2025

Taiwan Yeast Meeting

- 主辦單位

國立陽明交通大學
NATIONAL YANG MING CHIAO TUNG UNIVERSITY

- 協辦單位

國家衛生研究院
National Health Research Institutes

2025 Taiwan Yeast Meeting

◆贊助單位

- 國家科學及技術委員會補助 生命科學研究推動中心

◆Sponsored by:

- Life Sciences Research Promotion Center (LSRPC)

Organizing Committee

國立陽明交通大學 (National Yang Ming Chiao Tung University)

陳美瑜	生化暨分子生物研究所
Mei-Yu Chen	Institute of Biochemistry and Molecular Biology
陳瀅州	生命科學系暨基因體科學研究所
Ying-Chou Chen	Department of life Sciences and Institute of Genome Sciences
許博琛	微生物及免疫學研究所
Po-Chen Hsu	Institute of Microbiology and Immunology

2025 Taiwan Yeast Meeting

會議名稱：2025 Taiwan Yeast Meeting
會議日期：2025 年 8 月 15 日(星期五)
主辦單位：
國立陽明交通大學
財團法人國家衛生研究院 感染症與疫苗研究所
會議地點：國立陽明交通大學 陽明校區 活動中心 第三會議室

Conference: 2025 Taiwan Yeast Meeting
Date: August 15, 2025 (Friday)
Organizers:
National Yang Ming Chiao Tung University (NYCU)
National Health Research Institutes, Taiwan
Venue: Conference Room 3, Activity Center, Yang Ming Campus, NYCU

2025 Taiwan Yeast Meeting

會議地點：國立陽明交通大學 陽明校區 活動中心 第三會議室

WiFi：NYCU-Seminar

帳號：SE376858

密碼：jyz8q3

Venue: Conference Room 3, Activity Center, Yang Ming Campus, NYCU

WiFi：NYCU-Seminar

Account：SE376858

Password：jyz8q3

2025 Taiwan Yeast Meeting

Time	Topics	Speakers	Moderators
08:30 – 09:00	Registration 報到		
09:00 – 09:05	Opening Remark 開幕致詞	Dr. Tzu-Hao Cheng, Senior Vice President, NYCU 鄭子豪 副校長 陽明交通大學	
Session I			
09:05 – 09:50	**Keynote Speech 1** H3K4 Methylation Beyond Transcription: A Chromatin-Based System for Cellular Coordination	Dr. Cheng-Fu Kao, Deputy Director, ICOB, AS 高承福 副所長 細胞與個體生物學研究所/中央研究院	Dr. Po-Chen Hsu, NYCU 許博琛 陽明交通大學
09:50 – 10:20	The Role of the Succinate Dehydrogenase Assembly Factor Sdh5/SDHAF2 in Regulating Mitochondria in Yeast Cells	Dr. Chuang-Rung Chang, NTHU 張壯榮 清華大學	
10:20 – 10:40	Break/Posters 休息/壁報展示		
Session II			
10:40 – 11:10	Tom40 Functions as a Channel for Protein Retrotranslocation in the Mitochondria-Associated Degradation (MAD) Pathway	Dr. Pin-Chao Liao, NTHU 廖品超 清華大學	Dr. Ying-Chou Chen, NYCU 陳瀅州 陽明交通大學
11:10 – 11:40	Nuclear Envelope Assembly Proteins Are Essential for Sexual Reproduction in *Schizosaccharomyces japonicus*	Dr. I-Ju Lee, NTHU 李以如 清華大學	
11:40 – 12:10	Targeted Attenuation of an ER-Associated Degradation Pathway Enhances Mitochondrial Genome Quality Control in Yeast	Dr. Po-Chen Hsu, NYCU 許博琛 陽明交通大學	
12:10 – 13:30	Lunch / PI Meeting 午餐/研究主持人會議		
12:40 – 13:30	Poster presentation 壁報發表時段		
Session III			
13:30 – 14:15	**Keynote Speech 2** More than a Zip Code: Noncanonical Functions of Nuclear Transport Receptors Beyond Cargo Delivery	Dr. Kuo-Chiang Hsia, Deputy Director, IMB, AS 夏國強 副所長 分子生物研究所/中央研究院	Dr. Mei-Yu Chen, NYCU 陳美瑜 陽明交通大學
14:15 – 14:45	Exploring the Multifaceted Roles of Rap1 in *Candida albicans*	Dr. Chung-Yu Lan, NTHU 藍忠昱 清華大學	
14:45 – 15:15	Fanning the Flames: Turning Inflammation into a Colonization Weapon by a Commensal Fungus	Dr. Yu-Huan Tsai, CGU 蔡雨寰 長庚大學	
15:15 - 15:35	Break/Posters 休息/壁報展示		
Session IV			
15:35 – 15:51	Orthologous Transcription Factor Replacement Reveals that Stable TFIIIC Complexes Are Required for Proper Mitotic Chromosome Segregation	Akshi Gupta, IMB, AS 艾奇 分子生物研究所/中央研究院	Dr. Ying-Chou Chen, NYCU 陳瀅州 陽明交通大學
15:51 – 16:07	The Role of Ndt80 in Copper Tolerance of *Candida albicans*	Hsuan-Yu Chen, NTHU 陳宣妤 清華大學	
16:07 – 16:23	Predominant Azole-Resistant *Candida tropicalis* Clade 4 Genotype: Environmental Persistence and Advances in Molecular Diagnostics	Kuo-Yun Tseng, NHRI 曾國鋆 國家衛生研究院	
16:23 – 16:39	TRPML2 as a Molecular Sentinel Directing Vesicle Trafficking During Fungal Invasion	Zi-Qi Gu, NYCU 顧子奇 陽明交通大學	
16:39 – 16:55	Dectin-1 Triggered Mucus Acts as a Selective Barrier against Filamentous *Candida albicans*	Wei-Lin Chen, NYCU 陳瑋綝 陽明交通大學	
16:55 – 17:00	**Closing 閉幕**		

Curriculum Vitae

	NAME	POSITION TITLE		
	**Cheng-Fu Kao** 高承福	**Research Fellow** 研究員		
	Institution and Location	Degree	Year(S)	Field of Study
	University of Edinburgh, Scotland UK	Ph.D.	2001	Developmental and Molecular Biology
	FU-JEN Catholic University, Taiwan	M.S.	1993	Toxicology
	FU-JEN Catholic University, Taiwan.	B.A.	1991	Nutrition

Research Interests

Our lab investigates how chromatin dynamics influence the way cells manage and integrate stress responses and developmental signals. We are particularly interested in how changes in chromatin structure help coordinate transcription, replication, and repair to maintain genome stability and ensure proper cell function. Using molecular genetics, genomics, and in vivo models, we study how chromatin transitions support both short-term cellular adaptation and long-term regulation of identity and function. More recently, we've extended our work to explore how chromatin contributes to tissue-level processes such as maturation and regeneration, with the goal of understanding how nuclear organization shapes broader biological outcomes.

RESEARCH AND PROFESSIONAL EXPERIENCE:

A. Positions

9/01, 2022- present	**Deputy Director, Institute of Cellular and Organismic Biology, Academia Sinica**
June 1, 2020- present	**Research Fellow**, Institute of Cellular and Organismic Biology, Academia Sinica
August 1, 2017-July 31, 2018	**Visiting Scholar,** Basic Research Division, Fred Hutchinson Cancer Research Center, Seattle, USA
March 10, 2015-May 31, 2020	**Associate Research Fellow**, Institute of Cellular and Organismic Biology, Academia Sinica
November 8, 2006-March 9, 2015	**Assistant Research Fellow**, Institute of Cellular and Organismic Biology, Academia Sinica
July 1, 2005 - June 30, 2008	**Special Fellow**, Career Development Program, The Leukemia & Lymphoma Society.
March 1, 2005-Octber 31, 2006	**Research assistant professor**, University of New Mexico Health Sciences Center,
April, 2002-Febuary, 2005	**Postdoctoral Fellow**, University of New Mexico Health Sciences Center.
August, 1996- July, 1997	**Teaching Assistant**, in Biochemistry, FU-JEN Catholic University, Taiwan.

B. Selected Publications

1. Lin CY, Chang YM, Tseng HY, Shih YL, Yeh HH, Liao YR, Hsu CL, Chen CC, Yan YT*, **Kao CF***.

Epigenetic regulator RNF20 underlies temporal hierarchy of gene expression to regulate postnatal cardiomyocyte polarization. Cell Rep. 2023 Nov 28;42(11):113416. doi: 10.1016/j.celrep.2023.113416.

2. Chang KL, Chen JH, Lin TC, Leu JY, **Kao CF**, Wong JY, Tsai HK. Short human eccDNAs are predictable from sequences. Brief Bioinform. 2023 Apr 21;bbad147. doi: 10.1093/bib/bbad147.
3. Swygert SG, Lin D, Portillo-Ledesma S, Lin PY, Hunt DR, **Kao CF**, Schlick T, Noble WS, Tsukiyama T. Local chromatin fiber folding represses transcription and loop extrusion in quiescent cells. Elife. 2021 Nov 4;10:e72062. doi: 10.7554/eLife.72062.
4. Huang JH, Liao YR, Lin TC, Tsai CH, Lai WY, Chou YK, Leu JY, Tsai HK*, **Kao CF***. iTARGEX analysis of yeast deletome reveals novel regulators of transcriptional buffering in S phase and protein turnover. Nucleic Acids Res. 2021 Jul 21;49(13):7318-7329. doi: 10.1093/nar/gkab555.
5. Hsu CL, Chong SY, Lin CY, **Kao CF***. Histone dynamics during DNA replication stress. J Biomed Sci. 2021 Jun 19;28(1):48. doi: 10.1186/s12929-021-00743-5.
6. Hsu CL, Lo YC, **Kao CF*.** H3K4 Methylation in Aging and Metabolism. Epigenomes. 2021 Jun 18;5(2):14. doi: 10.3390/epigenomes5020014.
7. Chong SY, Cutler S, Lin JJ, Tsai CH, Tsai HK, Biggins S, Tsukiyama T, Lo YC and Kao CF*. H3K4 methylation at active genes mitigates transcription-replication conflicts during replication stress. Nat Commun. 2020 Feb 10;11(1):809. doi: 10.1038/s41467-020-14595-4.
8. Wu MY, Lin CY, Tseng HY, Hsu FM, Chen PY and **Kao CF***. H2B ubiquitylation and the Asf1 histone chaperone mediate the formation and maintenance of heterochromatin architecture. Nucleic Acids Res. Accepted on April 24, 2017. (*correspondent author)
9. Hung SH, Wong RP, Ulrich HD* and **Kao CF***. Bre1-mediated mono-ubiquitylation of H2B contributes to the bypass of DNA damage during and after DNA replication. Proc Natl Acad Sci U S A. 2017 Mar 14;114(11):E2205-E2214. (*correspondent authors)
10. Hsu HE, Liu TN, Yeh CS, Chang TH, Lo YC, Kao CF* Feedback Control of Snf1 Protein and Its Phosphorylation Is Necessary for Adaptation to Environmental Stress. J Biol Chem. 2015 Jul 3;290(27):16786-96. doi: 10.1074/jbc.M115.639443. (*co-correspondent authors)
11. Wright DE and **Kao CF***. (Ubi)quitin' the h2bit: recent insights into the roles of H2B ubiquitylation in DNA replication and transcription. Epigenetics. 2015 Feb;10(2):122-6. doi: 10.1080/15592294.2014.1003750. (*Correspondent author(SCI IF: 5.108; Rank 26 out of 165 journals in GENETICS & HEREDIT, 15.75%).
12. Lin CY, Wu MY, Gay S, Marjavaara L, Lai MS, Hsiao WC, Hung SH, Tseng HY, Wright DE, Wang CY, Hsu GSW, Devys D, Chabes A and **Kao CF***. (2014) H2B mono-ubiquitylation facilitates fork stalling and recovery during replication stress by coordinating Rad53 activation and chromatin assembly. PLoS Genet 10: e1004667. (*Correspondent author) **(**SCI IF: 8.167; Rank 14 out of 164 journals in GENETICS & HEREDIT, 8.53%)

Session I : Talk 1_Keynote Speech 1

H3K4 Methylation Beyond Transcription: A Chromatin-Based System for Cellular Coordination

Cheng-Fu Kao (高承福)

Institute of Cellular and Organismic Biology, Academia Sinica, Taipei, Taiwan

Histone modifications have long been studied for their roles in regulating gene expression, but their full range of functions is only beginning to come into view. In this talk, I will explore how histone H3 lysine 4 methylation (H3K4me) serves as a broader regulatory signal that helps coordinate complex cellular activities beyond transcription alone. Our studies suggest that H3K4me enables the chromatin environment to act as a flexible interface between nuclear and cytoplasmic systems. One line of work shows that chromatin marked by H3K4me can influence how the cell navigates competing demands during DNA replication and transcription—not simply by resolving direct collisions, but by tuning chromatin states to support long-term genome integrity under stress. In parallel, we discovered that H3K4me also connects to metabolic control by regulating redox conditions within the endoplasmic reticulum, pointing to unexpected cross-talk between chromatin and organelle function. Together, these findings suggest a conceptual shift: rather than viewing histone marks as static indicators of transcriptional activity, we propose that they function as dynamic information platforms. This broader view positions chromatin—and H3K4 methylation in particular—as a system for coordinating genome stability, stress responses, and cellular homeostasis.

Curriculum Vitae

張壯榮 CHANG, Chuang-Rung

新竹市 300044 東區光復路二段 101 號
(03)5742776, crchang@life.nthu.edu.tw

工作經驗

教授
國立清華大學 生物科技研究所 *2023/08 –*
基礎教學副主任
國立清華大學 學士後醫學系 *2023/08 –*

學歷

博士 Ph. D.
Cellular and Molecular Pharmacology
Rutgers University/University of Medicine and Dentistry of New Jersey, New Jersey, U.S.A. *2004/06*
理學士 B. S.
台北醫學大學 醫事技術學系 *1994/06*

獲獎紀錄

- **國立清華大學 2024 年第十六屆傑出導師獎**
- **國立清華大學 2020 年第十二屆傑出導師獎**
- **國立清華大學生命科學院 102 學年度教學績優獎**
- **國立清華大學生命科院學 101 學年度新進教師研究獎**

學會會員

- **臺灣粒線體醫學暨研究學會 (Taiwan Society for Mitochondrial Research and Medicine)**
  第四、五、六屆學會理事，第七屆學會常務理事 (2015 -)
- **亞洲粒線體醫學暨研究學會 (Asian Society for Mitochondrial Research and Medicine)**
  學會秘書長 (2016 - 2018)
  台灣理事代表 (2019 -)
- **台灣自由基學會 (Society for Free Radical Research-Taiwan (SFRR-Taiwan)**
  學會理事 (2015-2018)

專業證照

醫事檢驗師 考試院醫事檢驗師檢覈及格

相關著作目錄

- Chen CL, Huang WL, Rapoport A, Daugelavičius R, **Chang CR***. (2025) The molecular mechanisms and physiological roles of mitochondria dynamics in *Saccharomyces cerevisiae*. *Microb Cell*. 2025 Accepted
- Chakkalaparambil Dileep N, Chiu CW, Wu CT, **Chang CR***, Lo CY*. (2025) Phosphorylation of the fission protein Drp1 contributes to the impact of the

curcuminoid 1,7-bis(4-hydroxyphenyl)-1,4,6-heptatrien-3-one on mitochondrial and cellular processes. *BBA Advances*. 2025, 8, 100164.

- Lee CH, Wu CJ, Yen FY, Chiang JY, Shen TJ, Leu SJ, **Chang CR**, Lo HJ, Tsai BY, Mao YC, Andriani V, Thenaka PC, Wang WC, Chao YP, Yang YY. (2025, Apr). Identification of chicken-derived antibodies targeting the Candida albicans Als3 protein. *Appl Microbiol Biotechnol*. 2025 Apr 8;109(1):85.
- Huang WL, Chen CL, Lin ZJ, Hsieh CC, Hua MD, Cheng CC, Cheng TH, Lai LJ*, **Chang CR***. (2024, Nov). Soft X-ray Tomography Analysis of Mitochondria Dynamics in Saccharomyces cerevisiae. *Bio Direct*. 2024 Nov 29;19(1):126.
- Chen CL, Ishihara T, Pal S, Huang WL, Ogasawara E, **Chang CR***, Ishihara N*. (2024) SDHAF2 facilitates mitochondrial respiration through stabilizing succinate dehydrogenase and cytochrome c oxidase assemblies. *Mitochondrion*. 2024 Nov;79:101952. Epub 2024 Sep 3.
- Wu CC, Tam EH, Shih YY, Lin YR, Hsueh PC, Shen HY, Woung CH, Wang LT, Tsai JC, Lin SJ, **Chang CR**, Ke PY, Kuo RL. (2024) Exploration of influenza A virus PA protein-associated cellular proteins discloses its impact on mitochondrial function. *Virus Res*. 345, 2024, 199387.
- Wu CT, Chu CI, Wang FY, Yang HY, Tseng WS, **Chang CR***, Chang CC. (2022). A change of PD-1/PD-L1 expression on peripheral T cell subsets correlates with the different stages of Alzheimer's Disease. *Cell & Bioscience*., 2022 Sep 30;12(1):162.
- Miao CC, Hwang W, Chu LY, Yang LH, Ha CT, Chen PY, Kuo MH, Lin SC, Yang YY, Chuang SE, Yu CC, Pan ST, Kao MC, **Chang CR**, Chou YT. (2022) LC3A-mediated autophagy regulates lung cancer cell plasticity. *Autophagy.*, 2022 Apr;18(4):921-934.
- Wu TC, Liao CY, Lu WC, **Chang CR**, Tsai FY, Jiang SS, Chen TH, Lin KM, Chen LT, Chang WW. (2022) Identification of distinct slow mode of reversible adaptation of pancreatic ductal adenocarcinoma to the prolonged acidic pH microenvironment. *J Exp Clin Cancer Res*., 2022 Apr 11;41(1):137.
- Chen CL, Chen YC, Huang WL, Lin S, Daugelavičius R, Rapoport A, **Chang CR***. (2021) A crucial role of mitochondrial dynamics in dehydration resistance in *Saccharomyces cerevisiae*. *Int. J. Mol. Sci*., 2021 Apr 27;*22*(9):4607.
- Chen YC, Cheng TH, Lin WL, Chen CL, Yang WY, Blackstone C, **Chang CR***. (2019) Srv2 is a pro-fission factor that modulates yeast mitochondrial morphology and respiration by regulating actin assembly. *iScience*., 2019 Jan 25;11:305-317.

***h*-index** for Chuang-Rung Chang is **21.**

Session I : Talk 2

The Role of the Succinate Dehydrogenase Assembly Factor Sdh5/SDHAF2 in Regulating Mitochondria in Yeast Cells

Wei-Ling Huang (黃薇玲)[1], Chuang-Rung Chang (張壯榮)[1, 2]

[1]Iinstitute of Biotechnology, National Tsing Hua University; [2]School of Medicine, National Tsing Hua university

Succinate dehydrogenase (SDH), or complex II, plays a vital role in mitochondrial respiration. It catalyzes the conversion of succinate to fumarate during the tricarboxylic acid (TCA) cycle and facilitates the transfer of electrons to ubiquinone within the electron transport chain (ETC). Sdh5/SDHAF2 is an assembly factor for the SDH complex. Our previous research in mammalian cells has revealed that SDHAF2 not only assists in the assembly of succinate dehydrogenase (complex II) but also affects mitochondrial respiration by influencing the assembly of cytochrome c oxidase (COX, complex IV). We examined yeast cells with the deletion of *SDH5*. We found that mitochondrial respiration and network morphology were altered in *Δsdh5* cells. Our results indicated that Sdh5/SDHAF2 impacted mitochondrial respiration and network morphology by remodeling the electron transport chain complexes. These insights enhance our understanding of the regulatory mechanisms operating within mitochondria.

Curriculum Vitae

Liao, Pin-Chao (廖品超)

Institute of Molecular Medicine, Department of Life Science

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PROFESSIONAL EXPERIENCE

Assistant Professor (2022 – now)

Institute of Molecular Medicine, Department of Life Science, National Tsing Hua University, Hsinchu, Taiwan

Associate Research Scientist (2021 – 2022)

Department of Pathology and Cell Biology, Columbia University, NY, USA

Postdoc Researcher (2016 – 2021)

Department of Pathology and Cell Biology, Columbia University, NY, USA

Research Assistant (2009 – 2011)

Institute of Molecular Medicine, National Tsing Hua University, Hsinchu, Taiwan

EDUCATION

- **Ph.D. in Biological Sciences (2011 – 2016)**

Interdisciplinary Life Science Program, Department of Biological Sciences, Purdue University, IN, USA

- **M.S. in Life Science (2006 – 2008)**

Institute of Biotechnology, National Tsing Hua University (NTHU), Hsinchu, Taiwan

- **B.S. in Life Science (2002 – 2006)**

Department of Life Science, National Tsing Hua University (NTHU), Hsinchu, Taiwan

AWARDS and HONORS

- 2022 Yushan Young Fellow 玉山青年學者
- 2015 Purdue Research Foundation (PRF) Research Grants, Purdue
- 2015 Fall Purdue University Interdisciplinary Life Science (PULSe) Travel Awards, Purdue
- 2013 Fall Purdue University Interdisciplinary Life Science (PULSe) Travel Awards, Purdue
- 2008 Best Poster Competition of College of Life Science, National Tsing Hua University, Taiwan
- 2008 Honor member of the Phi Tau Phi Scholastic Honor Society of the Republic of China, Taiwan
- 2005 Research Grant Proposal for College-Level Students, National Science Council, Taiwan

SELECTED PUBLICATIONS

- **Pin-Chao Liao** *, Tzu-Ying Lin, Catherine A Tsang, Chen-Jing Huang, Katherine Filpo, Istvan Boldogh, Liza A Pon* (2025, July) Tom40 functions as a channel for protein retrotranslocation in the mitochondria-associated degradation (MAD) pathway. *Communications Biology,* 8:1122 (*Co-corresponding author)
- **Pin-Chao Liao** and Liza A Pon (2024, Sep). Analysis of the mitochondria-associated degradation pathway (MAD) in yeast cells. *Methods in Enzymology*, 707, 585-610.
- Tzu-Ying Lin, Shih-Hung Chien, Liza A Pon and **Pin-Chao Liao** (2024, Aug). Isolation of yeast mitochondria by affinity purification using magnetic beads. *Methods in Enzymology*, 706, 19-36.
- Emily J. Yang*, **Pin-Chao Liao*** and Liza A. Pon (2024, Feb) Mitochondrial protein and organelle quality control—Lessons from budding yeast. *IUBMB Life* 76(2):72 (*Co-first author)
- **Pin-Chao Liao** & Liza A. Pon (2022, May) Lipid droplets in stress protection: distinct mechanisms of lipid droplet microautophagy. *Autophagy Reports* 1(1):197, DOI: 10.1080/27694127.2022.2067643
- **Pin-Chao Liao*,** Emily J. Yang*, Taylor Borgman*, Istvan R. Boldogh*, Cierra N. Sing, Theresa C. Swayne and Liza A. Pon (2022, Feb). Touch and Go: Membrane Contact Sites Between Lipid Droplets and Other Organelles. *Frontiers in Cell and Developmental Biology* 10:852021. doi: 10.3389/fcell.2022.852021 (*Co-first author)
- **Pin-Chao Liao***, Enrique J. Garcia*, Gary Tan, Catherine A. Tsang, and Liza A. Pon (2021, Dec). Roles for L_o microdomains and ESCRT in ER stress-induced lipid droplet microautophagy in budding yeast. *Molecular Biology of the Cell* 32(22):br12 (*Co-first author)
- Enrique J. Garcia*, **Pin-Chao Liao***, Gary Tan, Jason D. Vevea, Cierra N. Sing, Catherine A. Tsang, J. Michael McCaffery, Istvan R. Boldogh and Liza A. Pon (2021, Sep). Membrane dynamics and protein targets of lipid droplet microautophagy during ER stress-induced proteostasis in the budding yeast, *Saccharomyces cerevisiae. Autophagy* 17(9):2363-2383 (*Co-first author)
- **Pin-Chao Liao**, Emily J. Yang and Liza A. Pon (2020, Nov). Live-cell imaging of mitochondrial redox state in yeast cells. *STAR Protocols* 1(3):100160
- **Pin-Chao Liao**, Dana M Alessi Wolken, Edith Serrano, Pallavi Srivastava and Liza A Pon (2020, Jul). Mitochondria-Associated Degradation Pathway (MAD) Function Beyond the Outer Membrane. *Cell Reports* 32(2):107902
- **Pin-Chao Liao**, Sandra Franco-Iborra, Yi Yang, and Liza A. Pon (2020, Mar). Live cell imaging of mitochondrial redox state in mammalian cells and yeast. *Methods in Cell Biology* 155:295-319
- **Pin-Chao Liao,** Ryo Higuchi-Sanabria, Theresa C. Swayne, Cierra N Sing, and Liza A. Pon (2020, Mar). Live-cell imaging of mitochondrial motility and interactions in Drosophila neurons and yeast. *Methods in Cell Biology* 155:519-544
- **Pin-Chao Liao**, Christian Bergamini, Romana Fato, Liza A. Pon and Francesco Pallotti (2020, Mar). Isolation of mitochondria from cells and tissues. *Methods in Cell Biology* 155:3-31

Session II : Talk 1

Tom40 Functions as a Channel for Protein Retrotranslocation in the Mitochondria-Associated Degradation (MAD) Pathway

Pin-Chao Liao (廖品超)[1, 2, 3*], Tzu-Ying Lin (林茲瑩)[2], Catherine A. Tsang[1], Chen-Jing Huang (黃晨淨)[2], Katherine Filipo[1], Istvan Boldogh[1] and Liza A. Pon[1*]

[1]Department of Pathology and Cell Biology, Columbia University, New York, NY, United States; [2]Institute of Molecular Medicine, National Tsing Hua University, Hsinchu, Taiwan; [3]Department of Life Science, National Tsing Hua University, Hsinchu, Taiwan

The mitochondria-associated degradation pathway (MAD) mediates removal and elimination of damaged, unfolded mitochondrial proteins by the ubiquitin-proteasome system (UPS). Previous studies revealed that MAD is critical for mitochondrial protein quality control and that MAD function extends beyond mitochondrial outer membrane (MOM) to proteins within the organelle. Here, we reconstitute retrotranslocation of MAD substrates from the mitochondrial matrix across mitochondrial inner and outer membranes in cell-free systems. This retrotranslocation is ATP-dependent but membrane potential-independent. We also identify a role for the TOM complex, the protein import channel in the MOM, in this process. Inhibition of protein translocation across the Tom40p channel reduces the retrotranslocation of MAD substrates. Our studies support the model that the TOM complex is a bidirectional protein channel in the MOM: it mediates retrotranslocation of damaged mitochondrial proteins across the MOM in the MAD pathway for mitochondrial protein quality control in addition to its function in import of proteins into the organelle.

Curriculum Vitae

I-Ju Lee 李以如

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Office Phone: (03) 5715131 ext. 42748; *Email:* ijlee@life.nthu.edu.tw
ORCID: 0000-0003-3205-7741;
Lab Website: https://ijuleelab.wixsite.com/ijl-lab

Professional Experience

2022-present **Assistant Professor** at Institute of Molecular Medicine, National Tsing Hua University, Taiwan
2014-2022 **Postdoctoral Research Fellow** at Dr. David Pellman's Laboratory, Dana-Farber Cancer Institute/Harvard Medical School, Boston, MA, USA
2014 **Postdoctoral Researcher** at Dr. Jian-Qiu Wu's Laboratory, The Ohio State University, Columbus, OH, USA

Education

2008-2013 **Ph.D.** in Molecular, Cellular, and Developmental Biology, The Ohio State University, Columbus, OH, USA
2005-2007 **M.S.** in Molecular and Cellular Biology, National Taiwan University, Taiwan
2001-2005 **B.S.** in Chemistry, National Taiwan University, Taiwan

Awards and Honors

2015-2018 Postdoctoral Fellowship, The Leukemia and Lymphoma Society
2012-2013 Pelotonia Graduate Fellowship for Cancer Research, The Ohio State University
2013 Outstanding Poster Award, Molecular Life Sciences Interdisciplinary Graduate Programs (MLS-IGP), The Ohio State University
2012 Graduate Student Travel Award, The America Society for Cell Biology

Publications

Lee I-J*, Stokasimov E, Dempsey N, Varberg J, Jacob E, Jaspersen SL, and Pellman D. 2020. Factors promoting nuclear envelope assembly independent of the canonical ESCRT pathway. *J. Cell Biol.* 219:e201908232. PMID: 32243490 **Co-corresponding author*

Liu Y, **Lee I-J**, Sun M, Lower CA, Runge KW, Ma J, Wu J-Q. 2016. Roles of the novel coiled-coil protein Rng10 in septum formation during fission yeast cytokinesis. *Mol. Biol. Cell.* 27:2528-2541. PMID: 27385337

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Sun L, Guan R, **Lee I-J**, Liu Y, Chen M, Wang J, Wu J-Q, and Chen Z. 2015. Mechanistic insights into the anchorage of the contractile ring by Anillin and Mid1. ***Dev. Cell.*** 33:1-14. PMID: 25959226

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Lee I-J and Wu J-Q. 2012. Characterization of Mid1 domains for targeting and scaffolding in fission yeast cytokinesis. ***J. Cell Sci.*** 125:2973-2985. PMID: 22427686

Ye Y, **Lee I-J**, Runge KW, and Wu J-Q. 2012. Roles of putative Rho-GEF Gef2 in division-site positioning and contractile-ring function in fission yeast cytokinesis. ***Mol. Biol. Cell.*** 23:1181-1195. PMID: 22298427

Laporte D, Coffman VC, **Lee I-J**, and Wu J-Q. 2011. Assembly and architecture of precursor nodes during fission yeast cytokinesis. ***J. Cell Biol.*** 192:1005-1021. PMID: 21422229

Yeh C-H, Yang H-J, **Lee I-J**, and Wu Y-C. 2010. *C. elegans* TLK-1 controls cytokinesis by localizing AIR-2/Aurora B to midzone microtubules. ***Biochem. Biophys. Res. Commun.*** 400:187-193. PMID: 20705056

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Book Chapter

Coffman VC, **Lee I-J**, and Wu J-Q. 2014. Counting molecules within cells. In: Marshall WF, editor. Colloquium series on quantitative cell biology #1. Morgan & Claypool Publishers.

Invited Talks

2025 Taiwan Yeast Meeting, Taipei, Taiwan
2024 OU (Osaka University)-NTHU Symposium, Hsinchu, Taiwan
2024 Taiwan Yeast Meeting, Taichung, Taiwan
2024 Institute of Molecular and Genomic Medicine, NHRI, Taiwan
2024 The 8th Transdisciplinary Workshops of Chemistry, Life Science, and Physics
2022 NHRI-NTHU Joint Research Conference, Miaoli, Taiwan
Taiwan Yeast Meeting, Hsinchu, Taiwan
2020 Pombe Talks Online Seminar
Institute of Cellular and Organismic Biology, Academia Sinica, Taiwan
2019 The Northeast Nuclear Envelope Meeting, New Haven, CT, USA
Boston Taiwanese Biotechnology Association, Boston, MA, USA
2018 FASEB Meeting Yeast Chromosome Biology and Cell Cycle, Steamboat Springs, CO, USA
2014 Institute of Molecular and Cellular Biology, National Taiwan University, Taiwan

Service and Leadership

2025 Poster Judge, The 12th International Fission Yeast Meeting
2023-present Manuscript Reviewer, Journal of Cell Science
2022-2024 Early-Career Reviewer, Genetics and G3, Genetics Society of America
2021 Abstract Reviewer, ASCB/EMBO meeting
2020 Panelist, Postdoc Workshop, Boston Taiwanese Biotechnology Association
2018-2020 Board Member, Boston Taiwanese Biotechnology Association
2018 Mentor, CURE program for students from underrepresented populations, Dana-Farber Cancer Institute
2017-2018 co-President, Boston Taiwanese Biotechnology Association
2010-2022 Reviewed manuscripts for EMBO Journal, Genes & Development, Journal of Cell Biology, Molecular Biology of the Cell, and Molecular Microbiology with advisors.

Session II : Talk 2

Nuclear Envelope Assembly Proteins are Essential for Sexual Reproduction in *Schizosaccharomyces japonicus*

Jia-Syuan Huang (黃嘉宣)[1], Tang-Yu Zhu (朱堂瑀)[1], Chi-Hsuan Chen (陳齊軒)[1], and I-Ju Lee (李以如)[1]

[1]Institute of Molecular Medicine, National Tsing Hua University, Taiwan

The nuclear envelope (NE) is a crucial structure in all eukaryotic cells that separates the genetic material from the cytoplasm. Defects in NE integrity may not cause immediate cell death but can lead to severe long-term consequences for both cellular and organismal health, many of which remains to be studied. Proper NE assembly after cell division and efficient repair following damage relies on an ESCRT-dependent pathway that involves the inner nuclear membrane protein Lem2, the ESCRT adaptor Cmp7, the ESCRT-III component Vps32, and the AAA ATPase Vps4. Surprisingly, we found that the loss of any of these four NE assembly proteins in *Schizosaccharomyces japonicus*, a unique yeast that partially disassembles and reassembles its NE during each cell division, abolished sexual reproduction and gamete formation. These findings suggest that compromised NE integrity can interfere with gametogenesis. Consistently, suppressor mutations that restore NE assembly during vegetative growth partially rescued the gametogenesis defects. Sexual reproduction in *S. japonicus* requires cell conjugation, nuclear fusion, two rounds of meiosis, and a final round of mitosis coupled with sporulation. In the NE assembly mutants, many cells arrested with two nuclei, indicating a failure in either nuclear fusion or meiosis II. Live-cell imaging revealed that nuclear fusion was severely impaired in the absence of Cmp7. Moreover, based on the morphology of the nuclei and the number of spindle-pole-body on the NE, nuclear fusion defects in cells lacking Cmp7 appear to be distinct from those observed in cells lacking Tht1, the only fission yeast protein previously implicated in nuclear fusion. Taken together, our results uncovered a previously unknown role for NE assembly proteins in sexual reproduction, highlighting a fundamental aspect of NE assembly and its long-term impacts. These insights may inform strategies for controlling parasitic yeast that relies on sexual reproduction for transmission.

Curriculum Vitae

Po-Chen Hsu (許博琛)

Personal information

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Education / Professional appointments

2025-now	**Assistant Professor.** Institute of Microbiology and Immunology, National Yang Ming Chiao Tung University, Taiwan
2020-2025	**Senior Scientist**. Jun-Yi Leu (呂俊毅老師)'s Lab.
2016–2020	**Post-doctoral Fellow**. Institute of Molecular Biology, Academia Sinica, Taiwan. Jun-Yi Leu's Lab.
2007–2013	**Ph.D.** Institute of Molecular and Cellular Biology, National Tsing-Hua University, Taiwan. Chung-Yu Lan (藍忠昱老師)'s Lab.
2006–2007	**M.S. program, then transferred into the Direct-Ph.D program**. Institute of Molecular and Cellular Biology, National Tsing-Hua University, Taiwan. Chung-Yu Lan's Lab.
2002–2006	**B.S.** Department of Life Science, National Tsing-Hua University, Taiwan.

Research interests

- Yeast Pathogenesis (酵母菌病源體的致病機制)
- Mitochondrial genome quality control & disease (粒線體基因體品管機制與疾病的關聯)
- Transcriptional rewiring (轉錄因子調控網路重組的分子機制)
- Experimental evolution (實驗室內的酵母菌人工演化)
- Molecular basis of complex traits (受多重因子影響性狀之分子機制)
- Pleiotropy (酵母菌基因多效性)
- Cell density-dependent phenotypes (細胞濃度依賴性之酵母菌表現型)

Selected Awards / Achievements

2022	**NSTC Academic Research Award for Postdoc Researchers (國科會 111 年度博士後研究人員學術研究獎)**
2020-2025	NSTC (Taiwan) Frontier Science Research Program (國科會尖端科學研究計畫) Postdoctoral Fellowship
2018-2020	Academia Sinica Thematic Research Program (中研院跨領域主題計畫) Postdoctoral Fellowship

2017-2018	Postdoctoral Fellowship supported by Institutional Funding of Institute of molecular Biology, Academia Sinica
2015-2017	Academia Sinica Regular Postdoctoral Scholar Program (中研院一般博士後研究學者) Fellowship
2014-2015	NSTC (Taiwan) Frontier Science Research Program (國科會尖端科學研究計畫) Postdoctoral Fellowship

Selected Publications

- **Hsu, P.-C.***, Lu, T.-C., Hung, P.-H., and Leu, J.-Y.**** (2024) Protein moonlighting by a target gene dominates phenotypic divergence of the Sef1 transcriptional regulatory network in yeasts. **Nucleic Acids Res.** 52(22):13914-13930 **(* co-corresponding author)**
- **Hsu, P.-C.***, Cheng, Y.-H., Liao, C.-W., Litan, R.R.R., Jhou, Y.-T., Opoc, F.J.G., Amine, A.A.A., and Leu, J.-Y. (2023) Rapid evolutionary repair by secondary perturbation of a primary disrupted transcriptional network. **EMBO Reports** 24(6):e56019 **(* corresponding author)**
- Chou, J.-Y.*, **Hsu, P.-C.**, and Leu, J.-Y.* (2022) Enforcement of postzygotic species boundaries in the fungal kingdom. **Microbiology and molecular biology reviews** 86(4), e0009822 **(Review article)**
- **Hsu, P.-C.***, Lu, T.-C., Hung, P.-H., Jhou, Y.-T., Amine, A.A.A., Liao, C.-W., Leu, J.-Y.* (2021) Plastic Rewiring of Sef1 Transcriptional Networks and the Potential of Nonfunctional Transcription Factor Binding in Facilitating Adaptive Evolution. **Molecular Biology and Evolution** 38(11): 4732-4747 **(* co-corresponding author)**
- Amine, A.A.A., Liao, C.-W., **Hsu, P.-C.**, Opoc, F.J.G., Leu, J.-Y. (2021) Experimental evolution improves mitochondrial genome quality control in *Saccharomyces cerevisiae* and extends its replicative lifespan. **Current Biology** 31(16):3663-3670e4 **(3/109 in Biology)**
- Chen, Y.-Y., Chao, C.-C., Liu, F.-C., **Hsu, P.-C.**, Chen, H.-F., Peng, S.-C., Chuang, Y.-J., Lan, C.-Y., Hsieh, W.-P., and Wong, D.S.-H.* (2013) Dynamic transcript profiling of *Candida albicans* infection in zebrafish: a pathogen-host interaction study. **PLoS One** 8(9):e72483
- **Hsu, P.-C.**, Chao, C.-C., Yang, C.-Y., Ye, Y.-L., Liu, F.-C., Chuang, Y.-J., and Lan, C.-Y.* (2013) Diverse Hap43-independent functions of *Candida albicans* CCAAT-binding complex. **Eukaryot Cell** 12(6):804-15
- Tsai, P.-W., Chen, Y.-T., **Hsu, P.-C.**, and Lan, C.-Y.* (2013) Study of *Candida albicans* and its interactions with the host: A mini-review. **BioMedicine** 3:51-64
- **Hsu, P.-C.**, Yang, C.-Y., and Lan, C.-Y.* (2011) *Candida albicans* Hap43 is a repressor induced under low-iron conditions and is essential for iron-responsive transcriptional regulation and virulence. **Eukaryot Cell** 10(2):207-225
- Chao, C.-C., **Hsu, P.-C.**, Jen, C.-F., Chen, I.-H., Wang, C.-H., Cham, H.-C., Tsai, P.-W., Tung, K.-C., Wang, C.-H., Lan, C.-Y.*, and Chuang, Y.-J.* (2010) Zebrafish as a model host for *Candida albicans* infection. **Infect Immun** 78(6): 2512-2521

Session II : Talk 3

Targeted Attenuation of an ER-Associated Degradation Pathway Enhances Mitochondrial Genome Quality Control in Yeast

Ruth Yofanka Febryani (葉茹思) [1], Jun-Yi Leu (呂俊毅)[1], and Po-Chen Hsu (許博琛)[2]

[1] Institute of Molecular Biology, Academia Sinica, Taipei, Taiwan; [2]Institute of Microbiology and Immunology, National Yang Ming Chiao Tung University, Taipei, Taiwan;

Mitochondria are dynamic, double-membraned organelles of endosymbiotic origin that retain autonomous genetic machinery to sustain multicopy genomes. Although most ancestral mitochondrial genes have been lost or relocated to the nuclear genome during evolution, mitochondria preserve a conserved core gene set essential for oxidative phosphorylation. In addition to ATP synthesis, they perform a wide array of fundamental functions, including amino acid metabolism, biosynthesis of iron–sulfur clusters, lipids, glutathione, nucleotides, heme, and biotin, as well as regulation of protein homeostasis, apoptotic signaling, redox equilibrium, aging, and calcium dynamics. Due to their central role in cellular physiology, mitochondrial dysfunction is implicated in numerous human pathologies, arising from mutations in both the nuclear and mitochondrial genomes (mtDNA). Mito–nuclear communication regulates mitochondrial integrity via antioxidant defenses, DNA repair, mitophagy, and mitochondrial biogenesis. *Saccharomyces cerevisiae* retains viability in the absence of mitochondrial respiration, thereby providing a robust model for dissecting mtDNA quality control (QC) mechanisms. In our previous study, yeast populations were subjected to ethidium bromide (EtBr), a mtDNA-damaging agent, and consequently, the experimentally evolved strains exhibited enhanced mtDNA QC. Genetic analysis revealed that the endoplasmic reticulum–associated degradation (ERAD) pathway negatively regulates mtDNA QC. While disruption of the luminal (ERAD-L) and cytosolic (ERAD-C) branches had a marginal impact, ablation of the integral membrane branch (ERAD-M) significantly improved mtDNA QC. These findings suggest that ERAD-M–specific substrates may serve as key modulators of mtDNA QC.

Curriculum Vitae

NAME: Kuo-Chiang Hsia　　中文姓名：夏國強

INSTITUTE/RESEARCH CENTER: Institute of Molecular Biology

Current POSITION: Research Fellow/Deputy Director

EDUCATION

INSTITUTION AND LOCATION	DEGREE	DURATION MM/YY-MM/YY	FIELD OF STUDY
FuJen Catholic University, Taipei, Taiwan	B.S.	09/94-07/98	Biology
National Yang-Ming University, Taipei, Taiwan	M.S.	09/98-07/00	Biochemistry
The Rockefeller University, New York, NY, USA	Ph.D.	01/05-04/09	Structural Biology and Biochemistry

EMPLOYMENT

INSTITUTION AND LOCATION	POSITION TITLE	DURATION MM/YY-MM/YY	FIELD OF STUDY
The Rockefeller University, Laboratory of Cell Biology, Howard Hughes Medical Institute	Postdoctoral Fellow	05/09-03/10	Structural Biology, Biochemistry and Cell Biology
The Rockefeller University, Laboratory of Chemistry and Cell Biology	Postdoctoral Fellow	04/10-08/15	Structural Biology, Biochemistry and Cell Biology
Institute of Molecular Biology, Academia Sinica, Taipei, Taiwan	Assistant Research Fellow	09/15-06/20	Structural Biology, Biochemistry and Cell Biology
Institute of Molecular Biology, Academia Sinica, Taipei, Taiwan	Associate Research Fellow	07/20-12/24	Structural Biology, Biochemistry and Cell Biology
Institute of Molecular Biology, Academia Sinica, Taipei, Taiwan	Research Fellow	01/25-present	Structural Biology, Biochemistry and Cell Biology

I. PERSONAL STATEMENT

The spatial organization of the cellular cytoplasm has fascinated cell biologists since the advent of microscopy. We are interested in elucidating how complex micron-sized microtubule arrays are organized and function to facilitate mitosis progression, ciliogenesis, and neuronal maturation. Moreover, we examine how nuclear transport receptors modulate the organization of microtubule-based structures (e.g., spindle and anemone) via canonical and non-canonical activities. Our approach is to *in vitro* reconstitute and image the self-organization of microtubule-based structures from the protein building blocks. We further apply an interdisciplinary approach to uncover cellular mechanisms, combining structural biology, biophysical, and cell biology methods. Through our work, I aim to discover the mechanistic links between the cytoskeletal organization and vital cell functions.

We tackle questions from an unconventional perspective, i.e., we start with in vitro assays

(e.g. biochemical and structural biology), and subsequently examine our in vitro findings in vivo (e.g. cell- based assay). Notably, beyond fundamental cell biology, my research extends to understanding the rolex of the cytoskeleton in cancer cell survival, drug resistance, and the progression of neurodevelopmental disorders such as autism. My academic background includes training in Dr. Gunter Blobel's laboratory during my Ph.D. studies and subsequent postdoctoral work in Dr. Tarun Kapoor's laboratory, both at Rockefeller University. Currently I serve as a Research Fellow and Deputy Director at the Institute of Molecular Biology, Academia Sinica.

II. SELECTED AWARDS

- Academia Sinica, Career Development Award, 2017-2021(106-L02)
- Academia Sinica, Investigator award, 2023-2027 (112-L05)
- Academia Sinica Early-Career Investigator Research Achievement Award, 2023
- NSTC Outstanding Research Award, 2023

III. SELECTED PUBLICATIONS (*corresponding author)

- Weng, T.H., Pien, Y.C., Chen, C.J., Chen, P.P., Tseng, Y.T., Chen, Y.C., Lee, Y.T., Chen, Y.A., Chen, Y.C., Lim, C., Hsu, T.H., Lin, S.J., Yen, H.Y., **Hsia, K.C.*,** Tsai, S.Y.* (2025) Human glycogenins maintain glucose homeostasis by regulating glycogen metabolism. *Nature Communications* 16(1):6556.
- Saju, A., Chen, P.P., Weng, T.H., Tsai, S.Y., Tanaka, A., Chang, C.C., Wang, C.H., Shimamoto, Y., **Hsia, K.C.*** (2024). HURP binding to the vinca domain of β-tubulin accounts for cancer drug resistance. *Nature Communications* 15(1):8844.
- Liao, C.C., Wang, Y.S., Pi, W.C., Wang, C.H., Wu, Y.M., Chen, W.Y.*, **Hsia, K.C.*** (2023). Structural convergence endows nuclear transport receptor Kap114p with a novel transcriptional repressor function toward TATA-box binding protein. *Nature Communications* 14(1):5518.
- Shankar, S., Hsu, Z.T., Ezquerra, A., Li, C.C., Huang, T.L., Coyaud, E., Viais, R., Grauffel, C., Raught, B. Lim, C., Lüders, J.*, Tsai, S.Y.*, **Hsia, K.C.*** (2022) □-tubulin complex-dependent pathway suppresses ciliogenesis by promoting cilia disassembly. *Cell Reports* 41(7):111642.
- Shih, P.Y., Shankar, S., Lee, S.P., Fang, Y.L., Chen, H., Wang, T.F., **Hsia, K.C.***, Hsueh, Y.P.* (2022). Zinc-induced phase transition modulates synaptic distribution of autism-linked CTTNBP2. *Nature Communications* 13(1):2664.
- Huang, T.L., Wang, H.J., Chang, Y.C., Wang, S.W., **Hsia, K.C.*** (2020) Promiscuous binding of microprotein Mozart1 to γ-TuRC mediates specific subcellular localization to control microtubule array formation. *Cell Reports* 31(13):107836.
- Wieczork, M., Huang, T.L., Urnavicius, L., **Hsia, K.C.***, Kapoor, T.M.* (2020) MZT proteins form multi-faceted structural modules within the γ-tubulin ring complex. *Cell Reports* 31(13):107791 (**Cover of the issue**).
- Liao, C.C., Shankar, S., Pi, W.C., Ahmed, G.R., Chen, W.Y., **Hsia, K.C.*** (2020) Karyopherin Kap114p-mediated trans-repression controls ribosomal gene expression under saline stress. *EMBO reports*, 21(7):e48324.
- Chang, C.C., Chen, C.J., Grauffel, C., Pien, Y.C., Lim, C., Tsai, S.Y.*, **Hsia, K.C.*** (2019) Ran pathway-independent regulation of mitotic Golgi disassembly by Importin-□. *Nature Communications* 10(1):4307.
- Chang, C.C., Huang, T.L., Shimamoto, Y., Tsai, S.Y., **Hsia, K.C.*** (2017) Regulation of mitotic spindle assembly factor NuMA by Importin-β. *J. Cell Biol.* 216(11):3453-3462. (Selected as one of twenty articles in JCB special collection (cell division))

Session III : Talk 1_Keynote Speech 2

More Than a Zip Code: Noncanonical Functions of Nuclear Transport Receptors Beyond Cargo Delivery

Kuo-Chiang Hsia (夏國強)

Institute of Molecular Biology, Academia Sinica, Taipei, 11529, Taiwan.

Soluble nuclear transport factors are conventionally known for facilitating nucleocytoplasmic transport by mediating cargo translocation across the nuclear pore complex (NPC). Among these cargos are transcription factors, such as the TATA-box binding protein (TBP), which localize to the nucleus to regulate gene expression. This process relies on nuclear transport receptors, collectively termed karyopherin-β (Kap-β) in yeast, and additional regulatory factors.

In our study, we focused on Kap114p, a Kap-β family member responsible for the nuclear import of yeast TBP (yTBP). Unexpectedly, we found that Kap114p also modulates yTBP-dependent transcription. Using single-particle cryo-electron microscopy, we determined the structure of Kap114p in complex with the core domain of yTBP ($yTBP^C$). Strikingly, Kap114p wraps around the N-terminal lobe of yTBPC, a configuration that closely resembles how classical transcriptional regulators interact with TBP. This structural similarity suggests a case of convergent evolution between transport receptors and transcriptional repressors. Functionally, Kap114p sequesters yTBP away from gene promoters, thereby modulating its availability for transcription initiation. Thus, our findings demonstrate that Kap114p ensures the proper temporal and spatial control of yTBP activity, particularly under environmental stress conditions that demand precise gene expression responses.

Collectively, our work reveals that nuclear transport receptors such as Kap114p are not merely transporters but can participate in gene regulatory networks. By functioning both as an import receptor and as a transcriptional modulator, Kap114p exemplifies the multifunctionality of transport factors in eukaryotic cells. These findings broaden our understanding of nuclear transport machinery and uncover its unexpected roles in regulating transcriptional output.

Curriculum Vitae

Chung-Yu Lan (藍忠昱)

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Phone: (O) 03-5742473; Email: cylan@life.nthu.edu.tw;
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Professional Experience

2017-present, Distinguished Professor, National Tsing Hua University (NTHU), Taiwan
2017-2018, Chairman, Department of Life Science, NTHU, Taiwan
2014-present, Professor, Institute of Molecular and Cellular Biology, Department of Life Science & School of Medicine (since 2022), NTHU, Taiwan
2014-2015, Chairman, Tsing Hua Interdisplinary Program, NTHU, Taiwan
2014-2015, Chief, Division of Faculty Development and Teaching Assistants Training, Center for Teaching and Learning Development, NTHU, Taiwan
2010-2014, Associate Professor, Institute of Molecular and Cellular Biology & Department of Life Science, NTHU, Taiwan
2005-2010, Assistant Professor, Institute of Molecular and Cellular Biology & Department of Life Science, NTHU, Taiwan
2004-2005, Assistant Researcher, Department of Cell and Tissue Biology, University of California, San Francisco (UCSF), USA
1998-2003, Postgraduate Researcher, Department of Cell and Tissue Biology, UCSF, USA
1998-2001, Postdoctoral Scholar, Department of Cell and Tissue Biology, UCSF, USA
1989-1990, Assistant Research Fellow, Division of Immunology, Development Center for Biotechnology, Taiwan

Education

1998, Ph.D. University of California-Davis, CA, USA
1989, M.S. National Taiwan University, Taipei, Taiwan
1987, B.S. National Chung-Hsing University, Taichung, Taiwan

Awards and Honors

2024, Talent Development Fund Academic Research Award, National Tsing Hua University, Taiwan
2022, Honorary Mentor Award, National Tsing Hua University, Taiwan
2022, 2017, 2013 Outstanding Mentor Award, National Tsing Hua University, Taiwan
2020, Honorary Teaching Award, National Tsing Hua University, Taiwan
2020, 2017, 2013 Teaching Award, National Tsing Hua University, Taiwan

Selected publications (2020-2025)

1. M.-C. Yang, W.-L. Huang, H.-Y. Chen, S.-H. Lin, Y.-S. Chang, K.-Y. Tseng, H.-J. Lo, I-C. Wang, C.-J. Lin*, **C.-Y. Lan*** (2025) Deletion of *RAP1* affects iron homeostasis, azole resistance, and virulence in *Candida albicans*. *mSphere.* 10(5):e0015525. (***Corresponding author**)
2. W.-H. Wang, H.-Y. Chen, S.-Y. Chen, **C.-Y. Lan*** (2024) Transcriptional profiling reveals the role of *Candida albicans* Rap1 in oxidative stress response. *Bioscience Reports.* Nov 22:BSR20240689. (***Corresponding author**)
3. Y.-R. Wang, S.-M. Chang, J.-J. Lin, H.-C. Chen, L.-T. Lee, D.-Y. Tsai, S.-D. Lee, **C.-Y. Lan**, C.-R. Chang, C.-F. Chen, C.-S. Ng (2024) A comprehensive study of Z-DNA density and its evolutionary implications in birds. *BMC Genomics.* 25:1123.
4. K.-Y. Tseng, Y.-C. Liao, Y.-Z. Chen, F.-C. Chen, F.-J. Chen, H.-K. Sytwu, L.-Y. Hsieh, **C.-Y. Lan***, H.-J. Lo* (2024) Rapid identification of the predominant azole-resistant genotype in *Candida tropicalis*. *FEMS Yeast Research.* 24:foae025. **(*Corresponding author)**
5. Y.-Z. Chen, K.-Y. Tseng, S.-C. Wang, C.-L. Huang, C.-C. Lin, Z.-L. Zhou, D.-J. Tsai, C.-M. Lin, Y.-L. Chen, K.-T. Chen, Y.-C. Liao, F.-J. Chen, H.-K. Sytwu, **C.-Y. Lan**, H.-J. Lo* (2023) Fruits are vehicles of drug-resistant pathogenic *Candida tropicalis*. *Microbiology Spectrum.* 11(6):e0147123.
6. S.-Y. Chen, C.-K. Chang, **C.-Y. Lan*** (2023) Antimicrobial peptide LL-37 disrupts plasma membrane and calcium homeostasis in *Candida albicans* via the Rim101 pathway. *Microbiology Spectrum.* 11(6):e0255123. **(*Corresponding author)**
7. W.-H. Wang, T.-X. Lai, Y.-C. Wu, Z.-T. Chen, K.-Y. Tseng, **C.-Y. Lan*** (2022) Associations of Rap1 with cell wall integrity, biofilm formation, and virulence in *Candida albicans*. *Microbiology Spectrum.* Nov 23:e0328522. **(*Corresponding author)**
8. C.-K. Chang, M.-C. Yang, H.-F. Chen, Y.-L. Liao, **C.-Y. Lan*** (2022) The role of Sfp1 in *Candida albicans* cell wall maintenance. *Journal of Fungi.* 8:1196. **(*Corresponding author)**
9. V. Nogueira, C.-K. Chang, **C.-Y. Lan**, C. Pereira, V. Costa, V. Teixeira (2022) Causative links between ER stress and oxidative damage in a yeast model of human N88S seipinopathy. *Free Radical Biology and Medicine. 192:165-181.* (The perspective of this article is published in Neural Regen Res, 2023, 18:1719-20.)
10. C.-Y. Tsai, E. O. Salawu, H. Li, G.-Y. Lin, T.-Y. Kuo, L. Voon, ... C.-C. Wu, **C.-Y. Lan***, H.-W. Fu* and L.-W. Yang* (2022) Helical structure motifs made searchable for functional peptide design. *Nature Communications.* 13:1062. **(*Corresponding author)**
11. C.-M. Hsu, Y.-L. Liao, C.-K. Chang, **C.-Y. Lan*** (2021) *Candida albicans* Sfp1 is involved in the cell wall and endoplasmic reticulum stress responses induced by human antimicrobial peptide LL-37. *International Journal of Molecular Sciences.* 22(19):10633. **(*Corresponding author)**
12. C.-K. Chang, M.-C. Kao*, **C.-Y. Lan*** (2021) Antimicrobial activity of the peptide LfcinB15 against *Candida albicans*. *Journal of Fungi*. 7(7):519. **(*Corresponding author)**
13. M.-F. Lin, Y.-Y. Lin, **C.-Y. Lan** (2020) Characterization of biofilm production in different strains of *Acinetobacter baumannii* and the effects of chemical compounds on biofilm formation. *PeerJ.* 8:e9020.
14. R.-C. Chen, **C.-Y. Lan*** (2020) Human antimicrobial peptide hepcidin 25-induced apoptosis in *Candida albicans*. *Microorganisms.* 8(4):585. **(*Corresponding author)**

15. G.-Y. Lin, C.-F. Chang, **C.-Y. Lan*** (2020) The interaction between carbohydrates and the antimicrobial peptide P-113Tri is involved in the killing of *Candida albicans*. *Microorganisms.* 8(2):299. **(*Corresponding author)**

Session III : Talk 2

Exploring the Multifaceted Roles of Rap1 in *Candida albicans*

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Rap1 is a DNA-binding protein conserved across yeasts, protozoa, and mammals for its role in telomeric maintenance. Notably, distinct differences have been identified between Rap1 in the model yeast *Saccharomyces cerevisiae* and the pathogenic yeast *Candida albicans*. First, *C. albicans* Rap1 lacks the C-terminal domain found in *S. cerevisiae* Rap1, which is implicated in telomere regulation. Second, *RAP1* is essential for viability in *S. cerevisiae,* whereas it is not-essential in *C. albicans*. These findings suggest that Rap1 has diverged functionally between these two species. This presentation will highlight our recent findings on the novel functions of *C. albicans* Rap1, particularly those related to pathogenesis and virulence.

Curriculum Vitae

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Education / Professional appointments

2024-now	**Assistant professor**, Laboratory of Host-microbe interactions and cell dynamics, Department of Medical Biotechnology and Laboratory Science, Chang Gung University, Taoyuan, Taiwan
2018-2024	**Assistant professor**, Laboratory of Host-microbe interactions and cell dynamics, Institute of Microbiology and Immunology, National Yang Ming Chiao Tung University, Taipei, Taiwan
2017-2018	**Postdoctoral research scholar**, Laboratory of Human Immunology and Infectious Diseases Graduate Institute of Clinical Medical Sciences, Chang Gung University (*Cheng-Lung Ku, PhD*), Taoyuan, Taiwan
2015-2017	**Postdoctoral fellow**, Department of Medical Biotechnology and Laboratory Science, Chang Gung University (*Hsin-Chih Lai, PhD*), Taoyuan, Taiwan
2010-2014	**Ph.D**, B3MI doctoral school, University Paris 7, France. (Pasteur-Paris University International doctoral program) Institut Pasteur, Biology of infection unit, INSERM U1117, (*Marc Lecuit, MD, PhD*)
2006-2008	**M.Sc.**, Department of Clinical Laboratory Sciences and Medical Biotechnology, National Taiwan University, College of Medicine, Taipei, Taiwan
2002-2006	**B.Sc.**, Department of Clinical Laboratory Sciences and Medical Biotechnology, National Taiwan University College of Medicine, Taipei, Taiwan

Research interests

- Microbe host interactions (微生物宿主交互作用)
- Reverse translational medicine in infectious diseases (感染症反轉譯醫學)
- Barrier tissue immunology (屏障組織免疫)

Selected Awards / Achievements

2024	Project for Excellent Junior Research Investigators (優秀年輕學者研究計畫), NSTC, Taiwan
2023	Fellow of Higher Education Academy, United Kingdom
2023	Excellent Teaching Award, National Yang Ming Chiao Tung University
2020	Excellent Teaching Award, National Yang Ming Chiao Tung University
2017-2020	Postdoctoral research scholar(獨立博士後研究學者計畫), MOST, Taiwan

Selected Publications

- Disson O, Charlier C, Pérot P, Leclercq A, Paz RN, Kathariou S, **Tsai YH**, Lecuit M. Listeriosis. **Nat Rev Dis Primers**. 2025. *In press*.
- Hafner L, Gadin E, Huang L, Frouin A, Laporte F, Gaultier C, Vieira A, Maudet C, Varet H, Moura A, Bracq-Dieye H, Tessaud-Rita, Maury M, Dazas M, Legendre R, Gastineau P, **Tsai YH**, Coppée JY, Charlier C, Patin E, Chikhi R, Rocha E, Leclercq A, Disson O, Hugues Aschard, Lecuit M. Differential stress responsiveness determines intraspecies virulence heterogeneity and host adaptation in *Listeria monocytogenes*. **Nat Microbiol.** 2024 Dec;9(12):3345-3361. doi: 10.1038/s41564-024-01859-8. Epub 2024 Nov 22.
- Tseng KY, Huang YT, Huang YT, Su YT, Wang AN, Weng WY, Ke CL, Yeh YC, Wang JJ, Du SH, Gu ZQ, Chen WL, Lin CH, **Tsai YH***. Regulation of candidalysin underlies *Candida albicans* persistence in intravascular catheters by modulating NETosis. **PLoS Pathog.** 2024 Jun 17;20(6):e1012319. doi: 10.1371/journal.ppat.1012319. ****lead contact***
- **Tsai YH***, Moura A*, Gu ZQ, Chang JH, Liao YS, Teng RH, Tseng KY, Chang DL, Liu WR, Huang YT, Leclercq A, Lo HJ, Lecuit M, Chiou CS. Genomic surveillance of *Listeria monocytogenes* in Taiwan, 2014 to 2019. **Microbiol Spectr.** 2022 Dec 21;10(6):e0182522. doi: 10.1128/spectrum.01825-22. ****co-first author***
- Herrera-Heredia SA, Hsu HP, Kao CY, **Tsai YH**, Yamaguchi Y, Roers A, Hsu CL, Dzhagalov IL. Heparin is required for the formation of granules in connective tissue mast cells. **Front Immunol.** 2022 Nov 9;13:1000405.
- Shih HP, Ding JY, Sotolongo Bellón J, Lo YF, Chung PH, Ting HT, Peng JJ, Wu TY, Lin CH, Lo CC, Lin YN, Yeh CF, Chen JB, Wu TS, Liu YM, Kuo CY, Wang SY, Tu KH, Ng CY, Lei WT, **Tsai YH**, Chen JH, Chuang YT, Huang JY, Rey FA, Chen HK, Chang TW, Piehler J, Chi CY, Ku CL. Pathogenic autoantibodies to IFN-γ act through the impedance of receptor assembly and Fc-mediated response. **J Exp Med**. 2022 Sep 5;219(9):e20212126. doi: 10.1084/jem.20212126.
- Maudet C, Kheloufi M, Levallois S, Gaillard J, Huang L, Gaultier C, **Tsai YH**, Disson O, Lecuit M. Bacterial inhibition of Fas-mediated killing promotes neuroinvasion and persistence. **Nature**. 2022 Mar;603(7903):900-906.
- Guo J, Ning XQ, Ding JY, Zheng YQ, Shi NN, Wu FY, Lin YK, Shih HP, Ting HT, Liang G, Lu XC, Kong JL, Wang K, Lu YB, Fu YJ, Hu R, Li TM, Pan KS, Li XY, Huang CY, Lo YF, Chang IY, Yeh CF, Tu KH, **Tsai YH**, Ku CL, Cao CW. Anti-IFN-γ autoantibodies underlie disseminated *Talaromyces marneffei* infections. **J Exp Med. 2020** Dec 7;217(12):e20190502.
- **Tsai YH*** and Chen WL. Host lipid rafts as the gates for *Listeria monocytogenes* infection: a mini-review. **Front Immunol. 2020** Aug 11;11;1666 ****lead contact***
- Gu ZQ, Tseng KY, and **Tsai YH***. *Candida* gut commensalism and inflammatory disease. **Medicine in Microecology 2020** Mar;3:100008 * ***lead contact***
- Maury MM*, **Tsai YH***, Charlier C, Touchon M, Chenal-Francisque V, Leclercq A, Criscuolo A, Gaultier C, Rousse S, Brisabois A, Disson O, Rocha EP, Brisse S, Lecuit M. Uncovering *Listeria monocytogenes* hypervirulence by harnessing its biodiversity. **Nat Genet. 2016** Mar 48(3)308-13 ****co-first author***
- Gessain G, **Tsai YH**, Travier L, Bonazzi M, Grayo S, Cossart P, Charlier C, Disson O, Lecuit M. PI3-kinase activation is critical for host barrier permissiveness to *Listeria monocytogenes*. **J Exp Med. 2015** Feb 9;212(2):165-83.
- **Tsai YH**, Disson O, Bierne H, Lecuit M. Murinization of internalin extends its receptor repertoire, altering *Listeria monocytogenes* cell tropism and host responses. **PLoS Pathog 2013**;9(5):e1003381

Session III : Talk 3

Fanning the Flames: Turning Inflammation into a Colonization Weapon by a Commensal Fungus

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Expansion of *Candida albicans* in the gut is associated with inflammatory diseases and susceptibility to systemic candidiasis. The hypha-specific adhesin Als3 and the secreted cytolytic peptide candidalysin are critical virulence factors for epithelial activation and infection. Here, we demonstrate that Als3 and candidalysin act synergistically to promote ileal retention and persistent fungal colonization. Their combined activity drives neutrophil infiltration into the lamina propria and their interactions with enteric neurons, thereby modulating intestinal motility. Mechanistically, Als3 facilitates fungal targeting to villous M cells, enabling candidalysin-dependent activation of the EGFR–c-Fos axis. Pharmacological inhibition of EGFR abrogates c-Fos nuclear translocation, neutrophil recruitment, and intestinal motility, ultimately impairing colonization. These findings reveal a coordinated adhesin-toxin strategy by which *C. albicans* subverts epithelial immune signaling to promote gut persistence while preserving barrier integrity. Our study highlights EGFR signaling as a potential therapeutic node for modulating fungal colonization in the context of host–fungus commensalism.

Session IV : Talk 1

Orthologous Transcription Factor Replacement Reveals that Stable TFIIIC Complexes are Required for Proper Mitotic Chromosome Segregation

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Transcription factors are speculated to play crucial roles in adaptive evolution. Using ortholog replacement of essential transcription factors (eTFs) from other yeast species, we investigated how eTFs can change. Several orthologs of eTFs could not fully complement deletion mutants of the *Saccharomyces cerevisiae* counterpart genes, indicating that functions or interactions of these eTFs have changed, rendering them incompatible. We further characterized TFIIIC, a fast-evolving protein complex that assists RNA polymerase III-mediated transcription, which exhibited complete or partial incompatibility in several subunits. In the orthologous Tfc7-replacement line, binding of TFIIIC to tRNA genes was reduced, but tRNA abundance was not severely affected. However, the chromosomes of Tfc7-replacement cells were often mis-segregated during mitosis and their fitness was further reduced in a spindle checkpoint mutant. Our chromatin-immunoprecipitation experiments uncovered that unstable TFIIIC binding results in defective cohesion loading, leading to chromosome mis-segregation. Swapping the highly divergent C-terminal domain of Tfc7 orthologs rescued its interaction with Tfc1 and cell fitness, supporting that altered interactions between complex subunits cause incompatibility. Our results reveal distinct essential functions of a well-studied protein complex.

Session IV : Talk 2

The Role of Ndt80 in Copper Tolerance of *Candida albicans*

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Copper is a crucial cofactor for sustaining multiple cellular electron-transfer reactions, making it an essential element for life. However, excess copper levels can cause structural damage and cell death through the production of reactive oxygen species (ROS) and non-specific attack on proteins. Moreover, immune cells, including neutrophils and macrophages, accumulate copper to induce oxidative bursts that kill engulfed pathogens. Therefore, a delicate copper homeostasis system is required for the human commensal fungus *Candida albicans* to thrive in extreme host environments. Interestingly, *C. albicans* exhibits higher copper tolerance than the nonpathogenic model yeast *Saccharomyces cerevisiae*, suggesting the presence of a specific copper tolerance mechanism that supports its adaptability to copper stress. Ndt80 is a multifunctional transcription factor that regulates several biological processes in *C. albicans*, including hyphae formation and biofilm maintenance, and drug resistance. This study further reveals that Ndt80 may contribute to the copper tolerance mechanism by maintaining plasma membrane integrity and regulating copper-dependent superoxide dismutases (Sods). Additionally, RNA-sequencing and other approaches uncovered the involvement of Ndt80 in mitochondrial respiration under cytotoxic copper conditions, further supporting the association of Ndt80 with copper tolerance. In summary, these results expand the previously known functions of Ndt80 and provide new insights into copper tolerance in *C. albicans*.

Session IV : Talk 3

Predominant Azole-Resistant *Candida tropicalis* Clade 4 Genotype: Environmental Persistence and Advances in Molecular Diagnostics

Kuo-Yun Tseng (曾國鋆)[1], Yin-Zhi Chen(陳盈之)[1], Min-Nan Tseng(曾敏南)[2], Jyh-Nong Tsai(蔡志濃)[3], Hsing-Lung Liu(劉興隆)[4], Yu-Chieh Liao(廖玉潔)[1], Chih-Chao Lin(林志兆)[1], De-Jiun Tsai(蔡德君)[1], Feng-Jui Chen(陳逢叡)[1], Li-Yun Hsieh(謝禮雲)[1], Chiao-Mei Lin(林巧梅)[1], Hsiu-Jung Lo (羅秀容)[1]

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The Taiwan Surveillance of Antimicrobial Resistance of Yeasts (TSARY), initiated in 1999 and conducted every four years, has identified *Candida tropicalis* as a major cause of non-albicans candidemia, with a notably higher resistance rate to fluconazole compared to other *Candida* species. Importantly, fluconazole-resistant *C. tropicalis* isolates also show cross-resistance to other azoles. Moreover, clade 4 is the predominant azole-resistant genotype (90.2%) among 92 isolates. In addition, of the 334 *C. tropicalis* isolates collected from TSARY 2022, most of the clade 4 isolates were resistant to azole (32/39). On the other hand, we conducted surveillance of pathogenic yeast in fruits, hospital environments, and orchards. From those surveillances, we found that *C. tropicalis* was one of most frequently identified species (8.1% in fruit surface, 8.0% in hospital environment and 13.0% in orchard) and clade 4 was the most frequently detected in fluconazole-resistant *C. tropicalis* isolates (100% in fruit surface and hospital environment and 81.8% in orchard). Furthermore, the primary resistance mechanism in clade 4 *C. tropicalis* is overexpressing mutated *ERG11*, the azole target. This study aims to develop methods for the rapid identification of the predominant azole-resistant *C. tropicalis* genotype. Our genomic analysis revealed that the sequences of CTRG_05978 and *SNQ2* are strongly linked to the clade 4 genotype and can be used for rapid detection (Sensitivity: 0.982; Specificity: 0.898, based on 165 Taiwanese isolates and 161 global isolates). Additionally, we established a comprehensive surveillance method using multiplex PCR targeting nine genes, followed by MinION sequencing. By optimizing annealing temperature, extension length, and primer set amounts, we established a method enabling the simultaneous processing of 96 samples in a 96-well plate. We also created a program for automatic assignment of diploid sequencing types, ensuring accurate and efficient routine molecular typing. Overall, this study offers significant advancements in the diagnosis and management of drug-resistant fungal infections, highlighting its potential impact on clinical practices.

Session IV : Talk 4

TRPML2 as a Molecular Sentinel Directing Vesicle Trafficking During Fungal Invasion

Zi-Qi Gu (顧子奇) [1, 2], Mu-Lin Liu (劉睦琳)[1], Yu-Huan Tsai (蔡雨寰)[1, 3, 4, 5] and Cheng-Chang Chen (陳政彰)[6]

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Candida albicans is a dimorphic fungal pathogen that extends hyphae to invade host cells. The host's intracellular vesicle trafficking system plays a crucial role in maintaining *Candida*-containing vacuoles, thereby supporting host cell survival. However, the molecular mechanisms governing vesicle dynamics during *C. albicans* invasion remain poorly understood. In this study, we used an *ece1Δ/Δ* mutant strain lacking candidalysin to uncover the role of the endolysosomal ion channel TRPML2 as a key regulator of $Rab4^+$ fast recycling endosomes involved in antifungal defense. Human monocytes deficient in TRPML2 showed increased *C. albicans* invasion, indicating a critical role for this channel in restricting fungal entry. We demonstrate that TRPML2 is activated by the acidic phosphoinositide $PI(3,5)P_2$. A phosphoinositide-insensitive TRPML2 mutant (R310A) exhibited diminished channel activity and impaired $Rab4^+$ endosome motility, with the vesicles accumulating in the Golgi. Notably, pharmacological activation of the R310A mutant restored vesicle dynamics and antifungal function, suggesting that TRPML2 is a druggable target essential for maintaining Rab4-dependent trafficking during fungal invasion. Interestingly, these TRPML2-dependent defenses were absent during infection with candidalysin-expressing *C. albicans*. In these cases, $TRPML2^+$ vesicles failed to localize to invasion sites and did not reduce fungal invasion, implicating candidalysin as a potential immune evasion factor that disrupts TRPML2-mediated host defense. Together, our findings identify TRPML2 as a $PI(3,5)P_2$-gated ion channel that orchestrates endosomal trafficking to counter fungal invasion and suggest a novel mechanism by which *C. albicans* subverts intracellular defenses via candidalysin.

Session IV : Talk 5

Dectin-1 Triggered Mucus Acts as a Selective Barrier against Filamentous *Candida albicans*

Wei-Lin Chen (陳瑋蓀)[1, 2, 3], Kuo-Yao Tseng(曾國堯)[2], Zi-Qi Gu (顧子奇)[1, 2], and Yu-Huan Tsai (蔡雨寰)[1, 4, 5, 6]

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Candida albicans is a polymorphic fungus commonly found in the human gastrointestinal tract. Its overgrowth in feces has been linked to systemic candidemia and various inflammatory diseases. While genes associated with adhesion and host cell damage are highly expressed during filamentous growth, *C. albicans* mutants with reduced filamentation and virulence gene expression exhibit an advantage in gut colonization. These low-virulence mutants can be selected in vivo through experimental evolution, highlighting a role for host factors in shaping *C. albicans* commensalism. Here, we demonstrate that *C. albicans* rapidly induced mucus secretion in the mouse small intestine within 25 minutes via the β-glucan–Dectin-1 axis, but not in the colon. We further show that the secreted mucus selectively bound to filamentous, but not yeast-form, *Candida* cells in a manner dependent on Sap6, a secreted and surface-anchored protein highly expressed in filamentous growth. This Dectin-1–dependent mucus burst in the small intestine facilitated the clearance of filamentous cells by transiting them to the colon and limiting their interaction with the intestinal epithelium. Our findings uncover a segment-specific host defense mechanism that reduces the burden of virulent *C. albicans* cells without triggering barrier-compromising inflammation. This mechanism supports the predominance of yeast-form cells in the small intestine and explains the selective advantage of low-virulence *C. albicans* strains during gut colonization.

Posters		
Poster 1	Fidelia Amanda Djinarwan 徐明霞	Developing a Tetracycline-Inducible System in *Schizosaccharomyces japonicus* to Investigate Nuclear Envelope Remodeling and Sexual Reproduction
Poster 2	HAO YUNYIN 殷浩雲	Feeding Behavior of Slime Mold and the Effects of Symbiotic Microorganisms on Its Growth
Poster 3	Dong-Sheng Yang 楊東昇	酵母菌於針蕊絨泡黏菌共生實驗中的不可替代性與體內菌適合度影響探討
Poster 4	Dinh-Dong Le 黎廷東	Exploring the role of the CCAAT-binding complex in cell wall maintenance and biofilm formation in *Candida albicans*
Poster 5	Tang-Yu Zhu 朱堂瑀	Beyond Mitosis: Investigating the Function of Nuclear Envelope Assembly Proteins in Sexual Reproduction
Poster 6	Yao-De Huang 黃耀德	Vacuolar Iron Storage is Correlated to Mitochondrial Decline Across the Stationary Yeast Cells
Poster 7	Shu-Yao Hsu 許書堯	Quantitative Analysis of Yeast Mitochondrial Morphology Under Conditioned Medium Treatment
Poster 8	Li-Ting Jang 詹琍婷	Taiwan Yeast Bioresources Center
Poster 9	Po-Chen Hsu 許博琛	Transcriptional Rewiring and Mitochondrial Evolution in Yeasts
Poster 10	Pei-Juan Cai 蔡佩娟	Phosphorylation of Golgin Imh1 by AMPK/Snf1 Compromises Golgi Compartmentalization by Releasing Arl1-Imh1 Axis
Poster 11	Hsuan-Te Chao 趙軒德	Dissecting the Role of SUMOylation in Ribosome Biogenesis: Insights from the Mss4–Bcp1–uL14 Pathway
Poster 12	Lavernchy Jovanska 陳秋玲	Differential Control of Meiotic Sister Chromatin Cohesion and Interhomolog Recombination in Different Ascomycete Fungi
Poster 13	Wei-Tang Xu 徐偉棠	Mechanisms Contributing to Fluconazole Resistance of *Nakaseomyces glabratus* (*Candida glabrata*) isolated in Taiwan
Poster 14	Ya-Ling Hung 洪雅玲	Rad51, Rad54, and ZMM Proteins Antagonize the Mismatch Repair System to Promote Fertility of Budding Yeast Intraspecies Hybrid Zygotes
Poster 15	Kuan-Yu Lin 林冠妤	Investigating the Underlying Dynamics of Fission Yeast Cell Polarity
Poster 16	Yu-Wen Ni 倪郁雯	Regulatory Mechanisms and Interplay Among the Dedicated Chaperones of Ribosomal Proteins
Poster 17	Wei-Ming Lee 李偉銘	Ribosomal Lysine Methyltransferases Correlate with Stress Responses
Poster 18	Man-Ling Chao 趙曼伶	The Function of RNA Helicase Fal1 in 90S Ribosome Biogenesis and its Relationship with eIF4G1 and Sgd1
Poster 19	Hao-Yun Tang 湯皓雲	The role of Mitochondrial Membrane Contact Sites in Turnover of Sdh1 in the Mitochondria-Associated Degradation (MAD) Pathway
Poster 20	Hsin-Hui Su 蘇歆惠	Characteristics of Fluconazole Resistance Mechanisms in *Candida tropicalis* Isolates from Taiwan
Poster 21	Yen-Chen Liu 劉晏甄	The Molecular Mechanism of Arl3-Mediated Arl1 Activation
Poster 22	Pei-Jung Chung 鍾佩蓉	Mixed Yeast Infections in TSARYs 2002-2022
Poster 23	Qian-Yu Guo 郭倩妤	The Distribution of Clinically Isolated *Candida* Species from Sterile Sites in Taiwan (2002–2022)
Poster 24	Yu-Lian Chen 陳玉蓮	Surveillance of *Candida* Species and Antifungal Susceptibility in Taiwan: Insights from TSARY in 2022

Poster 1

Developing a Tetracycline-Inducible System in *Schizosaccharomyces japonicus* to Investigate Nuclear Envelope Remodeling and Sexual Reproduction

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Integrity of the nuclear envelope (NE) is critical for the function of the genome. The fission yeast *Schizosaccharomyces japonicus* is an emerging model organism for NE remodeling. However, research tools for acute manipulation of gene expression levels are still unavailable for *S. japonicus*. This study aims to develop an improved tetracycline-inducible system for *S. japonicus*, enabling precise control over gene expression for the study of NE remodeling and other cellular processes. Using this potent tool, we aim to determine whether Cmp7, a protein known to be required for NE assembly after mitosis, has a specific and previously undescribed function during sexual reproduction, as our unpublished data found that sexual reproduction is abolished in *S. japonicus* cells lacking Cmp7. To further investigate Cmp7's impact on sexual reproduction, we will also employ genetically encoded multimeric nanoparticles (GEMs) as nuclear fusion markers, a key step during sexual reproduction of *S. japonicus* cells. The completion of proposed research will advance our understanding of the function of Cmp7 and importance of NE integrity under distinct cellular contexts.

Poster 2

Feeding Behavior of Slime Mold and the Effects of Symbiotic Microorganisms on Its Growth

HAO-YUN YIN (殷浩雲)[1]

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Slime molds, although unicellular, exhibit remarkable behavioral complexity and are widely used as model organisms in studies of decision-making and symbiosis. They typically inhabit dark, moist environments such as decaying wood or damp soil and are broadly categorized into plasmodial and cellular types. ***Physarum oblonga*** and ***Physarum polycephalum***, members of the plasmodial slime molds (Myxogastria), were the focus of this study.

We investigated both the behavioral ecology and microbial associations of ***P.oblonga***. In feeding experiments, ***P.oblonga*** demonstrated distinct preferences for specific yeast strains. For example, it significantly preferred ***Hanseniaspora osmophila*** over ***Wickerhamomyces anomalus***, and ***Dekkera bruxellensis*** over ***Kazachstania exigua*** ($p < 0.05$). When a third yeast species was introduced as a decoy, a strong preference shift was observed ($p < 0.001$), suggesting the presence of context-dependent decision-making and possible memory-like behavior. Preferences varied among different slime mold strains, highlighting intraspecific behavioral diversity.

In parallel, microbial profiling revealed that even after multiple subcultures under sterile conditions, ***P. polycephalum*** retained a specific symbiotic bacterium, ***Leifsonia shinshuensis***, within its body. Different strains of this bacterium were selectively retained by ***P. polycephalum***. We found that ***L. shinshuensis*** produces carotenoids in response to light and enhances slime mold growth under oxidative stress. Co-culturing experiments under light and hydrogen peroxide exposure further demonstrated that this bacterial symbiont conferred stress resistance, likely via peroxidase activity.

Together, our findings illustrate both the cognitive-like decision-making abilities and adaptive microbial symbiosis of ***P. polycephalum***, shedding light on the complex interactions between slime molds and their microbial environment.

Poster 3

酵母菌於針箍絨泡黏菌共生實驗中的不可替代性與體內菌適合度影響探討

Dong-Sheng Yang (楊東昇)[1]

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This study focuses on the impact of symbiotic bacteria on the fitness of the slime mold *Physarella oblonga*, and investigates whether the baking yeast(*Saccharomyces cerevisiae*) plays an un-replaceable role as food source. First, bacteria were isolated from different slime mold strains and used in feeding experiments to compare their effects on survival through subcultures and fruting body formation. Results revealed that no symbiotic bacteria can use for yeast across three generations of subcultures, indicating the significance of yeast in slime mold cultivation. To explore more about this, mixed feeding experiments were conducted using combinations of yeast and symbiotic bacteria at different ratios, assessing the impact on reproductive fitness as indicated by spore count.

Some bacteria, such as *Brucella pseudogrignonense* (W2), were found to enhance slime mold fitness when present at high concentrations. However, this effect diminished after symbiotic bacteria were removed from slime mold, suggesting that these symbiotics may play a support role in digestion food. Try to remove symbiotic bacteria using antibiotics showed that *Serratia marcescens* was particularly resilient, indicating a potentially specific symbiotic relationship and possible protective mechanisms within the slime mold.

The study also explored whether slime molds exhibit selective preferences when encountering their symbiotic bacteria. Results shows no significant diffrence, shows that symbiotic selection may not be choose by slime mold contact but by bacteria. Overall, yeast remains un-replaceable in the cultivation of slime molds, while symbiotic bacteria may indirectly enhance fitness by helping digestion and nutrient absorption.

Poster 4

Exploring the role of the CCAAT-binding complex in cell wall maintenance and biofilm formation in *Candida albicans*

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The *Candida albicans* Hap complex is a CCAAT-binding complex (CBC) known to regulate iron homeostasis through its association with another transcription factor, Hap43. Interestingly, diverse functions of the CBC independent of Hap43 have also been identified. In this study, we further explored new roles of the CBC and found that it is also involved in cell wall maintenance and biofilm formation in *C. albicans*. Moreover, while the small GTPase Rhb1 and the Mkc1 signaling pathway contribute to CBC-mediated maintenance of the cell wall, CBC-mediated biofilm formation appears to be independent of Rhb1. Finally, RNA sequencing (RNA-seq) and data analysis revealed functional divergence between the two Hap3 orthologs, Hap31 and Hap32. Notably, this work provides new insights into the broad influence of the CBC in *C. albicans*.

Poster 5

Beyond Mitosis: Investigating the Function of Nuclear Envelope Assembly Proteins in Sexual Reproduction

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The nuclear envelope (NE) is essential for cellular function and integrity in all eukaryotic cells. Mutations in NE assembly proteins compromise NE integrity, causing nuclear leakage that are associated with various diseases such as cancers, premature ageing, and neurodegenerative diseases. Previously, using *Schizosaccharomyces japonicus*, a fission yeast that has conserved nuclear assembly mechanisms with humans, we demonstrated how the inner nuclear membrane protein Lem2, the ESCRT adaptor Cmp7, the ESCRT-III protein Vps32, and the ATPase Vps4, contributed to NE integrity in mitotic cells. Strikingly, we found that sporulation is abolished in cells lacking any of the aforementioned proteins. In addition, nuclear fusion, a critical step for gametogenesis, might be impaired in these mutants. To understand the chromosome dynamics during nuclear fusion, we crossed a strain carrying mCherry-tagged histone to another strain carrying GFP-tagged histone. In wild-type cells, successful nuclear fusion, indicated by overlapping signals of mCherry and GFP, was observed almost immediately after the two nuclei contacted each other. In *cmp7*Δ cells, however, the two signals did not merge, verifying failure in nuclear fusion. To understand if these NE assembly proteins play specific functions during gametogenesis, we are in the process of examining their localization during nuclear fusion. We found that Lem2 localized throughout the NE, including a thread-like structure connecting the two fusing nuclei. The importance and composition of the thread-like structure remains to be investigated. In parallel, we found that two mutations that could partially rescue the NE assembly defects in *cmp7*Δ could also partially rescue gametogenesis, suggesting that NE integrity may be required for sporulation in *S. japonicus*. Taken together, our study elucidated unexpected importance and potential functions of NE assembly proteins in sexual reproduction. Since sporulation plays an important role in transmission of parasitic yeasts, our study may inform on strategies to disrupt sexual reproduction in pathogenic yeasts for antifungal development in the future.

Poster 6

Vacuolar Iron Storage is Correlated to Mitochondrial Decline Across the Stationary Yeast Cells

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Yeast is one of the most widely used and accessible eukaryotic model organisms in biological research, owing to its short generation time and low cultivation cost. These advantages make it an ideal model for investigating time-dependent physiological changes. Various physiological parameters can be tracked over stationary growth to study progressive alterations in cellular function with time. To address this issue, we focus on a central regulator of cellular homeostasis, the mitochondria. Mitochondria are essential for cellular metabolism, playing key roles in oxidative phosphorylation and metabolic pathways. They also frequently interact with other organelles, such as the ER and lysosomes, to regulate metabolite trafficking and ion homeostasis. The functional integrity of mitochondria and lysosomes diminishes over time, contributing to progressive cellular decline and dysfunction commonly observed in long-lived eukaryotic cells. In *Saccharomyces cerevisiae*, the lysosome-like vacuole acts as the principal intracellular reservoir of iron. Proper sequestration of iron into the vacuole is essential to prevent excessive accumulation in the cytosol, which can lead to oxidative stress through uncontrolled redox activity. Emerging evidence highlights iron metabolism as a key factor influencing cellular integrity and metabolic resilience. We proposed that mitochondrial dysfunction, characterized by altered morphology and impaired bioenergetic output, may be linked to disrupted iron regulation. To examine this hypothesis, we constructed a Δ*ccc1* yeast strain lacking the vacuolar iron transporter. *CCC1* deletion resulted in a loss of vacuolar iron retention and a shift toward cytosolic iron accumulation. We monitored mitochondrial structure and physiological parameters across multiple growth phases to evaluate how impaired vacuolar iron storage contributes to metabolic instability. Our results revealed that a distinct mitochondrial morphology—characterized as granular—emerges under both high-iron conditions and during senescence in the Δ*ccc1* strain. These findings uncover mechanistic links between iron dysregulation and mitochondrial vulnerability, providing insight into the molecular basis of time-dependent cellular dysfunction.

Poster 7

Quantitative Analysis of Yeast Mitochondrial Morphology Under Conditioned Medium Treatment

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Mitochondria are essential organelles that not only generate ATP but also participate in key metabolic processes and maintain cellular homeostasis. They are highly dynamic and adapt their morphology in response to environmental changes. In yeast, mitochondria typically exhibit a tubular morphology during the log phase in fresh medium but shift to a fragmented form in the stationary phase. In this study, we aimed to develop a strategy for characterizing mitochondrial morphology changes as yeast transition from the log phase to the stationary phase. To mimic this shift, we treated log-phase yeast cells with conditioned medium derived from stationary-phase cultures. Interestingly, instead of fragmenting, mitochondria adopted a hyperfused morphology, distinct from both the log-phase and stationary-phase forms. We reconstructed the 3D mitochondrial structure and analyzed it using 14 descriptive factors, which were categorized into mitochondrial volume, surface area, and skeletonized length. We visualized these metrics with a t-SNE plot, which revealed that cells treated with conditioned medium for 30 minutes displayed a significant shift from the control group, characterized by larger and more interconnected mitochondrial networks. Notably, mitochondrial morphology reverted to its original form after more than 3 hours of recovery in fresh medium. These findings suggest that mitochondria respond dynamically and reversibly to environmental cues and that yeast cells in different growth phases exhibit distinct mitochondrial responses to the same stimulus. Moreover, our approach to characterizing mitochondrial morphology offers a more objective and quantitative framework for analyzing organelle dynamics. This work contributes to a better understanding of mitochondrial dynamic behavior and may be used to detect cellular adaptation during aging as well as metabolic switches.

Poster 8

Taiwan Yeast Bioresources Center

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The yeast model system has been used in biological research for over 60 years. In Taiwan, more than 40 laboratories use yeast as a model organism to investigate important biological questions. To support this research community, the Taiwan Yeast Bioresource Center was established to provide high-quality yeast strains, plasmids, antibodies and research reagents. The main tasks of the center include: (1) the identification and procurement of yeast strains and plasmids suitable for research; (2) the verification and preservation of yeast strains in frozen stocks; (3) the distribution of strains, plasmids and antibodies to researchers; (4) the production and supply of yeast-specific protein antibodies; and (5) the promotion of its services through participation in research conferences. Given the limited commercial availability of yeast antibodies, the Center has established a dedicated yeast antibody resource. To date, it has produced 30 common antibodies and is committed to producing at least five specific yeast protein antibodies annually, provided that more than five laboratories apply and the Central Executive Committee approves. The Center also provides CRISPR/Cas9 plasmids for genome editing applications. In addition, validated experimental methods and detailed protocols are made available to researchers free of charge. By offering a comprehensive collection of yeast research tools, the Taiwan Yeast Bioresource Center aims to support and strengthen the scientific research capacity of Taiwan's yeast research community.

Poster 9

Transcriptional Rewiring and Mitochondrial Evolution in Yeasts

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Organisms often evolve novel or adaptive phenotypes through the accumulation of mutations. These mutations may exert pleiotropic effects, influencing phenotypic plasticity or shaping evolutionary trajectories in response to specific selective pressures. Our research focuses on these processes in yeasts, investigating the evolution of transcriptional regulatory networks (TRNs) and the improvement of mitochondrial genome quality control (mtDNA QC). We have characterized transcriptional rewiring, a process in which transcriptional regulation evolves across species via modifications to cis-regulatory elements in promoters or substitutions of trans-acting regulatory factors within gene regulatory networks. This phenomenon is exemplified in Sef1 TRNs, which we have traced across multiple yeast species spanning at least 300 million years of divergence (Hsu et al., Mol Biol Evol, 2021; Hsu et al., Nucleic Acids Res, 2024). Additionally, we have demonstrated that mutations in a secondary TRN can compensate for defects arising from disruptions in the primary TRN (Hsu et al., EMBO Rep, 2023). Furthermore, using experimental evolution, we have validated the feasibility of improving mtDNA QC (Amine et al., Curr Biol, 2021). The evolution of TRNs and mitochondria is fundamental to speciation and phenotypic diversification, occurring through both neutral processes and selective pressures. Elucidating the underlying mechanisms will consolidate and invigorate not only basic molecular biology but also applied biomedicine.

Poster 10

Phosphorylation of Golgin Imh1 by AMPK/Snf1 Compromises Golgi Compartmentalization by Releasing Arl1-Imh1 Axis

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Golgins, coiled-coil proteins, are crucial for Golgi architecture and intracellular transport. Mammals have four GRIP-domain-containing Golgins, while budding yeast has a single conserved Golgin, Imh1. Imh1 is recruited to the Golgi membrane by the active small GTPase Arl1 via its GRIP domain. Despite extensive phosphorylation of Imh1 under various stress conditions observed in previous screenings, the biological significance and regulatory mechanisms of Imh1 phosphorylation remain unclear. This study reveals that Snf1, a yeast AMPK homologue, regulates the dissociation of the Arl1-Imh1 axis from the Golgi during glucose deprivation by phosphorylating Imh1 at Ser606, Ser802, and Ser804. The phosphomimetic mutant Imh1^{S606D,S802D,S804D} mislocalizes away from the Golgi, while the phospho-deficient mutant Imh1^{S606A,S802A,S804A}, prevents this mislocalization in an Arl1-dependent manner under glucose deprivation, indicating this change is not due to Arl1 inactivation. We also provide evidence that AMPK/Snf1 associates with Imh1 and directly phosphorylates Imh1, resulting in conformational change. Furthermore, we demonstrate that AMPK/Snf1-regulated Imh1 phosphorylation impairs its ability to support SNARE recycling in *ypt6*Δ mutants and compromises Golgi homeostasis. Collectively, these findings reveal how AMPK/Snf1-mediated phosphorylation drives the disassembly of the Arl1-Imh1 axis from the Golgi in response to low-energy conditions, highlighting the critical role of Imh1 phosphorylation in regulating Golgi function.

Poster 11

Dissecting the Role of SUMOylation in Ribosome Biogenesis: Insights from the Mss4–Bcp1–uL14 Pathway

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More than 200 factors are involved in the regulation of ribosome biogenesis, a process that must be tightly controlled to ensure proper cellular function and development. Post-translational modifications (PTMs) are dynamic protein alterations that respond to physiological conditions and environmental stimuli. Numerous ribosome biogenesis factors have been shown to undergo various PTMs, which can modulate their activity and interactions with the ribosome. SUMO (Small Ubiquitin-like Modifier) is a ubiquitin-like protein that is covalently attached to lysine residues on target proteins. Mutations in key components of the SUMOylation pathway have been shown to disrupt ribosome biogenesis, although the underlying mechanisms remain unclear. Mss4 is a phosphatidylinositol 4-phosphate 5-kinase responsible for generating $PI(4,5)P_2$ at the plasma membrane. Its export is regulated by Bcp1, a chaperone of the ribosomal protein uL14. Our data show that SUMOylation levels on pre-ribosomal subunits are elevated in the *mss4ts* mutant. Aberrant regulation of SUMO levels impairs ribosome synthesis, leading to defects in growth and ribosomal subunit export. The SUMOylation status of Bcp1 is regulated by the SUMO protease Ulp2 and the SUMO E3 ligase Mms21, which modulate its transport and interaction with other proteins. We also identified SUMOylation sites on uL14 and demonstrated that the loss of SUMOylation results in growth retardation and defects in ribosome synthesis, particularly at low temperatures. Furthermore, impaired SUMOylation of uL14 leads to abnormal retention of the essential biogenesis factor Tif6 on the ribosome. In conclusion, the phosphatidylinositol pathway can influence ribosome biogenesis by modulating the SUMOylation status of ribosome biogenesis factors and ribosomal proteins.

Poster 12

Differential Control of Meiotic Sister Chromatin Cohesion and Interhomolog Recombination in Different Ascomycete Fungi

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Regulation of meiotic sister chromatid cohesion protein Rec8 is known to be controlled genetically in budding and fission yeast. In budding yeast, Scc2 is required for Rec8 mRNA production and it recruits cohesin to activate Rec8 promoter for transcription. In fission yeast *Schizosaccharomyces pombe*, Mmi1 binding to a particular motif regulates the splicing and transcription of genes, including rec8, during vegetative or meiotic stage. In this study, we observed the epigenetic regulation of Rec8 in ascomycete fungus *Trichoderma reesei*. DNA methyltransferases (DNMTs) perform multiple tasks in the meiosis of the fungus. Three DNMT genes (*rid1*, *dim2*, and *dimX*) differentially regulate genome-wide cytosine methylation and C:G-to-T:A hypermutations in different chromosomal regions. Deletion of rid1 disrupted the Rad51-mediated DSB repair and normal meiosis, including the development of mature ascospores, with most *rid1*Δ asci producing only one nuclei. Similar phenotypes were observed in *rad51*Δ, showing locus heterogeneity (LH) relationship, in which LH-associated proteins often regulate interconnectivity in protein interaction networks. Interestingly, down-regulation of *rec8* (but not *rad51*) was observed in *rid1*Δ. Deletion of rec8 disrupted Rad51-mediated DSB repair, with 60% of asci producing only one nuclei, showing similar phenotypes with *rid1*Δ and *rad51*Δ. These indicated that *rid1-rad51* LH relationship is bridged through rec8 regulation epigenetically. In conclusion, our studies provide the first evidence of the involvement of DNMTs during meiosis.

Poster 13

Mechanisms Contributing to Fluconazole Resistance of *Nakaseomyces glabratus* (*Candida glabrata*) Isolated in Taiwan

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Candidiasis, caused by pathogenic *Candida* yeasts, frequently affects immunocompromised patients or those undergoing invasive treatments. *Candida albicans* is the most common species, followed by *Candida tropicalis* in Asia and *Nakaseomyces glabratus* (*Candida glabrata*) in western countries. *N. glabratus*' high azole resistance and biofilm-forming ability complicate treatment. This study utilizes *N. glabratus* isolates collected from 2002 to 2022 in the Taiwan Surveillance of Antimicrobial Resistance of Yeasts (TSARY), which tracks species distribution and drug susceptibilities in Taiwan. Among 1717 isolates, four were fluconazole-resistant, and fourteen showed reduced voriconazole susceptibility. This study investigated resistance mechanisms in all four fluconazole-resistant isolates and the two isolates with the lowest voriconazole susceptibility. Comparing the levels of expression of genes involved in drug resistance to that of the susceptible control isolate, all six resistant isolates had higher mRNA levels of *PDR1*, *CDR1*, and *PDH1* and four of them also had higher mRNA levels of *RPN4* and *FLR2.* Whole-genome sequencing revealed no increase in gene copy number, but identified fourteen mutations in the *CDR1* promoter, four in the *PDH1* promoter, and ten in the *FLR2* promoter, potentially regulating gene expression. Additionally, three nonsynonymous mutations in the *PDH1* open reading frame (ORF) may enhance protein activity. The transcription factor *PDR1*, regulating these efflux pump genes, harbored seven nonsynonymous ORF mutations, likely influencing its activity. Four of these mutations, including Y372C, have been previously linked to drug resistance and significantly upregulate *CDR1* and *PDH1* expression, while the three novel mutations await further validation. On the other hand, *PDR1* expression was higher in resistant strains compared to sensitive strains, and four mutations were detected in its promoter region. In contrast, no notable mutations were identified in the *RPN4* ORF or its promoter. These findings suggest that *PDR1* plays a key role in *N. glabratus* resistance through nonsynonymous ORF mutations and resultant overexpression of downstream ATP-binding cassette (*CDR1* and *PDH1*) and major facilitator superfamily (*FLR2*) efflux pump genes. Mutations in *PDR1, CDR1, PDH1*, and *FLR2* promoters may contribute to *N. glabratus* resistance, but their specific roles need further study. This study provides critical insights into *N. glabratus* resistance mechanisms in Taiwan.

Poster 14

Rad51, Rad54, and ZMM Proteins Antagonize the Mismatch Repair System to Promote Fertility of Budding Yeast Intraspecies Hybrid Zygotes

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Rad51 and meiosis-specific Dmc1 catalyze homologous recombination (HR) between maternal and paternal chromosomes during meiosis in many sexual eukaryotes, generating three interhomolog (IH) recombination products: noncrossovers (NCOs), class I crossovers (COs), and class II COs. Some COs form chiasmata, which *physically connect homologous chromosomes* and ensure proper chromosome segregation during meiosis I. Meiosis is highly relevant to speciation, with the mismatch repair (MMR) system believed to prevent IH-HR, leading to postzygotic isolation between closely related species. We report that several *Saccharomyces cerevisiae* HR proteins exhibit anti-MMR activities, including Rad51, Rad54, and synapsis-promoting ZMM proteins (Mer3, Zip1, Zip4, and Msh4) in SK1/S288c hybrid zygotes. Srs2 (an ortholog of *Escherichia coli* helicase UvrD) facilitates MMR by dissembling Rad51-ssDNA presynaptic filaments. Rad51 antagonizes MMR and Srs2 to catalyze D-loop formation. Rad54's anti-MMR activity acts after Srs2 and outcompetes its pro-HR function to promote Rad51-mediated IH-HR in hybrid zygotes. Dmc1 does not possess anti-MMR activity, but exhibits better mismatch tolerability than Rad51. Following D-loop formation mediated by Dmc1 and/or Rad51, ZMM proteins promote class I IH-CO formation while limiting MMR to promote NCO formation by Sgs1 (an ortholog of *E. coli* RecQ helicase) and prevent class II IH-CO formation by the Mms4•Mus81 endonuclease.

Poster 15

Investigating the Underlying Dynamics of Fission Yeast Cell Polarity

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Fission yeast establishes cell polarity to maintain its rod shape and enable bipolar growth. Polarity sites form at the cell ends primarily through the self-organization of polarity proteins, including the Rho GTPase Cdc42 and its nucleotide exchange factor Scd1. After cytokinesis, polarity is initially established only at the pre-existing end; later, the opposite end begins to accumulate polarity proteins, known as New End Take Off (NETO). Over time, the polarity site oscillates between the two ends, promoting bipolar growth. The mechanism underlying polarity establishment is better understood. In contrast, the oscillatory behavior of polarity sites is thought to involve p21-activated kinase (PAK), Shk1, a member of the polarity proteins, in negative feedback mechanisms of cell polarity by its kinase activity, but the mechanistic details and the underlying dynamics remain unclear. With the basis of core machinery, polarity site formation can be simulated with reaction-diffusion models. Here, we first investigate the interactions—both binding and phosphorylation—between PAK and other polarity proteins mathematically. Second, we quantified and analyzed signals of the polarity probe derived from numerous populations of cells to capture the dynamics of polarity sites. Together, by genetically introducing disruptions or modifications of these protein interactions, we would be able to verify predicted behaviors under microscopy and thus further understand the mechanisms leading to dynamics of polarity sites.

Poster 16

Regulatory Mechanisms and Interplay Among the Dedicated Chaperones of Ribosomal Proteins

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The ribosome complex is composed of ribosomal RNA (rRNA) and ribosomal proteins and plays a critical role in protein translation. Proper ribosome biogenesis is essential for cellular growth and development. Newly synthesized ribosomal proteins often contain unstructured extensions before associating with rRNA, rendering them susceptible to misfolding, aggregation, and degradation. To prevent these issues, the dedicated chaperones interact with specific ribosomal proteins, assisting in their proper folding and guiding them to the appropriate locations for ribosome assembly. However, the regulatory relationships among different chaperones remain poorly understood. In this study, we used *Saccharomyces cerevisiae* as a model to examine the expression patterns and subcellular localization under various environmental stresses of chaperones, and analyzed how the deletion or mutation of individual chaperones affects the expression and distribution of the others. Our results revealed that Sqt1, known to function in the cytoplasm as a chaperone for Rpl10, also interacts with the Bcp1–Rpl23 complex. Moreover, the expression level of Sqt1 influences both the abundance and nuclear/cytoplasmic distribution of Bcp1. Bcp1, in turn, affects the growth of mutants lacking Sqt1, Rpl10, and related factors. The expression, stability, and localization of Bcp1 are regulated by Rpl10 and Sqt1, while Bcp1 itself also modulates the expression and distribution of Sqt1, indicating an interdependence relationship. These findings suggest that disruption in the synthesis or function of a specific ribosomal protein can influence the production of another, forming a feedback mechanism that coordinates overall ribosome biogenesis.

Poster 17

Ribosomal Lysine Methyltransferases Correlate with Stress Responses

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Post-translational modifications (PTMs) play crucial roles in living organisms. While previous studies on methylation have focused on how histone methylation regulates transcription, the functional significance of methylation on ribosomal proteins remains largely unexplored. Moreover, no known demethyltransferase that removes methyl groups from lysine residues on ribosomal proteins. Understanding the regulatory role of this post-translational modification is the main objective of this study. In this research, we investigate the role of ribosomal protein methylation by deletion the expression of specific ribosomal lysine methyltransferases (RKMs) and observing their effects under various environmental stresses. Our results show that the *rkm5*Δ strain is highly sensitive to oxidative stress. This sensitivity is reduced translational capacity under oxidative stress conditions. Furthermore, activity assays of oxidative stress response enzymes “catalase and superoxide dismutase” revealed that while the deletion of *RKM1-4* had no significant effect during the stationary phase, *rkm5*Δ exhibited a decrease in enzyme activity. This trend persisted after hydrogen peroxide treatment and was particularly pronounced in SC medium. These findings indicate that Rkm5 plays a critical role in enabling yeast cells to cope with oxidative stress.

Poster 18

The Function of RNA Helicase Fal1 in 90S Ribosome Biogenesis and its Relationship with eIF4G1 and Sgd1

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In living organisms, ribosomes are primarily responsible for mRNA translation and protein synthesis, making ribosome biogenesis crucially linked to protein yield, quality, and cellular growth. In eukaryotes, ribosome assembly begins in the nucleolus and involves approximately 200 ribosome biogenesis factors, which assist in rRNA processing and modification, ribosomal protein assembly, and facilitate the export of the 90S pre-ribosome from the nucleus to the cytoplasm. The pre-ribosome matures into the 40S small subunit and the 60S large subunit, completing mRNA translation and protein synthesis in the cytoplasm. Eukaryotic translation initiation factor eIF4G, as a scaffold protein in the eIF4F complex, plays a key regulatory role in protein synthesis by linking eIF4A, eIF4E, and mRNA. Previous studies in the lab have shown that eIF4G1, in addition to playing a role in translation initiation and 60S large subunit biogenesis, also interacts with various 90S assembly factors, thereby contributing to the biogenesis of 40S small ribosomal subunit. Ribosome profiling analyses indicate that the absence of eIF4G1 reduces the levels of 40S ribosomes and abnormalities in pre-rRNA processing within upstream 90S pre-ribosomes. This suggests that eIF4G1 is closely associated with 90S assembly and interacts with the 90S assembly factor Fal1. Fal1, a homolog of eIF4A, is an ATP-dependent RNA helicase of the DEAD-box family. It forms a stable trimeric complex with the cofactor Sgd1 and eIF4G1, collectively regulating Fal1's ATPase activity. Helicase assays show that Fal1 can unwind RNA of specific lengths. Fal1 is an essential gene, and in the GAL::FAL1 strain, where Fal1 expression is reduced, the depletion of Fal1 results in a decrease in the levels of 40S ribosomes and polysomes, which subsequently impacts ribosome biogenesis, cellular growth, and translation. Mutants of Fal1, such as A67V (affecting ATP binding) and D173N (affecting helicase activity), also impair cellular growth, exhibiting phenotypes similar to Fal1 depletion. Therefore, Fal1 plays a critical physiological role in 90S pre-ribosome assembly.

Poster 19

The role of Mitochondrial Membrane Contact Sites in Turnover of Sdh1 in the Mitochondria-Associated Degradation (MAD) Pathway

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Mitochondrial-associated degradation (MAD) is a mitochondrial quality control pathway that degrades mitochondrial unfolded proteins by ubiquitination of the proteins, removal of the segregase Cdc48 and its co-factor Doa1, and delivery to the proteasome for degradation. Our recent research revealed that proteins located within the matrix, such as Pim1 and Kgd1, are MAD substrates and identified putative MAD substrates in electron transport chain complexes (ETC) by mass spectrometry. Here, we found that Sdh1, a flavoprotein subunit of succinate dehydrogenase in complex II, is a MAD substrate. Specifically, inhibition of MAD by deletion of *DOA1* resulted in increased steady-state and ubiquitinated Sdh1 in mitochondria, and reduced interaction with the segregase Cdc48. Furthermore, inhibition of MAD resulted in reduced ETC complex assembly and reduced complex II activity, mitochondrial membrane potential and oxygen consumption rates. These data support the idea that MAD-mediated turnover of Sdh1 is critical for ETC complex assembly and activity, and mitochondrial quality. Since Sdh1 is a subunit in the complex II, Sdh1 must be retrotranslocated from mitochondria and recognized and bound by the cytosolic Cdc48 segregase. Thus, we tested whether the removal of Sdh1 is through the contact sites of mitochondrial outer and inner membranes. Indeed, we found that deletion of Mic60, a subunit of mitochondrial contact site and cristae organizing system (MICOS), results in increased steady-state and ubiquitinated Sdh1. Importantly, deletion both *DOA1* and *MIC60* did not further increase the steay-state and ubiquitintated levels, suggesting that the contact sites are essential for the degradation of Sdh1 in MAD. Our studies provide a potential mechanism of how MAD regulates mitochondrial respiration: it maintains mitochondrial respiration by removing damaged Sdh1 from the mitochondrial membrane contact sites to prevent defects of ETC complex assembly and activity.

Poster 20

Characteristics of Fluconazole Resistance Mechanisms in *Candida tropicalis* Isolates from Taiwan

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Fungal infections cause approximately 3.8 million deaths annually, with mortality rates more than tuberculosis. In Taiwan, yeasts are a leading cause of ICU-acquired infections, with *Candida tropicalis* being particularly prevalent. Taiwan Surveillance of Antimicrobial Resistance of Yeasts (TSARY) data reveal a rising trend in fluconazole-resistant *C. tropicalis*, reaching 10.8% in 2022 from 8.5% in 2014. These resistant strains, characterized by circulating highly resistant genotypes and azole cross-resistance, frequently cause treatment failure and complications, thus complicating clinical management. While resistance has been largely associated with clade 4 and the major mechanism is overexpressing mutated *ERG11*, encoding the target of azole drug, by copy number variation. In this study, we analyzed 24 clinical and environmental isolates to investigate molecular mechanisms contributing to azole resistance of non-clade 4 strains. Isolate YM221350 carried *ERG11* Y132F and S154F mutations, showed elevated gene expression, and exhibited a 1 plus 4 copy number of *ERG11*. Isolate YFA183118 harbored a homozygous K143R mutation with strong *ERG11* and *CDR1* upregulation, with fluconazole minimum inhibitory concentration (MIC) 32 μg/mL. In contrast, another isolate, YFA181245, with the same K143R mutation did not show increased gene expression and with fluconazole MIC was 32 μg/mL. Additionally, nine resistant or susceptible-dose dependent isolates from clade 1 showed *CDR1* overexpression and a nonsynonymous *TAC1* N939D mutation. This residue corresponds to N977D in *Candida albicans*, a confirmed gain-of-function mutation associated with *CDR1* upregulation. Two resistant isolates and six trailing growth isolates lacked identifiable resistance mechanisms, indicating that further investigation is needed. These findings suggest that fluconazole resistance in *C. tropicalis* in Taiwan is not limited to the clade 4 genotype and that other genetic lineages can acquire resistance through distinct molecular mechanisms.

Poster 21

The Molecular Mechanism of Arl3-Mediated Arl1 Activation

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Vesicular trafficking maintains cellular homeostasis and signaling by regulating proper protein transport. *ADP ribosylation factor* proteins (Arf) are conserved small GTPases in eukaryotes responsible for membrane trafficking and cargo transport. We have identified two yeast Arf-like proteins (Arls), Arl1 and Arl3, serve as critical regulators of vesicle transport in both exocytotic and endocytic pathways. Arl3 and Arl1 are essential for cell wall integrity, endosomal transport, and the unfolded protein response (UPR) under stress conditions. Arl1, like other small GTPases, transitions between an inactive GDP-bound form and an active GTP-bound form, regulated by the guanine nucleotide-exchange factors (GEFs) such as Syt1 and the GTPase-activating proteins (GAPs) like Gcs1. Importantly, Arl3 modulates GEF activity to regulate Arl1 functions and associated cellular processes. However, the mechanism by which Arl3 regulates Arl1 GEFs to modulate Arl1 signaling pathways remains unclear. In this study, we report that Arl3 acts as a supreme modulator of Arl1 activation. We first demonstrated that Arl3 interacts with Arl1 in both in vitro and in vivo settings. Additionally, we found that Arl3 associates with Syt1 and Arl1 in vivo, with Arl1 deletion impairing Arl3-Syt1 association. Furthermore, the deletion of Arl3 impairs the association between Syt1 and $Arl1^{T32N}$. Thus, we propose that the Arl3-Arl1 interaction enhances the GEF activity of Syt1 to promote the activation of Arl1.

Poster 22

Mixed Yeast Infections in TSARYs 2002-2022

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Clinically significant yeast isolates were collected via Taiwan Surveillance of Antimicrobial Resistance of Yeasts (TSARY) and mixed infections were investigated among isolates collected from 2002 to 2022. Among 321 out of 6912 specimens containing multiple species, 109, 94, 31, 30, 12, 9, 8, 7, 7 and 14 were from urine, sputum, ascites, blood, pus, tip, wound, BAL, bile and 8 others, respectively. The predominant combinations of mixed infection were 113 *Candida albicans*/ *Nakaseomyces glabratus* (*Candida glabrata*), 60 *N. glabratus* / *Candida tropicalis* and 51 *C. albicans/ C. tropicalis*. The prevalence of mixed infections was similar each survey. There were 63/911 (6.9%), 47/984 (4.8%), 46/1056 (4.4%), 43/1095 (3.9%), 47/1245 (3.8%), 75/1270 (5.9%) in 2002, 2006, 2010, 2014, 2018, and 2022, respectively mixed infections detected. Among the 321 mixed infections, 145 cases were originally identified in the providing hospitals, the remaining 172 ones were identified in the core laboratory at National Health Research Institutes. The great news is that the proportion of mixed infections detected by hospitals is significantly increased in the last decade (52/156 vs. 93/165, $P = 0.00003$), especially in 2022 (47/75, 62.7%). The ability to accurately identify species with different drug sensitivities in the same specimen is crucial for treatment since different species may have different susceptibilities to antifungal drugs. A total of 39 of the 65 mixed cases from sterile sites were detected by providing hospitals. It is worthy to note that hospitals failed to detect one third of cases (26/65). Hence, applying CHROMagar *Candida* medium to culture yeast isolates directly from the specimens, especially from sterile sites, is highly recommended.

Poster 23

The Distribution of Clinically Isolated *Candida* Species from Sterile Sites in Taiwan (2002–2022)

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Since 1999, we have conducted the Taiwan Surveillance of Antimicrobial Resistance of Yeasts (TSARY) every four years to monitor trends in the distribution of yeast species and their susceptibility to commonly used antifungal agents. Each participating hospital is requested to submit all yeast isolates from sterile body sites, along with up to 10 *Candida albicans* and 40 clinically significant non-*Candida albicans* yeast isolates from non-sterile sites. In this study, we focused on isolates collected from sterile body sites, including blood, ascites, cerebrospinal fluid (CSF), synovial fluid, pleural fluid, aqueous/vitreous fluid, and bone. Notably, *Candida auris*, a species recognized for its multidrug resistance, was not detected in any TSARY collections. Among the 1,604 *Candida* isolates collected in TSARY from 2002 to 2022, we observed a significant decrease in the proportion of *Candida albicans* isolates, which declined from 41.4% (2002–2010) to 33.9% (2014–2022) ($p \leq 0.001$). In contrast, there was a notable increase in non-*Candida albicans* species isolated from sterile sites during the same period, specifically *Nakaseomyces glabratus* (*Candida glabrata*) (from 12.4% to 18.4%, $p \leq 0.001$) and *Candida parapsilosis* (from 8.7% to 11.5%, $p \leq 0.005$). Regarding antifungal susceptibility, a total of 42 fluconazole-resistant isolates were identified between 2002 and 2022, with 17 of these detected in 2022 alone. Notably, fluconazole resistance among *Candida tropicalis* increased significantly from 2.29% in 2018 to 4.31% in 2022 ($p \leq 0.001$). These findings underscore the significance of TSARY as a valuable source of epidemiological data on yeast infections in Taiwan, enabling continued surveillance and informed clinical decision-making.

Poster 24

Surveillance of *Candida* Species and Antifungal Susceptibility in Taiwan: Insights from TSARY in 2022

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Candida species are major causes of fungal infections, with increasing prevalence of non-*albicans Candida* (NAC) and antifungal resistance posing clinical challenges. The Taiwan Surveillance of Antimicrobial Resistance of Yeasts (TSARY) monitors national trends in species distribution and drug susceptibility of pathogenic yeasts. In 2022, 1,302 *Candida* isolates were collected from 25 hospitals. Species identification was assessed by rDNA sequencing, and susceptibility to fluconazole, voriconazole, anidulafungin, and amphotericin B was determined following CLSI guidelines. In TSARY 2022, participating hospitals were asked to submit all isolates from sterile sites and the first 10 *Candida albicans* and 40 non-*C. albicans* isolates from non-sterile sites; therefore, the prevalence of *C. albicans* was likely underestimated. Even so, *C. albicans* remained the most common species (31.5%), followed by *Nakaseomyces glabratus* (*C. glabrata*) (28.2%) and *C. tropicalis* (25.5%). The proportion of *C. glabrata* isolates from sterile sites significantly increased over time, from 10.9% in TSARY 2002–2010 to 17.9% in TSARY 2014–2022 ($p < 0.0001$). A similar trend was observed for *C. tropicalis*, increasing from 16.8% to 22.0% over the same periods ($p = 0.008$). Importantly, a significant increase in fluconazole resistance among *C. tropicalis* isolates was also noted, increasing from 5.9% during TSARY 2002–2010 to 9.7% in TSARY 2014–2022 ($p = 0.004$). This trend highlights the growing challenge of azole resistance in *C. tropicalis* over the past decade. All 29 voriconazole resistant *C. tropicalis* were cross-resistant to fluconazole. Rare species—including *C. haemulonii* (n=6), *C. duobushaemulonii* (n=2), *C. pseudointermedia*, and *Kluyveromyces marxianus*—were detected, mostly with low MICs. One *C. haemulonii* isolate showed less fluconazole susceptibility with fluconazole MIC = 32 mg/L. These findings underscore the importance of continued surveillance, accurate species identification, and local susceptibility data to guide effective antifungal therapy.