Dec 4;516(7529):121-5. PubMed Central PMCID: PMC4380172.

5. Smith ES, Omerbašić D, Lechner SG, Anirudhan G, Lapatsina L, **Lewin GR**. The molecular basis of acid insensitivity in the African naked mole-rat. **Science**. 2011 Dec 16;334(6062):1557-60. PubMed PMID: 22174253.

6. Chiang LY, Poole K, Oliveira BE, Duarte N, Sierra YA, Bruckner-Tuderman L, Koch M, Hu J, **Lewin GR**. Laminin-332 coordinates mechanotransduction and growth cone bifurcation in sensory neurons. **Nat Neurosci**. 2011 Jul 3;14(8):993-1000. PubMed PMID: 21725315.

7. Wetzel C, Hu J, Riethmacher D, Benckendorff A, Harder L, Eilers A, Moshourab R, Kozlenkov A, Labuz D, Caspani O, Erdmann B, Machelska H, Heppenstall PA, **Lewin GR**. A stomatin-domain protein essential for touch sensation in the mouse. **Nature**. 2007 Jan 11;445(7124):206-9. PubMed PMID: 17167420.

8. Price MP, McIlwrath SL, Xie J, Cheng C, Qiao J, Tarr DE, Sluka KA, Brennan TJ, **Lewin GR**, Welsh MJ. The DRASIC cation channel contributes to the detection of cutaneous touch and acid stimuli in mice. **Neuron**. 2001 Dec 20;32(6):1071-83. PubMed PMID: 11754838.

9. Lewin GR, Barde YA. Physiology of the neurotrophins. **Annu Rev Neurosci**. 1996;19:289-317. PubMed PMID: 8833445.

10. **Lewin GR**, Ritter AM, Mendell LM. Nerve growth factor-induced hyperalgesia in the neonatal and adult rat. **J Neurosci**. 1993 May;13(5):2136-48. PubMed Central PMCID: PMC6576576. 10.

# Exploring neuronal proteomes to understand touch and pain

**Gary R. Lewin Ph.D.**

A gentle touch or a painful pinch are relayed to the brain and spinal cord by rapidly conducting myelinated sensory neurons. Many sensory neurons, especially vibration sensitive mechanoreceptors that innervate Meissner's or Pacinian corpuscles, are exquisitely sensitive to mechanical force, being activated by sub-micrometer displacements. Such mechanoreceptors possess mechanically-activated ion channels, like PIEZO2 and ELKIN1 that can be gated by small movements of the substrate[1,2]. Both ELKIN1 and PIEZO2 are genetically required in mice for normal touch sensation. Transduction takes place at specialized neuroglial endings in the skin where mechanically gated ion channels function[3]. Comparison with simpler organisms like nematodes strongly suggests that mechanically gated ion channels are embedded in a protein complexes some components of which function as gating tethers linking to the extracellular matrix. We identified a candidate component for these tethers recently called TENM4[4]. I will describe how we are adapting advanced proteomic techniques to get insights into the protein composition of sensory ending where transduction takes place. We have recently been able to use ultra-low input proteomics to obtain rich sensory cell type proteomes which we can use to discover novel transduction components or even mediators of pain sensitization. Such methods may ultimately allow us to determine the protein composition of the sites of mechanotransduction in peripheral sensory endings.

References

1. Poole K, Herget R, Lapatsina L, Ngo HD, Lewin GR. Tuning Piezo ion channels to detect molecular-scale movements relevant for fine touch. Nature communications. 2014;5:3520. doi:10.1038/ncomms4520

2. Chakrabarti S, Klich JD, Khallaf MA, et al. Touch sensation requires the mechanically gated ion channel ELKIN1. Science. 2024;383(6686):992-998. doi:10.1126/science.adl0495

3. Ojeda-Alonso J, Calvo-Enrique L, Paricio-Montesinos R, et al. Sensory Schwann cells set perceptual thresholds for touch and selectively regulate mechanical nociception. Nat Commun. 2024;15(1):898. doi:10.1038/s41467-024-44845-8

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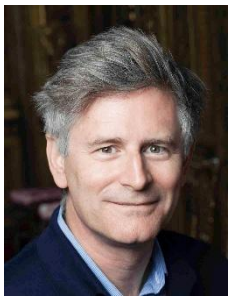

**Michael Hausser Ph.D.**
University College London, Gower Street, London, UK

Website: https://www.ucl.ac.uk/wolfson-institute-biomedical-research/circuit-neuroscience/professor-michael-hausser

## Education

Ph.D. Oxford University, UK

## Academic leadership and service

| | |
|---|---|
| 2016 - present | Scientific Advisory Board, Instituto de Neurociencias de Alicante |
| 2016 - present | Scientific Advisory Board, École des Neurosciences Paris Île de France |
| 2016 - present | Scientific Advisory Board, Hong Kong University School of Biomedical Sciences • |
| 2015 - present | Scientific Advisory Board, Institute of Molecular Pathology (IMP), Vienna |
| 2014 - present | Member, Committee of Higher Education and Training (CHET), FENS |
| 2015 | International review panel, Einstein Foundation Berlin |
| 2014, 2015 | NIH BRAIN Initiative Panel Member |
| 2014, 2015 | Interview committee, Sainsbury-Wellcome Centre Recruitment Symposium |

## Selected Publications

1. Packer, A, Roska, B, Häusser, M (2013). Targeting neurons and photons for optogenetics. Nature Neuroscience 16:805-15.

2. Schmidt-Hieber, C, Häusser, M (2013). Cellular mechanisms of spatial navigation in the medial entorhinal cortex. Nature Neurosci. 16(3):325-31

3. Smith, SL, Smith IT, Branco T, Häusser M (2013). Dendritic spikes enhance stimulus selectivity in cortical neurons in vivo. Nature 503:115-20

4. Haider B, Häusser M, Carandini M (2013). Inhibition dominates sensory responses in the awake cortex. Nature 493:97-100.

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# Illuminating causal links between neural circuit activity and behaviour

**Michael Hausser Ph.D.**
University College London, Gower Street, London, UK

Understanding the causal relationship between activity patterns in neural circuits and behavior is one of the fundamental questions in systems neuroscience. Addressing this problem requires the ability to perform rapid and targeted interventions in ongoing neuronal activity at cellular resolution and with millisecond precision. I will describe results of experiments using a powerful new & quot; all-optical & quot; strategy for interrogating neural circuits which combines simultaneous two-photon imaging and two-photon optogenetics. This enables the activity of functionally characterized and genetically defined ensembles of neurons to be manipulated with sufficient temporal and spatial resolution to enable physiological patterns of network activity to be reproduced. We have used this approach to identify the lower bound for perception of cortical activity, probe how brain state influences the role of cortex in perception, and provide causal tests of the role of hippocampal place cells in spatial navigation.

**Ann-Shyn Chiang (江安世), Ph.D.**
Brain Research Center, National Tsing Hua University, Taiwan

Website: https://www.genomics.sinica.edu.tw/en/chiang-ann-shyn

## Education

1990 Ph.D. in Entomology, Rutgers University, New Jersey, USA
1983 M.S. in Plant Pathology and Entomology, National Taiwan University, Taiwan
1981 B.S. in Entomology, National Chung-Hsing University, Taiwan

### Academic Position

2024-present Tenured Chair Professor, National Chiao Tung University
2023-present Adjunct Professor, Institute of Life Sciences, National Defense Medical Center
2021-present Adjunct Distinguished Chair Professor, Institute of Brain and Mind Sciences, NTU
2019-present Adjunct Chair Professor, Tunghai University
2017-present Adjunct Investigator, Institute of Molecular and Genomic Medicine, NIH
2016-present Adjunct Investigator, Institute of Physics, Academia Sinica
2016-present Adjunct Distinguished Investigator, National Synchrotron Radiation Research Center
2016-present Adjunct Distinguished Chair Professor, Graduate Institute of Clinical Medical Science, CMU
2014-present Adjunct Chair Professor, Kaohsiung Medical University
2019-present Faculty, Cell and Molecular Biology Program, UT Southwestern

## Awards and Honors:

2022 Honorary Doctorate of Kaohsiung Medical University
2022, 21, 20, 18 「FUTURE TECH Award」National Science and Technology Council
2021 National Chair Professorship Award, Ministry of Education
2020 Memorial Lecture of Li Yih-Yuen Emeritus Chair Professor
2020 Hou-De Honorary Chair (厚德榮譽講座)
2016 TWAS Fellow
2016 Presidential Special Lecturer, The Society for Neuroscience 2016 Annual Meeting
2015 National Chair Professorship Award, Ministry of Education
2014 Academician, Academia Sinica (中央研究院 院士)

**Major Scientific Achievements**

1. Odor Representation in Higher Brain Centers: Uncovered neuronal temporal identity and mechanisms for gating CO2 information, advancing understanding of olfactory processing (Cell, 2006, 2007; Science, 2013).

2. Long-Term Memory in Engram Neurons: Found that long-term memory requires protein synthesis in a small subset of neurons, essential for memory consolidation (Science, 2012; Cell Reports, 2023).

3. 3D Fly Brain Database: Developed the first brain-wide wiring map at single-neuron resolution, aiding sensory circuit mapping (Current Biology, 2011; Nature Communications, 2015).

4. Tissue Clearing Technology: Created FocusClear technology, the first tissue-clearing reagent compatible with GFP for high-resolution 3D imaging of intact brains at 30 nm resolution (Nature Communications, 2019).

# Dynamic Memory Retrieval and Individualized Decision-Making in *Drosophila*

**Ann-Shyn Chiang (江安世)**
Brain Research Center, National Tsing Hua University, Taiwan

Memory-guided behaviors allow animals to adapt to dynamic environments. In *Drosophila melanogaster*, the combination of genetic tools and connectome data provides a powerful framework for studying how neural circuits encode, retrieve, and utilize memories. Our analysis of *Drosophila* olfactory networks uncovers hybrid circuit architectures underlying memory encoding. Critically, memory retrieval is not fixed—identical memories can drive diverse decisions across individuals or contexts. We found that aversive olfactory long-term memory (LTM) is significantly more variable than short-term memory, likely reflecting trial-by-trial differences in memory-guided behavior. Using targeted optogenetic activation, we mapped LTM-retrieving circuits and their downstream pathways, linking memory dynamics to navigation and individualized choices. These findings illuminate how memory retrieval shapes context-specific behaviors, providing insights into the neural basis of behavioral individuality.

**Key reference**

1. Abubaker MB, Hsu FY, Feng KL, Chu LA, de Belle JS, Chiang AS (2024). Asymmetric neurons are necessary for olfactory learning in the *Drosophila* brain. *Current Biology*, 34, 1–12.
2. Chen CC, Lin HW, Feng KL, Tseng DW, de Belle JS, Chiang AS (2023). A subset of cholinergic mushroom body neurons blocks long-term memory formation in *Drosophila*. *Cell Reports*, 42, 112974.
3. Lin HW, Chen CC, Jhang RY, Chen L, de Belle JS, Tully T, Chiang AS (2022) CREBB repression of protein synthesis in mushroom body gates long-term memory formation in *Drosophila. Proc Natl Acad Sci USA* 119, e2211308119.
4. Lin HW, Chen CC, de Belle JS, Tully T, Chiang AS (2021) CREBA and CREBB in two identified neurons gate long-term memory formation in *Drosophila*. *Proc. Natl. Acad. Sci. U.S.A.* 118, e2100624118.
5. Lin HH, Chu LA, Fu TF, Dickson BJ, Chiang AS (2013) Parallel neural pathways mediate $CO_2$ avoidance responses in *Drosophila*. *Science* 340:1338-1341.
6. Chen CC, Wu JK, Lin HW, Pai TP, Fu TF, Wu CL, Tully T, Chiang AS (2012) Visualing long-term memory formation in two neurons of the *Drosophila* brain. *Science* 335:678–685.

**Peter McNaughton, PhD**
Professor of Pharmacology, King's College London, UK
Website: https://www.kcl.ac.uk/people/peter-mcnaughton

## Education

| | |
|---|---|
| 1962-1966 | Auckland Grammar School, New Zealand. |
| 1967-1970 | Auckland University, studying for BSc (Hons) in Physics. Sir George Grey Prize for top science student in the University. |
| 1971 | Graduated BSc (Hons), Class I. |
| 1971 | Awarded Rhodes Scholarship for study in Oxford. |
| 1971-1974 | Balliol College, Oxford, studying for D.Phil. in Physiology. Graduated D.Phil. (equivalent of PhD) |

## Awards and Honors

| | |
|---|---|
| 2011 | Prize "Investigación en Dolor 2011" (Investigation in Pain 2011), from the Grünenthal Foundation, Salamanca |
| 2013 | Elected Fellow of the Academy of Medical Sciences |
| 2017 | Elected Fellow of the British Pharmacological Society |

## Selected Publications

1. McNaughton PA, Williams K, Cramp S, Naylor A (2022) Pyrimidine or pyridine derivatives useful as HCN2 modulators. Patent number: WO 2022/185055.
2. McNaughton PA, Williams K, Cramp S, Naylor A (2022) Pyrimidine or pyridine derivatives useful as HCN2 modulators. Patent number: WO 2022/185057.
3. McNaughton PA, Williams K, Cramp S, Naylor A (2022) Pyridine derivatives useful as HCN2 modulators. Patent number: WO 2022/185058.
4. Tsantoulas, C., Ng, A., Pinto, L. G., Andreou, A.P. & McNaughton, P.A. (2022). HCN2 ion channels drive pain in rodent models of migraine. Journal of Neuroscience 42:7513–7529. (NB highlighted in https://www.jneurosci.org/content/jneuro/42/40/7512.full.pdf)
5. Buijs, T.J., Vilar, B., Tan, C.-H. & McNaughton P.A. (2023). STIM1 and ORAI1 form a novel cold transduction mechanism in sensory and sympathetic neurons. EMBO J., 42(3), e111348. doi:10.15252/embj.2022111348. (NB see commentary in https://www.sciencedirect.com/science/article/pii/S0143416023000477?via%3Dihub)
6. Han, X., Pinto, L. G., Vilar, B., & McNaughton, P. A. (2024). Opioid-Induced Hyperalgesia and Tolerance Are Driven by HCN Ion Channels. J Neurosci, 44(6)doi:10.1523/jneurosci.1368-23.2023

# The role of HCN2 ion channels in chronic pain, tinnitus and opioid addiction

**Peter McNaughton, PhD**
Professor of Pharmacology, King's College London, UK

Pain, tinnitus and opioid addiction may seem to have little in common, but recent evidence shows that in all cases important features are driven by one common cause: HCN2 ion channels.

In contrast to all other ion channels, members of the HCN family open in response to membrane *hyperpolarization*. Another unique feature is that two out of the four members of this family, HCN2 and HCN4, are directly activated by the binding of intracellular cAMP. This last feature places these channels in a unique position to trigger an inward current when cAMP levels rise, and thus to stimulate the firing of action potentials in excitable cells. In the heart it is well known that HCN4 plays a critical role in regulating cardiac action potential frequency. In pain-sensitive neurons (nociceptors) HCN2 plays a similar role, and recent work has extended the list to tinnitus and to some of the features of opioid addiction.

In our lab we have found that HCN2 mediates pain, by increasing nociceptor excitability, in animal models of inflammatory pain, such as arthritic and migraine pain, and also in neuropathic pain, such as that induced by nerve damage, chemotherapy and diabetes. Casting the net wider, we have more recently found that auditory tinnitus is also driven by HCN2. In addiction to opioids, HCN2 plays a critical role in important features such as hyperalgesia and opioid tolerance. Developing HCN2-selective blockers may therefore relieve some of the most agonising aspects of human existence.

**Eric J. Huang, MD, PhD**
Edward Mallinckrodt Professor & Chair, Department of Pathology & Immunology, Washington University School of Medicine, St. Louis, U.S.A.

Website: https://pathology.wustl.edu/people/eric-huang-md-phd/

## Education

| | | |
|---|---|---|
| 1986 | M.D. | National Taiwan University, Taipei, Taiwan |
| 1993 | Ph.D. | Weill Cornell Graduate School of Medical Science & Sloan-Kettering Institute, New York, NY |
| 1997 | Ph.D. | University of California, San Francisco |

## Positions

2000 – 2005 Assistant Professor, University of California San Francisco
2005 – 2009 Associate Professor, University of California San Francisco
2009 – 2024 Professor, University of California San Francisco
2019 – 2024 Vice Chair of Research, University of California San Francisco
2025 – now Edward Mallinckrodt Professor & Chair, Washington University School of Medicine, St. Louis, MO
2025 – now Pathologist-in-Chief, Barnes-Jewish Hospital

## Honors (Selected)

2013 Moore Award (AANP Annual Meeting)
2014 Keynote Speaker, 3rd Annual Concussion Symposium, University of Toronto
2015 Robert Terry Award (AANP Annual Meeting)
2016 The DeArmond Lecture (AANP Annual Meeting)
2017 The Stowell Lecture, Dept of Pathology, UC Davis
2018 Keynote Speaker, 27th International Complement Workshop, Santa Fe, NM
2019 Mentoring Award, UCSF BMS Graduate Program
2021 Organizer, Keystone Symposium on Neurodegenerative Diseases
2021 Award of Distinction, Weill Cornell Graduate School of Medical Sciences
2022 Plenary Lecture, ISFTD, Lille, France
2023 Institutional Seminar Speaker, Institute of Biomedical Sciences (IBMS), Academia Sinica
2024 Keynote Speaker, 14th Hersey Meeting on Developmental Brain Injury, Gothenburg, Sweden
2024 Academician, Academia Sinica

**Selected Publications**


1. Lui H, Zhang J, Makinson SR, Cahill MK, Kelley KW, Huang HY, Shang Y, Oldham MC, Martens LH, Gao FY, Coppola G, Sloan SA, Hsieh CL, Kim CC, Bigio EH, Weintraub S, Mesulam MM, Rademakers R, Mackenzie IR, Seeley WW, Karydas A, Miller BL, Borroni B, Ghidoni R, Farese RV, Paz JT, Barres BA, Huang EJ. Progranulin Deficiency Promotes Circuit-Specific Synaptic Pruning by Microglia via Complement Activation. Cell. 2016 May 5;165(4):921-35. PMCID: PMC4860138.
2. Kao AW, McKay A, Singh PP, Brunet A, Huan**g EJ**. Progranulin, lysosomal regulation and neurodegenerative disease. ***Nature Rev Neurosci.*** 2017 Jun;18(6):325-333. PubMed PMCID: PMC6040832.
3. Zhang J, Velmeshev D, Hashimoto K, Huang HY, Hofmann JW, Shi X, Chen J, Leidal AM, Dishart JG, Cahill MK, Kelley KW, Liddelow SA, Seeley WW, Miller BL, Walther TC, Farese RV, Jr, Taylor JP, Ullian EM, Huang B, Debnath J, Wittmann T, Kriegstein AR, **Huang EJ.** Neurotoxic microglia promote TDP-43 proteinopathy in progranulin deficiency. ***Natur***e. 2020 Dec;588(7838):459-465. PMCID: PMC7746606.
4. Marsan E, Velmeshev D, Ramsey A, Patel RK, Zhang J, Koontz M, Andrews MG, de Majo M, Mora C, Blumenfeld J, Li AN, Spina S, Grinberg LT, Seeley W, Miller BL, Ullian EM, Krummel MF, Kriegstein A, Huang EJ. Astroglial toxicity promotes synaptic degeneration in the thalamocortical circuit in frontotemporal dementia with GRN mutations. J Clin Invest. 2023 Mar 15;133(6):e164919. doi: 10.1172/JCI164919. PMID: 36602862; PMCID: PMC10014110.
5. Wang WY, Pan L, Su SC, Quinn EJ, Sasaki M, Jimenez JC, Mackenzie IR, Huang EJ, Tsai LH. Interaction of FUS and HDAC1 regulates DNA damage response and repair in neurons. Nat Neurosci. 2013 Oct;16(10):1383-91. PMCID: PMC5564396.
6. Qiu H*, Lee S*, Shang Y, Wang WY, Au KF, Kamiya S, Barmada SJ, Finkbeiner S, Lui H, Tang AA, Oldham MC, Wang H, Shorter J, Filiano AJ, Roberson ED, Tourtellotte WG, Chen B, Tsai LH, Huang EJ. ALS-associated mutation FUS-R521C causes DNA damage and RNA splicing defects. J Clin Invest. 2014 Mar 3;124(3):981-99. PMCID: PMC3938263.
7. Lee S, Shang Y, Redmond SR, Urisman A, Tang AA, Li KH, Burlingame AL, Pak RA, Jovičić A, Gitler AD, Wang J, Gray NS, Seeley WW, Siddique T, Bigio EH, Lee VMY, Trojanowski JQ, Chan JR, Huang EJ. Activation of HIPK2 Promotes ER Stress-Mediated Neurodegeneration in Amyotrophic Lateral Sclerosis. Neuron. 2016 July 5;91:41-55. PMCID: PMC4938715.
8. Paredes MF, James D, Gil-Perotin S, Kim H, Cotter JA, Ng C, Sandoval K, Rowitch DH, Xu D, McQuillen PS, Garcia-Verdugo JM, Huang EJ, Alvarez-Buylla A. Extensive migration of young neurons into the infant human frontal lobe. Science. 2016 Oct 7;354(6308). pii: aaf7073. PMCID: PMC5436574. (*Co-corresponding author)

# Disentangling immune-vascular interface in the prenatal human brain

**Eric J. Huang, MD, PhD**
Edward Mallinckrodt Professor & Chair, Department of Pathology & Immunology, Washington University School of Medicine, St. Louis, U.S.A.

The germinal matrix harbors neurogenic niches in the subpallium of the prenatal human brain that produce abundant GABAergic neurons. In preterm infants, the germinal matrix is particularly vulnerable to hemorrhage, which disrupts neurogenesis and causes severe neurodevelopmental sequelae. However, the disease mechanism(s) promoting germinal matrix hemorrhage remain unclear. My presentation will focus on recent discoveries using single-cell transcriptomics to uncover novel mechanisms that govern neurogenesis and angiogenesis in the germinal matrix of the prenatal human brain. These approaches also reveal the critical role of immune-vascular interaction that promotes vascular morphogenesis in the germinal matrix and how proinflammatory factors from activated neutrophils and monocytes can disrupt this process, leading to hemorrhage. Collectively, these results reveal fundamental disease mechanisms and therapeutic targets to prevent or treat germinal matrix hemorrhage.

**Florian Lesage**
Institut de pharmacologie moléculaire et cellulaire, Université Côte dAzur / CNRS / Inserm, Sophia Antipolis, France

Website: https://cvscience.aviesan.fr/cv/601/florian-lesage

## Education

| | |
|---|---|
| 1995 | PhD Life Sciences, Université de Nice-Sophia Antipolis |
| 1991 | MSc Université de Nice-Sophia Antipolis |

## Positions

| | |
|---|---|
| 2023-2028 | director, Institut de Pharmacologie Moléculaire et Cellulaire (IPMC) |
| 2018-2023 | deputy director, Institut de Pharmacologie Moléculaire et Cellulaire (IPMC) |
| 2012-2024 | director, Laboratoire d'Excellence - Ion Channel Science and Therapeutics (LabEx ICST) |
| 2020-2023 | Scientific delegate CNRS Biology, for interdisciplinarity, Atip/Avenir program, Conférences J. Monod |
| 2021-present | senior director of research, "classe exceptionnelle" (DRE, Inserm) |
| 2008-2021 | senior director of research (DR1, Inserm) |
| 2006-present | head of the team Molecular physiology and physiopathology of ion channels, IPMC |
| 2003-2013 | contrat d'interface/CHRT, University Hospital of Nice, Department of Neurology |
| 2002-2008 | director of research (DR2, Inserm) |
| 2000-2002 | visiting associate professor, HHMI and The Rockefeller University, NY, USA |
| 1996-2002 | researcher (CR Inserm) |

## Awards

| | |
|---|---|
| 2000 | medal Maurice Nicloux, Société française de biochimie et biologie moléculaire |
| 2000 | fellowship, North Atlantic Treaty Organization |
| 2002 | special prize, Fondation pour la recherche médicale |
| 2008 | laureate of the French National Academy of Medicine (Prize Achard/Medicine) |
| 2009 | invitation fellowship, Japan Society for the Promotion of Science |
| 2015 | Chevalier - French National order of merit |

**Recent Publications**

1. Chatelain et al. (2024). *Mechanistic basis of the dynamic response of TWIK1 ionic selectivity to pH*. **Nat Commun**. 15 (1): 3849
2. Landra-Willm A et al. (2023). *A photoswitchable inhibitor of TREK channels controls pain in wild-type intact freely moving animals.* **Nat Commun.** 14 (1): 1160
3. Glogowska E et al. (2021). *Piezo1 and Piezo2 foster mechanical gating of K(2P) channels*. **Cell Rep**, 37 (9): 110070
4. Cooper A et al. (2020). *Inhibition of histone deacetylation rescues phenotype in a mouse model of Birk-Barel intellectual disability syndrome*. **Nat Commun**, 11 (1): 480
5. Ben Soussia I et al. (2019). *Mutation of a single residue promotes gating of vertebrate and invertebrate two-pore domain potassium channels*. **Nat Commun**, 10 (1): 787
6. Royal P et al. (2019). *Migraine-Associated TRESK Mutations Increase Neuronal Excitability through Alternative Translation Initiation and Inhibition of TREK*. **Neuron**, 101 (2): 232-245.e6

# From potassium selectivity to sodium permeability, a property specific to two pore-domain potassium channels

**Florian Lesage**
Institut de pharmacologie moléculaire et cellulaire,
Université Côte dAzur / CNRS / Inserm, Sophia Antipolis, France

Two pore-domain K2P channels contribute to the background conductance that regulates resting potential and membrane resistance, two key elements of cellular excitability. Some K2P channels behave as signal integrators responding to a wide range of stimuli, including membrane stress, temperature, pH and cellular signals, while others are expressed in specific intracellular compartments. Some K2P channels exhibit dynamic ion selectivity, reversibly switching from inhibitory to excitatory behavior. In my presentation, I will give an overview of these channels as well as recent results related to the dynamic ionic selectivity of TWIK1.

**Ying-Hui Fu (傅瑩惠), Ph.D.**

Professor of Neurology, University of California San Francisco, USA

Website: https://profiles.ucsf.edu/ying-hui.fu

## Education

| | | |
|---|---|---|
| 1980 | B.S. | National Chung-Hsing University; Taichung, Taiwan |
| 1986 | Ph.D. | Ohio State University; Columbus, Ohio, USA |

## Positions, Scientific Appointments, and Honors

2021 Harvard Medical School Division of Sleep Medicine Prize

2018 Outstanding Alumni Award, Chung-Hsing University, Taiwan

2018 Member, National Academy of Medicine, USA

2018 Member, Academia Sinica, Taiwan

2018 Member, National Academy of Science, USA

2015 TEDx, California

2012 Presidential Lecture, University of Vermont

2012 Faculty Research Lecture in Basic Research, UCSF

2009 Distinguished Visiting Professorship, Tamkang University, Taiwan

2008 Distinguished Guest, Bollum Symposium, University of Minnesota, Minneapolis, MN

2006 Bauer Foundation Colloquium Distinguished Guest, Brandeis University, Boston, MA

2006 Sleep Science Award, American Academy of Neurology

2006-present Professor, Department of Neurology, University of California at San Francisco

2002-2006 Associate Professor, Department of Neurology, University of California at San Francisco

1998-2002 Research Associate Professor, University of Utah

1997-1998 Senior Scientist, University of Utah

1995-1997 Senior Scientist, Darwin Molecular Corp.

1993-1995 Scientist, Millennium Pharmaceuticals, Inc.

## Selected Publications:


1. **Fu Y-H**, Kuhl DP, Pizzuti A, Pieretti M, Sutcliffe JS, Richards S, Verkerk AJ, Holden JJ, Fenwick RG Jr, Warren ST, Oostra BA, Nelson DL, Caskey CT. Variation of the CGG repeat at the fragile X site results in genetic instability: resolution of the Sherman paradox. *Cell* 1991 Dec 20; 67(6):1047-1056. PMID:1760838
2. **Fu Y-H**, Pizzuti A, Fenwick,Jr., RG, King J, Rajnarayan S, Dunne PW, Dubel J, Nasser GA, Ashizawa T, DeJong P, Wieringa B, Korneluk R, Perryman BM, Epstein HF, Caskey CT. An unstable triplet repeat in a gene related to myotonic muscular dystrophy. *Science* 1992; 255(5049):1256-1258. PMID:1546326
3. Caskey CT, Pizzuti A, **Fu Y-H**, Fenwick RG Jr, Nelson DL. Triplet repeat mutations in human disease. *Science* 1992 May 8; 256(5058):784-789. PMID:1589758
4. **Fu Y-H**, Friedman DL, Richards S, Pearlman JA, Gibbs RA, Pizzuti A, Ashizawa T, Perryman MB, Fenwick RG Jr, Caskey CT. Decreased expression of myotonin-protein kinase messenger RNA and protein in adult form of myotonic dystrophy. *Science* 1993 Apr 9; 260(5105):235-238. PMID:8469976
5. Toh KL, Jones CR, He Y, Eide EJ, Hinz WA, Virshup DM, Ptáček LJ, **Fu Y-H**. An h*Per2* phosphorylation site mutation in familial advanced sleep-phase syndrome. *Science* 2001; 291:1040-3. PMID:11232563
6. Xu Y, Padiath QS, Shapiro RE, Jones CR, Wu SC, Saigoh N, Saigoh K, Ptáček LJ, **Fu Y-H**. Functional consequences of a *CKIδ* mutation causing familial advanced sleep phase syndrome. *Nature* 2005; 434(7033):640-4. PMID:15800623
7. Hirano A, Shi G, Jones CR, Lipzen A, Pennacchio LA, Xu Y, Hallows WC, McMahon T, Yamazaki M, Ptáček LJ, **Fu Y-H**. A Cryptochrome 2 mutation yields Advanced Sleep Phase in human. *Elife* 2016 Aug 16;5:e16695. PMCID:PMC5398888
8. Kurien P, Hsu P-K, Leon J, Wu D, McMahon T, Shi G, Xu Y, Lipzen A, Pennacchio LA, Jones CR, **Fu Y-H**, Ptacek LJ. TIMELESS mutation alters phase responsiveness and causes advanced sleep phase. *Proc Natl Acad Sci U S A.* 2019 Jun 11; 116(24):12045-12053. PMCID:PMC6575169

# Piecing together the SLEEP puzzle: genes, molecules, and circuits

**Ying-Hui Fu (傅瑩惠), Ph.D.**

Professor of Neurology, University of California San Francisco, USA

Sleep occupies a significant portion of our daily lives, yet our understanding of sleep, in general, is minimal. Sleep of sufficient duration, continuity, and intensity is necessary to promote high levels of cognitive performance during the wake period and prevent physiological changes that may predispose individuals to many adverse health outcomes. Sleep disruption and insufficiency is prevalent in our society due to the high demand for work, school, and many environmental factors, thus significantly contributing to many health conditions we face. Over the last twenty-five years, we have focused on studying sleep schedule and duration, both of which are highly genetical wired. Interestingly, we found that the biological need for sleep varies dramatically among humans. We have identified “Familial Advanced Sleep” and “Familial Natural Short Sleep (FNSS)” people with unusual sleep behaviors and have used human genetics approach to identify many genes/mutations that give them these unusual sleep behaviors. Mouse models recapitulate the human condition, and in vitro molecular and neurocircuitry studies offer insight into the underlying mechanisms. Because of sleep's fundamental role in our health, the pathways regulating sleep are intertwined with those regulating other functions. Thus, our method also offers opportunities to investigate how sleep can impact other conditions, including mood, pain, and other disease pathology.

**Dr. Chen, Chih-Cheng 陳志成**

Distinguished Research Fellow

Institute of Biomedical Sciences, Academia Sinica,

https://www.ibms.sinica.edu.tw/chih-cheng-chen/en/

chih@ibms.sinica.edu.tw

## A role for proprioceptors in sngception

The perception of acid-sensation can be regarded as one of the most mysterious somatosensory functions, which are engaged in a wide range of somatosensory neurons, including nociceptors, pruriceptors, and non-nociceptive mechanoreceptors (e.g., proprioceptors). 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 */səŋ/*), 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. We have previously demonstrated sng and pain are 2 independent symptoms with differential clinical impacts in fibromyalgia and lumbar radicular low-back pain. To understand how sngception works, we first demonstrated that intramuscular injection of pH5.2 acidic saline induced sng but not pain in humans. Furthermore, we combined genetic, chemo-optogenetic, pharmacological, and electrophysiological approaches, to probe the molecular and neurobiological basis of sngception in mouse models of fibromyalgia. We found genetic deletion of Asic3 in proprioceptors but not in nociceptors abolished acid-induced chronic hyperalgesia in mice. Chemo-optogenetically activating proprioceptors results in hyperalgesic priming that favored chronic pain induced by acidosis. Together, we showed sng and pain can be segregated in humans and proprioceptors are sngceptors involved in acid-induced pain chronicity. Also, ASIC3 of proprioceptor is the major sng transducer to sense acidosis.

**Dr. Pan, Ming-Kai 潘明楷**

Joint Appointment Assistant Research Fellow, Institute of Biomedical Sciences, Academia Sinica

https://www.ibms.sinica.edu.tw/pan-ming-kai/en/

emorymkpan@ntu.edu.tw

## Neural dynamic approaches to cerebellar motor control

My key research focus is investigating the neural dynamic mechanisms for cerebellar motor control in health and diseases. Using single-unit recording and optogenetics in mice and clinical electrophysiology in humans, we have identified the pathophysiology of essential tremor, the most common movement disorder with the defining feature of action tremor, a rhythmic involuntary movement. We have identified mutually referenced mouse and human evidence that GluRδ2 loss in cerebellar Purkinje cells causes climbing fiber overgrowth, which leads to excessive cerebellar oscillations and tremors. A follow-up study showed that the olivocerebellum utilizes neuronal population codes to compute tremor frequency (e.g. how the cerebellum computes the “7” of a “7Hz” tremor). This study reveals that the cerebellum frequency codes for motions in a disease state. We next investigated the frequency coding mechanisms in cerebellar ataxias with a defining feature of loss of motor rhythm. In both mouse and patients, we found that the CF regression in the cerebellum causes loss of cerebellar rhythm that leads to loss of corresponding motor rhythm and ataxia. The degree of rhythm loss is also correlated with ataxia severity in patients. As a unifying theory, we also identified that the cerebellum uses neuronal population codes to compute instantaneous motor frequencies for motor kinematics, with numerical precision and cross-individual uniformity. This level of precision allows us to create mouse motions with designed frequency dynamics, and manipulate the frequency stability of volitional human motions.

**Dr. Cheng, Sin-Jhong 鄭信忠**

Associate Research Scientist

Institute of Biomedical Sciences, Academia Sinica

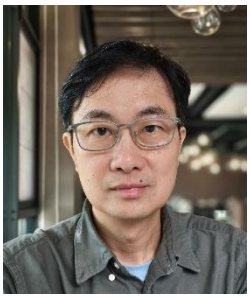
https://www.ibms.sinica.edu.tw/sin-jhong-cheng/en/

ashinn420@ibms.sinica.edu.tw

## Management and services of the Neuroscience core facility

My primary responsibility is to manage and develop the neuroscience core facility, providing electrophysiological technology support and neuroimaging recording services for researchers in Taiwan. Electrophysiology is a powerful tool in neuroscience that studies the connectivity and function of the nervous system by recording electrical activity in neurons and neuronal ensembles. Techniques such as field potential or whole-cell patch-clamp recordings are commonly used. These techniques allow for recording intracellular signal/intrinsic cell properties changes from single cells or monitoring synapse signal connections across brain regions from the extracellular space. Intracellular recordings are usually applied to cultured cells or slices. The core facility assists approximately 30 labs and provides teaching or assistance to more than 40 students and assistants annually.

Conducting in vivo recordings in a complex brain environment is much more challenging than in vitro recordings. However, electrophysiology is a powerful analytical technique investigating the correlation between behavior and in vivo neuronal dynamics in freely behaving animals. Therefore, our current focus is on establishing new platforms, including high-speed photography systems and NeuLive in vivo local field potential (LFP) recording platforms to address this issue. Through our core technical support and services, 33 papers were published in international journals and my name is listed as a coauthor in 6 published papers in the past five years.



**Dr. Lo, Chung-Chuan 羅中泉**

Professor, Institute of Systems Neuroscience

National Tsing Hua University

http://life.nthu.edu.tw/~lablcc

cclo@mx.nthu.edu.tw

# From Neural Stalemates to Robust Decisions: Insights into Goal-Directed Control in Drosophila Brains

Insects are evolutionary marvels, renowned for their ability to survive and adapt across diverse environments. These extraordinary capabilities are driven by their compact brains, which excel in efficient neural computation through small, highly specialized circuits. Recent advances in connectomics of fruit fly brains have unlocked unprecedented opportunities to explore the neural mechanisms underlying insect behavior in remarkable detail.

In this presentation, I will discuss recent studies and our latest findings on the neural circuits governing steering control in fruit flies. By analyzing connectomic data and constructing computational models, we have identified intricate circuits within the lateral accessory lobes (LAL) that exhibit a remarkable balance of robustness and flexibility, surpassing mechanisms proposed in earlier studies. Specifically, we uncovered two visual pathways that converge in the LAL, enabling the integration of sensory information to make steering decisions. Notably, the LAL circuits resolve conflicts between a desired goal and an aversive stimulus, as well as overcome conditions where neural signals result in a standstill—a phenomenon we term neural stalemate.

Our findings shed light on the sophisticated neural control mechanisms in small brains and offer inspiration for developing novel control theories for next-generation neuromorphic navigation systems in miniature drones and robots.

**Dr. Hsu, Ching-Lung 徐經倫**

Assistant Research Fellow

Institute of Biomedical Sciences, Academia Sinica

https://www.ibms.sinica.edu.tw/ching-lung-hsu/en/

hsuc@ibms.sinica.edu.tw

# Biophysics-Informed One-Shot Synaptic Algorithms for Learning Place Fields in Precise Memory-Dependent Spatial Behavior

Decades of Neurophysiology successfully established that neurons in the hippocampus, a region crucial for space-related memory, use cellular biophysics to perform experience-dependent computations. Meanwhile, 50-years neuropsychological analyses of place cells (pyramidal cells as spatial correlates) and their receptive fields delineated algorithms underlying navigational behaviors. Nevertheless, these bottom-up and top-down approaches are not causally connected.

The hippocampus, as the “fast learner”, substantiates flexible goal-directed behavior by encoding one instance and generalizing information toward the complementary cortico-hippocampal system. Here, we leveraged our *in-vivo* patch-clamp experiments, showing incredibly fast (seconds) formation of new CA1 place-cell firing in mice exhibiting lick patterns as anticipation and triggers for spatially precise rewards in VR. In acute brain slices, we identified relevant dynamic characteristics of distributed circuit inputs over the dendritic tree, driving large $Ca^{2+}$-based depolarization (dendritic plateau potentials). Dendritic plateaus induced one-shot synaptic potentiation (LTP), with temporal properties explaining place-field formation in numerical simulations. We reason that theta-rhythms and ramping of frequency which organized output from the upstream circuits support comparisons of their behavioral states in CA1 dendrites, resulting in a permissive condition for rapid, powerful LTP, and neuromodulatory feedback reflecting rewards or goals “instructs” plateaus and weight update as a third learning factor. Our data are consistent with this idea, and modeling-assisted pharmacological dissection revealed biophysics of NMDARs, R-type $Ca^{2+}$ and TRPM4 channels support this algorithm.

Our work suggests biophysics shapes one-shot learning algorithms for feature selectivity of hippocampal memory.

**Dr. Hwang, Dennis W. 黃聖言**

Associate Research Fellow

Institute of Biomedical Sciences, Academia Sinica

https://www.ibms.sinica.edu.tw/dennis-hwang/en/

dwhwang@ibms.sinica.edu.tw

# Advances in Dynamic Imaging Techniques in Neuroscience: A Multifaceted Analysis of Metabolism and Vascular Function

Dynamic imaging techniques are revolutionizing our ability to probe brain metabolism and vascular function with unprecedented temporal and spatial resolution. By integrating two cutting-edge approaches—dynamic glucose-enhanced imaging (DGE) and dynamic contrast-enhanced imaging (DCE)—this study unveils novel insights into metabolic dysregulation and vascular pathophysiology across neurodegenerative and neurovascular disorders.

Through DGE, we delineate spatially heterogeneous glucose uptake patterns in glioblastoma models, revealing intratumoral cellular subpopulations defined by their glycolytic activity. In Huntington's disease (HD) models, longitudinal DGE analysis captures a biphasic metabolic trajectory: early-stage stability (5–6 weeks) transitions to mid-stage hypometabolism (10 weeks), culminating in late-stage compensatory hypermetabolism (12 weeks) within striatal and cortical circuits. This metabolic plasticity coincides with GPI1 overexpression, suggesting a dynamic interplay between neuronal degeneration and glial-driven metabolic adaptation.

Concurrently, DCE imaging with gadolinium-based tracers exposes early vascular permeability alterations in CADASIL mouse models, detectable prior to overt neuropathological manifestations. Quantitative mapping of blood-brain barrier integrity reveals region-specific permeability gradients that spatially correlate with emerging white matter hyperintensities and microinfarct development.

Our findings establish dynamic imaging as a dual-purpose paradigm in modern neuroscience. The technique not only deciphers spatiotemporal glucose dynamics to pinpoint disease-specific energy crises but also resolves incipient vascular dysfunction as a sentinel biomarker for neurodegenerative cascades. By bridging metabolic and vascular neuroscience, this work provides a unified framework for early therapeutic intervention, with implications spanning cerebral small vessel diseases to triplet repeat disorders. The integration of these modalities promises to redefine our approach to decoding brain pathophysiology across the neurovascular-metabolic continuum.

**Dr. Lee, Kuo-Sheng 李國昇**

Assistant Research Fellow

Institute of Biomedical Sciences, Academia Sinica

https://www.ibms.sinica.edu.tw/kuo-sheng-lee/en/

leeku@ibms.sinica.edu.tw

# Exploring the Fundamental Circuit Mechanisms Underlying Somatosensation

Somatosensation enables organisms to detect and respond to mechanical, thermal, and nociceptive stimuli, providing a critical interface between the external environment and internal physiological states. Despite significant progress in sensory neuroscience, the precise mechanisms by which the nervous system encodes, processes, and adapts to somatosensory inputs remain poorly understood. My future research seeks to bridge these knowledge gaps by integrating advanced optical imaging, computational modeling, and functional circuit mapping to explore somatosensory circuit dynamics at multiple levels of the neuraxis. This research will focus on four key areas: (1) Biomechanical and functional simulation of Pacinian corpuscles (PCs) in interoception, investigating how these mechanoreceptors contribute to visceral sensing; (2) All-optical circuit mapping and plasticity analysis in the dorsal root ganglion (DRG) and dorsal column nuclei (DCN) to uncover how somatosensory information is encoded and transmitted; (3) In situ investigation of multicellular circuits within muscle spindles, revealing their role in proprioception and movement control; and (4) Nociceptive neuraxis dysfunction in neuropathic pain and migraine, examining maladaptive plasticity from the periphery to the cortex. By integrating experimental and computational approaches, this work will provide a comprehensive understanding of somatosensory processing across hierarchical levels, offering new insights into sensory perception, pain mechanisms, and potential therapeutic interventions for neurological disorders.

**Dr. Chang, Chun-hui 張鈞惠**

Professor, Institute of Systems Neuroscience

National Tsing Hua University

https://tsinghuachanglab.wixsite.com/changlab

changch@life.nthu.edu.tw

# Aberrant Activation of the Orbitofrontal Cortex on Fear Regulation: A Systems Neuroscience Study

Hyperactivation of the orbitofrontal cortex (OFC) is commonly seen in obsessive-compulsive disorder (OCD) patients. Moreover, many of the OCD patients have anxiety issues, and there is high comorbidity of OCD and post-traumatic stress disorder (PTSD). Based on this clinical premise, we think that related brain structures must have interacted at the circuitry level. The key candidate for the interaction is the basolateral amygdala (BLA), the emotion center in the brain, which receives inputs from neocortical areas including the medial prefrontal cortex (mPFC) and lateral orbitofrontal cortex (lOFC). Indeed, it is well documented that input from the mPFC to the amygdala is critical for proper fear regulation after extinction, and malfunction of the mPFC-BLA pathway has been suggested as the leading cause of PTSD. In a series of studies, we assessed how aberrant activation of the lOFC mimicking OCD condition may have interfered with the regulation of fear at the behavioral and neurobiological level. We first examined the hypothesis that hyperactivation of the lOFC negatively affects fear extinction. In behaving rats, lOFC was pharmacologically activated during the encoding phase of Pavlovian fear extinction, and we found that aberrant lOFC activation during extinction training resulted in impaired encoding of extinction demonstrated by a general up-shift of fear levels during retrieval test compared to controls. To further examine how aberrant lOFC activation interferes with the fear-related circuit, the activation levels of non-GABAergic and GABAergic neurons were quantified using c-Fos signals as indexes during the late phase of extinction. Our data showed that pharmacological activation of the lOFC during extinction increased the neuronal activities on the injection side(s) in the mPFC, the lateral amygdala (LA), the BLA (preferentially the non-GABAergic neurons), and the medial intercalated cells (mITC; preferentially the right side). The c-Fos assessment results at the end of the extinction acquisition session indicated that lOFC activation disturbed the excitatory/inhibitory balance of neuronal activities in numerous fear-related brain regions, in that increased neuronal activities were robust in immediate downstream areas that received lOFC inputs (mPFC and LA), relayed downward (BLA and mITC), but did not last into the central amygdala (CeA), the major output interface of the amygdala. We proposed that persistent aberrant lOFC activation acted like a source of disturbance, but the excitatory drive eventually diminished after multi-synaptic signal transduction and complicated information processing within local amygdala circuit. It is worth noticing that the animals were acquiring the fear extinction under disturbed excitatory/inhibitory balance in the mPFC and the LA/BLA complex, and such condition may have interfered with the proper plasticity necessary for the acquisition and consolidation of extinction memory. Through the intensive connections of the lOFC with fear-related brain regions, the lOFC may exert its impact on fear expression and extinction acquisition. Even though acute activation cannot be viewed as equivalent to the chronic condition in OCD patients, aberrant activation of the lOFC in whatever situation may initiate its interaction with the fear-related circuit and leading to deteriorated fear control if the circumstances cannot be properly dissolved.

**Dr. Wang, Guey-Shin 王桂馨**

Associate Research Fellow

Institute of Biomedical Sciences, Academia Sinica

https://www.ibms.sinica.edu.tw/guey-shin-wang/en/

gswang@ibms.sinica.edu.tw

# Investigating the Neural Pathogenesis of Myotonic Dystrophy: From Neurodevelopmental Disorders to Neurodegeneration

Lee-Hsin Wang[1], Hsiang-Ling Yu[1, 2], Pei-Ying Wang[1], Ting-Yu Kuo[1], Guey-Shin Wang[1,2]
[1]Institute of Biomedical Sciences, Academia Sinica, Taipei, Taiwan
[2]Program in Molecular Medicine, National Yang Ming Chao Tung University and Academia Sinica, Taipei, Taiwan

Cognitive impairments are commonly seen in individuals with myotonic dystrophy type 1 (DM1), including mental retardation, autism spectrum disorder (ASD), depression, attention deficit hyperactivity disorder (ADHD), and neurodegeneration. The genetic basis of DM1 is caused by an expansion of CTG repeats in the 3' untranslated region (UTR) of DMPK gene. The severity of symptoms increases with repeat length, while the age of onset decreases as repeat length grows. Nuclear accumulation of *DMPK* mRNA containing expanded CUG repeats disrupts the activities of RNA binding protein families such as muscleblind like (MBNL) and CUGBP Elav-like family member (CELF). To investigate the pathogenesis of adult-onset DM1, we established a mouse model, EpA960/CaMKII-Cre, for expression of expanded CUG (CUGexp) RNA in postnatal neurons. EpA960/CaMKII-Cre mice recapitulated DM1 features including learning disability, neurodegeneration and dysregulation of RNA processing. We identified features of neurodegeneration associated with DM1, including impaired retrograde BDNF signaling due to loss of MBNL1 function and the activation of a developmental RNA processing program triggered by calpain-mediated degradation of MBNL2. We are currently investigating the role of neuroinflammation in synapse loss during the early stages of disease progression. We also generated mouse models for investigating the pathogenesis of congenital and childhood-onset DM1. We found that expression of $CUG^{exp}$ RNA in neural progenitors induced p53-mediated apoptosis resulting in partial agenesis of the corpus callosum and brain atrophy. We are investigating how $CUG^{exp}$ RNA impairs neurogenesis. We hope that a better understanding of pathogenesis will provide insights for developing more effective therapeutic strategies for treating DM1.

**Dr. Yang, Shi-Bing 楊世斌**

Associate Research Fellow

Institute of Biomedical Sciences, Academia Sinica

https://www.ibms.sinica.edu.tw/shi-bing-yang/en/

sbyang@ibms.sinica.edu.tw

## Neural circuit basis of social investigative behaviors

Social behaviors are critical for survival and are governed by complex neural circuits. This study explores how internal states, particularly those encoded by hypothalamic neurons, drive social investigative behaviors in mice. We focused on the ventromedial hypothalamic nucleus (VMH) as a central hub for innate behaviors, including aggression, mating, and social interactions. Using fiber photometric recordings of VMH neurons, we demonstrated that these neurons respond robustly to social stimuli, including conspecific interactions, and are closely linked to investigative behaviors. Our results further reveal that VMH neurons show distinct activity patterns depending on the sensory cues received, such as pheromonal signals. Additionally, we employed machine-learning-based decoders to predict social behaviors based on neuronal activity. Disruption of pheromone processing or silencing specific pathways altered social behavior, suggesting a vital role for the hypothalamic neurons in encoding and processing social information. These findings provide new insights into the neural mechanisms underlying social behavior, emphasizing the dynamic interaction between internal states and external stimuli in the regulation of social interactions.

**Dr. Lin, Wan-Chen 林宛蓁**

Assistant Research Fellow

Institute of Biomedical Sciences, Academia Sinica

https://www.ibms.sinica.edu.tw/wan-chen-lin/en/

wchlin@ibms.sinica.edu.tw

## Engineering high-performance optogenetic tools for specialized neural inhibition

Optogenetics is a powerful technology that employs light to control genetically targeted cells with high spatial and temporal precision. While the technology has been used extensively by neuroscientists, its application in manipulating synaptic transmission remains challenging. My laboratory aims to engineer high-performance optogenetic tools for robust inhibition of neurotransmission at pre- and post-synaptic sites. We use HcKCR1, a recently discovered kalium channelrhodopsins (KCR) from *Hyphochytrium catenoides*, as the engineering template. KCRs are potentially advantageous over prevailing opsins (e.g., NpHR and ArchT) regarding the unwanted chloride accumulation or pH change after prolonged activation of those ion pumps. We first improve the expression and axonal localization of HcKCR1 for presynaptic inhibition. The resulting axon-targeted HcKCR1 (HcKCR1.AT) can efficiently travel to both ipsilateral and contralateral CA1 regions when the KCR-encoded adeno-associated virus (AAV) infects the CA3 principal neurons in the mouse hippocampus. When recording electrically evoked excitatory postsynaptic potentials (EPSPs) in CA1 pyramidal cells, a brief flash of green light can cause a strong reduction in the EPSP amplitude. To further enhance the opsin's neuron-silencing performance in behaving animals, we also optimize the spectral and gating properties of HcKCR1. Moreover, we are engineering synapse-targeted opsins for suppressing neurotransmission on the postsynaptic sites. Through these efforts, we hope to provide new and better tools to enable neurobiology studies that were previously hindered by the limited power of inhibitory opsins.

**Dr. Huang, Yi-Shuian 黃怡萱**

Research Fellow

Institute of Biomedical Sciences, Academia Sinica

https://www.ibms.sinica.edu.tw/yi-shuian-huang/en/

yishuian@ibms.sinica.edu.tw

# CMTR1-catalyzed mRNA capping in neurogenesis and brain development

Eukaryotic mRNAs are capped with a 7-methylguanosine ($^{m7}$G) via triphosphate bond at the 5' end of mRNAs, which is important for nuclear export, stability and cap-dependent translation of mRNAs. Unlike yeast mRNAs which contain only primitive $^{m7}$G cap (i.e. cap0, $^{m7}$GpppNN, N: any nucleotide), cap methyltransferase 1 (CMTR1) catalyzes 2′-O-ribose methylation of the first transcribed ribonucleotide in higher eukaryotic organisms to generate cap1 ($^{m7}$GpppN$_m$N) structure. Previous studies indicated that cap1 methylation could promote translation and mRNA stability, and mask cellular mRNAs from being recognized as foreign RNAs. Nevertheless, our study found that CMTR1 deficiency impairs dendritic development by affecting *Camk2α* transcription rather than activating innate immunity in both cultured neurons and *in vivo*.

CMTR1-conditional knockout (cKO) mice generated by crossing fCmtr1 (floxed *Cmtr1* allele) and Emx1-Cre mice have reduced cerebral volume and cortical layer thickness, and exhibit abnormal social behavior. Moreover, the reduced number of cortical neurons is mainly caused by impaired proliferation of embryonic neural stem cells (eNSCs). Although the expression of nearly all transcripts encoding ribosomal subunits was downregulated in CMTR1-KO eNSCs, we observed no evident decrease in global protein synthesis. The proteomic analysis revealed that almost all ribosomal subunits expressed normally at the protein level. Currently, we are comparing both transcriptomic and proteomic data to identify how CMTR1 promotes eNSC proliferation.

Keywords: CMTR1, mRNA capping, neurogenesis, cortical development

## Poster Presentation (Poster Session Group A, 15:05-16:25, March 11)

Venue: B1, Life Science Building II, NTHU

| No. | Name | Title |
|---|---|---|
| 1 | Patrick C.H. Hsieh 謝清河 | Gut Microbiota Modulation by Immunosuppression and Cardiac Cell Therapy in a Nonhuman Primate Ischemia/Reperfusion Model of Cardiac Regeneration |
| 3 | Shih-Lei Ben Lai 賴時磊 | Characterize novel regulators of heart regeneration revealed by comparative time-ordered gene coexpression network (TO-GCN) |
| 5 | Ya-Jen Chang 張雅貞 | Pulmonary fibroblast-derived stem cell factor promotes neutrophilic asthma by augmenting IL-17A production from ILC3s |
| 7 | Cathy S.-J. Fann 范盛娟 | Precision Risk Prediction for Endotypes of Complex Diseases |
| 9 | Yungling Leo Lee 李永凌 | Dendritic Cell-Targeted Liposome for Cancer Immunotherapy via Inhibition of Aryl Hydrocarbon Receptor |
| 11 | Jr-Wen Shui 徐志文 | Loss of Paneth-specific RNF186 disrupts Notch/Wnt gradients leading to Type 2 remodeling |
| 13 | Che-Ming Jack Hu 胡哲銘 | Divulging tumor-reactive T cells through biomaterials innovation |
| 15 | Kevin Tsai 蔡松智 | m6A methylation of mRNAs protects the poly(A) tails of cellular and viral transcripts |
| 17 | Tai-Ming Ko 柯泰名 | Decoding Immune Complexity in Circulating Cells with Advanced Single-Cell Multi-Omics and Multi-Modal Approaches for Inflammatory and Vascular Diseases |
| 19 | I-Hsuan Wang 王宜萱 | The roles of host membrane trafficking system at the late stage of influenza virus and SARS-CoV-2 infection |
| 21 | Yang Cheng 鄭 揚 | Highly activated intrahepatic HBV-specific resident-memory T and B cells correlated with acute-on-chronic liver failure |
| 23 | Ling-Hui Li 李玲慧 | The Taiwan Precision Medicine Initiative: A Cohort for Large-Scale Studies |
| 25 | Hsin-Chen Tsai 蔡幸真 | Epigenetic modulation of polyamine biosynthetic pathways rectifies T cell dysfunction to enhance anti-tumor immunity |

| No. | Name | Title |
|---|---|---|
| 27 | Huan-Yuan Chen 陳煥源 | Pathological research in diseases is improved with studies with Inflammation-related approaches |
| 29 | Yao-Ming Chang 張耀明 | Identify Evolutionary Conserved Sub-Cell Types by Cross-Species Single-Cell Transcriptome Data Integration |
| 31 | Yu-Ru Lee 李育儒 | Targeting Novel Post-translational Modifications and Long Non-coding RNA for Cancer Immunotherapy |
| 33 | Chia-Wei Li 李家偉 | Improve CAR-T therapy by single cell repertoire sequencing |
| 35 | Shu-Ping Wang 王書品 | An Oncogenic Ubiquitin E3 ligase Controls the HIPK2–Slug Axis in Lung Cancer Metastasis |
| 37 | Hung-Hsin Chen 陳弘昕 | Medical Phenome-Wide Genomic Profiling and Risk Prediction in Han Chinese from Taiwan Precision Medicine Initiative |
| 39 | Yu-Feng Hu 胡瑜峰 | Aging-induced metabolic dysregulation disrupts sinoatrial node rhythmicity |
| 41 | Chuang-Rung Chang 張壯榮 | Cryo Soft X-ray Tomography Study of Yeast Mitochondrial Conformation. |
| 43 | Yi-Shiuan Liu 劉懿璇 | Rejuvenation of anti-cancer responses using novel multifunctional nanoparticles to block CTLA-4 |
| 45 | Pin-Chao Liao 廖品超 | Mitochondrial Quality Control by the Ubiquitin-Proteasome System: Mitochondria-Associated Degradation (MAD) |
| 47 | Mei-Chuan Peng 彭美娟 | Investigate the role of Phospholipase A2-activating protein (PLAP) in mitochondrial quality control and healthspan in Drosophila |
| 49 | Alexander James White 白安雷 | Desynchronizing the Olivo-Cerebellar Loop: A Computational Study of Pharmacological Intervention for Climbing Fiber Overgrowth Induced Essential Tremor. |
| 51 | Keng-Han Lee 李庚翰 | Constructing a Reward-Based 2D Treadmill System with Visual Recognition for Navigation and Spatial Memory in Virtual Reality for Drosophila melanogaster |
| 53 | Jhih-Yu Hsu 許芝瑜 | Agent-based disease simulator |
| 55 | Sin-Bo Liao 廖信博 | Inhibition of Pfrx/pfkb-1.1 in neuron improves lifespan and healthspan in Drosophila and C.elegans |

| No. | Name | Title |
|---|---|---|
| 57 | Yen-Hung Lin 林彥宏 | Phosphatidylinositol Transfer Protein-1 Regulates Lifespan by Modulating TOR Signaling in C. elegans |
| 59 | Yi-Ling Wu 吳苡伶 | Novel Naphthyridones Targeting Pannexin 1 for Colitis Management |
| 61 | Zhong-Ting Chang 張仲廷 | The role of Xiap in colonic Treg stability and pathogenicity in inflammatory bowel disease progression |
| 63 | Ming-Hong Chao 趙明鴻 | Molecular mechanism of KIF2C in DNA damage repair |
| 65 | Tang-Yu Zhu 朱堂瑀 | Beyond mitosis: investigating the function of nuclear envelope assembly proteins in sexual reproduction |
| 67 | Pei-Yi Kao 高珮翊 | ESCRT-III Independent Nuclear Envelope Sealing: The Role of Vid27 |
| 69 | Hung-Yu Chen 陳虹妤 | Exploring an Overlooked Class of Micronuclei Using a Unique Fission Yeast, Schizosaccharomyces japonicus |
| 71 | Tu-Ting Shih 施宇廷 | TBA |

## Poster Presentation (Poster Session Group B, 10:30-11:45, March 12)

Venue: B1, Life Science Building II, NTHU

| No. | Name | Title |
|---|---|---|
| 2 | Yi-Ling Lin 林宜玲 | Dengue Virus Evolution and Epidemic Potential |
| 4 | Ruey-Bing Yang 楊瑞彬 | The Biology of the SCUBE Protein Family |
| 6 | Song-Kun Shyue 徐松錕 | Antibody targeting monocyte-derived macrophages to M2 for the treatment of inflammatory brain diseases |
| 8 | Mi-Hua Tao 陶秘華 | Chimeric RBD library: A novel strategy to develop a broadspectrum Sarbecovirus vaccine |
| 10 | Jer-Yuarn Wu 鄔哲源 | Clinical Impact of Pharmacogenetic Risk Variants in a Large Chinese Cohort |
| 12 | Yi-Chang Chang 張以承 | PTGR2 inhibition as a therapy for treating diabetes and obesity |
| 14 | Chien-Hsiun Chen 陳建勳 | Predicting Human Height |
| 16 | Yen-Hui Chen 陳燕輝 | Laboratory Animal Facility and Pathology Core Laboratory |
| 18 | Yen-Chun Ho 何彥君 | Differential Mechanisms of Aortic and Mitral Valve Diseases and the Role of Lymphatics in Vascular Disorders |
| 20 | Ying-Han Chen 陳英翰 | Interplay between environmental microbes and the immune system |
| 22 | Yen-Li Li 李彥莉 | Decoding the molecular mechanisms of viral evasion and host defense |
| 24 | Yuh-Shan Jou 周玉山 | Nuclear glycosylated SLC29A2 acts as an oncogene and promote tumor progression in hepatocellular carcinoma |
| 26 | Hsiu-Ming Shih 施修明 | A SUMO binder selectively regulates lncRNA stability |
| 28 | Wen-chang Lin 林文昌 | Developing a bioinformatics tool to visualize tissue-specific expression profiles of protein-coding genes related with sexual dimorphism |
| 30 | Sheau-Yann Shieh 謝小燕 | BTG3 in epidermal health and disease |
| 32 | Woan-Yuh Tarn 譚婉玉 | DDX3 in Lipid Raft-facilitated Transport of PD-L1 |

| No. | Name | Title |
|---|---|---|
| 34 | Steve Roffler 羅傳倫 | Glycosidic switch liposomes for cancer therapy |
| 36 | Shih-Yu Chen 陳世淯 | Single Cell Systems-Structured View of Immune Responses |
| 38 | Fu-An Li 李福安 | Services of Common Equipment Core |
| 40 | Choo Hock Tan 陳子福 | Comparative Proteomics of King Cobra Venom: Geographical Variations and Molecular Insights |
| 42 | Hui-Chun Cheng 鄭惠春 | Human Cep57 regulates the centrosome through phase separation |
| 44 | Ya-Ching Chou 周雅菁 | Study the pathogenesis of endometriosis |
| 46 | Hsiao-Han Chang 張筱涵 | The application of mathematical modeling and pathogen genomics to infectious diseases |
| 48 | Hsiao-Hsi Hsiao 蕭曉希 | Cardioprotective Potential of Sodium-Glucose Cotransporter 2 Inhibitors in Doxorubicin-Induced Cardiotoxicity |
| 50 | Cheng-Sheng Lee 李政昇 | Cohesin-mediated loop extrusion promotes search for intrachromosomal homologous template to facilitate DNA double-strand break repair |
| 52 | Ching-Che Charng 強敬哲 | Hybrid Neural Networks in the Mushroom Body Drive Olfactory Preference in Drosophila |
| 54 | Chen-Chieh Liao 廖晨絜 | Sensorimotor transformation in Drosophila lateral accessory lobe |
| 56 | Qing Chen 陳慶 | Investigate the role of Sirt1-UPRmt in muscular atrophy in zebrafish |
| 58 | Hsien-Chu Wang 王嫻築 | Engineering Mechanosensitive Channels and Auditory Sensing Proteins for Next-generation Sonogenetics |
| 60 | Yi-Chien Chuang 莊貽茜 | Centrosomal Glutamylation Recruits the Microtubule Nucleation Factors to Ensure Its Functions |
| 62 | Shiau-Chi Chen 陳筱奇 | Precise Control of Intracellular Trafficking and Receptor-Mediated Endocytosis in Living Cells and Behaving Animals |
| 64 | Bo-Hua Yu 余柏樺 | Using Drosophila nuclear size regulation as a model to investigate the interplay between DNA damage repair response and autophagy |

| No. | Name | Title |
|---|---|---|
| 66 | Odvogmed Bayansan<br>狄歐梅 | UNC-10/SYD-2 complex links kinesin-3 to rab-3 tagged vesicles in the absence of motor's PH domain |
| 68 | Shin-Huey Lin 林欣慧 | Foxm1 regulates macrophage polarization and cytokine secretion to drive renal fibrosis after acute kidney injury |