目　錄

大會議程

日期	時間	主題/講題	講者	主持人
8/16	1100-1300	報到/午餐		
(日)	1300-1310	開幕致詞	李怡萱(陽明生理)	李青澔(北醫生理)
	1300-1330	開幕演講: 生科司發展現況與展望	陳鴻震 (科技部)	李怡萱
	1330-1510	以小雞的角度來分享整合型計畫的申請 把實驗室研究成果帶到業界: 轉譯經驗分享 綜合討論	楊尚訓 (成大生理) 何應瑞 (中山醫心理)	林赫 (中興生科)
	1510-1530	中場休息 (大合照)		
	1530-1620	大會特別演講: 研究生涯的桃花源記	華瑜 (高雄長庚轉譯)	蔡少正 (成大生理)
	1620-1800	科學研究與教學的火花 一個像廚師的大學老師 綜合討論	余佳慧 (台大生理) 林佑穗 (北醫生理)	阮琪昌 (陽明生理)
	1800-1900	晚餐交流		
	1900-2100	生理學研究與教學圓桌座談 (分四組進行)	謝坤叡* (慈濟生理) 盧主欽 (長庚生理) 吳莉玲 (陽明生理)	採分組進行
			林天南* (中研生醫) 許諄諄 (北醫呼吸) 黃菁英 (中興食生)	
			廖娟妙* (中山醫生理) 蔡克勵 (高醫生理)	
			劉哲育* (中國醫生醫) 李憶菁 (輔仁醫學)	
	2100-	夜宿惠蓀林場		
8/17	0700-0800	早餐		
(一)	0800-0900	林場生態探索活動		
	0900-1030	職涯規劃論壇 (大會議室) A1. 新進研究人員應徵博士後研究員與教職	李昆澤* (中山生科) 吳偉立 (成大生理)	採分組進行
		職涯規劃論壇 (會議室 1) A2. 新進研究人員應徵博士後研究員與教職	張原翊* (陽明生理) 黃炯瑋 (高醫生理)	
		職涯規劃論壇 B1. 新進教師建立研究室及升等 (會議室 2)	張雅雯* (成大生理) 彭賢祐 (馬偕醫學)	
		職涯規劃論壇 B2. 新進教師建立研究室及升等 (會議室 3)	林惠菁* (陽明生理) 賴亮全 (台大生理)	
	1030-1050	中場休息		
	1050-1130	形態與生理醫學研究推動展望	陳景宗 (長庚生醫)	謝博軒 (國防生理)
	1130-1200	中國生理學會 會員大會	李怡萱	李青澔
	1200-1210	閉幕致詞		
	1210-1300	午餐賦歸		

理事長歡迎詞

各位親愛的舊雨新知們，大家好：

歡迎大家來到惠蓀林場，參加2020年由科技部生科司形態及生理醫學學門及中國生理學會所共同舉辦的「生理醫學研習會暨科技部研究成果發表會」。學會歷年舉辦此研習活動，以促進學界交流及提攜新進，並廣獲好評。在今年，全球因新冠肺炎疫情而導致諸多國內外學術活動無法順利舉行之際，在台灣的我們有幸因著全民努力防疫得宜，而能持續舉辦，實彌足珍貴，也稍加彌補了今年初生醫年會因當時疫情不明而延辦之憾! 尤其今年報名出現超乎預期的踴躍且迅速額滿，更深感各位伙伴對於這場生理學界成長饗宴的熱切期待!

今年的研習會涵蓋教學研究、整合轉譯、學職涯發展三大主軸。其中以年輕學子及教師成長為主的論壇，在去年研習營第一次由年輕學生及博士後參與活動籌劃後，反應熱烈；再加上我們邀請了今年初經過近兩百篇摘要評選後入圍生醫年會口頭及壁報論文競賽的學生來發表壁報論文，使得今年參加研習營的年輕學生會員人數比去年多出將近一倍，展現了學生會員對學會的旺盛向心力，更是學會持續薪火相傳的最珍貴動能!

這次的會議中，除了如往年透過各場演講、研究發表與座談，使資深傑出學者和年輕潛力新秀互相分享經驗心得、共同激發對科學熱誠繼續築夢踏實之外，本學會身為國際生理學聯盟的會員，在國際上仍有許多國家的教研環境受到疫情嚴重影響之際，我們也將在此研習營中集思廣益，討論如何與各會員國學會攜手合作，共同因應未來國際學界因疫情或其它社會變遷所帶來的教學與研究困境與挑戰。

這次的活動，萬分感佩前理事長蔡少正教授和學門召集人陳景宗教授的鼎力支持，並感謝活動籌備主持人李昆澤教授為與會師生精心規劃各項活動、及籌備委員們的集思廣益與經驗傳承。非常感謝科技部生科司陳鴻震司長及國際生理學會聯盟主席華瑜教授的大力支持蒞會擔任大會演講，及所有生理學界此次來以演講或與談進行經驗分享的老師們。尤其感謝新任秘書長李青澔老師承擔重任、帶領本屆秘書處團隊為此活動大小細節安排及提昇效能的用心付出!

最後，預祝本次研習活動圓滿成功，所有與會嘉賓都收獲滿滿、健康快樂！並冀盼全球各國早日脫離疫情，全世界的老師和學生們能早日回歸平安快樂的教與學，國際學術交流的恢復能早日到來!

中國生理學會 理事長
李怡萱 敬上
中華民國一〇九年八月十六日

籌備委員會

李昆澤 (國立中山大學生物科學系)
林　赫 (國立中興大學生命科學系)
廖娟妙 (中山醫學大學生理學科)
何應瑞 (中山醫學大學應用心理系)
吳鈺琳 (國立陽明大學生理學研究所)
張原翊 (國立陽明大學生理學研究所)
李青澔 (臺北醫學大學生理學科)

工作幹部群

徐松柏 (臺北醫學大學生理學科)
林君樺 (康寧大學護理系)
黃美鳳 (國立成功大學生理學研究所)
王羿忻 (中山醫學大學生理學科)

學生籌備委員會

白宇辰 (國立台灣大學生理學研究所)
李婉寧 (國立成功大學生理學研究所)
周心喬 (高雄醫學大學醫學研究所)
陳嘉蕙 (國立陽明大學生理學研究所)
黃予庭 (長庚大學生物醫學研究所)

學生工作群

許筠、湯宜禎、鍾懿柔、吳慶龍 (成功大學生理學研究所)
張主龍 (中山醫學大學生理學科)
周心喬 (高雄醫學大學醫學研究所)
張皓程、劉大維 (長庚大學 生物醫學研究所)
辛予蕎、游博凱、廖堉臻、初昭暄 (國立陽明大學生理學研究所)
林妤家 (台北醫學大學醫學系)

講者及主持人介紹

李怡萱 教授

國立陽明大學 生理學科暨研究所

中國生理學會 理事長

專長學科	細胞及分子神經生理
學經歷	國立陽明大學生理學研究所教授 (2010-) 臺北醫學大學生理學科教授 (2003-2010) 臺北醫學大學生理學科主任 (2005-2010) 臺北醫學大學生理學科副教授 (1996-2003) 堪薩斯大學生理暨細胞生物系博士 (1993) 臺灣大學農業化學系學士 (1988)
獎勵事項	國立陽明大學特聘教授 (2017-2021) 國立陽明大學教師學術卓越獎勵 (2011-2021) 國立陽明大學醫學系優良教師 (2011-2020) 臺北醫學大學教學創新獎特優 (2008) 臺北醫學大學優良教師獎 (1996-2009)
研究方向及興趣	1. 神經膠細胞對神經傳導及神經血管功能之調節 (Neuroglia in regulation of neurotransmission and neurovascular functions) 2. 神經退化之基因與環境因子 (Gene-environment interaction in neurodegeneration) 3. 慢性病造成之腦功能失序(Chronic disease-associated brain disorders) 4. 優化人與動物連結於促進健康老化之生理機轉 (Optimization of human-animal bond for promoting healthy aging)

陳鴻震 講座教授

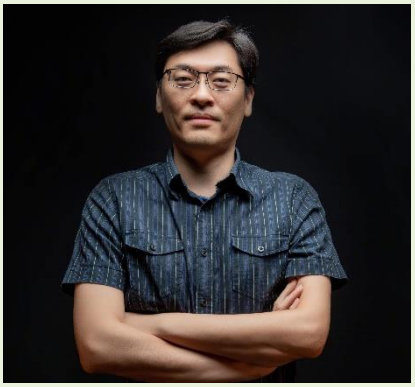
陽明大學生化暨分子生物研究所

科技部生命科學研究發展司司長

專長學科	生物化學、癌瘤學
學經歷	美國康乃爾大學病理博士 國立中興大學生命科學系教授 國立中興大學校友總會顧問 國立中興大學生物醫學研究所所長 國立中興大學生命科學院院長 台灣生物化學及分子生物學學會理事長 國際生物化學及分子生物學聯盟(IUBMB)台灣代表 中華民國細胞及分子生物學學會理事 台灣生物化學及分子生物學學會理事 台灣基因體暨遺傳學會理事 國立陽明大學生化暨分子生物研究所講座教授 國立陽明大學生命科學院院長
獎勵事項	科技部傑出研究獎(2015) 國際生物化學及分子生物學聯盟(IUBMB)年輕科學家論壇主席(2014) 國科會傑出研究獎(2003) 中央研究院年輕學者研究著作獎(2001)
研究方向及興趣	探討細胞癌化的機轉: (1) 癌細胞移動的機轉 (2) 中間絲骨架蛋白在細胞癌化過程中的角色

林 赫 教授

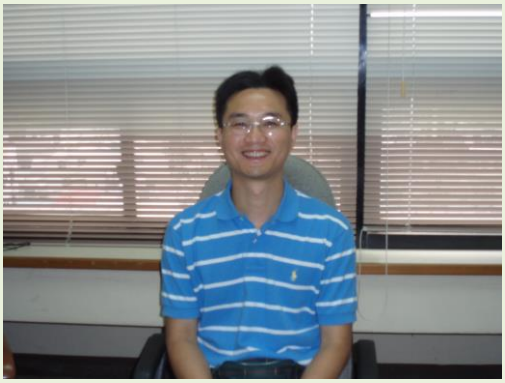
國立中興大學 生命科學系



專長學科	內分泌學、癌症生物學、神經生物學
學經歷	國立陽明大學 生理學研究所博士(2000) 國立中興大學生命科學系特聘教授兼系主任 (現職) 亞洲大學生物科技系 合聘教授 (2013~迄今) 美國德州大學西南醫學中心泌尿外科 訪問教授 (2012~2013)Chinese 國立中興大學生命科學系 助理教授 (2004~2009)、副教授 (2009~2014) 台灣調適科學會 理事 (2007~2009) 台灣內分泌及代謝學會 理事 (2014~迄今) 中國生理學會 理事 (2014~迄今) American Journal of Cancer Review 編輯 (2013~迄今) Evidence-based Complementary and Alternative Medicine 編輯 (2014~迄今) Adaptive Medicine 期刊 副主編 (2011~迄今) Journal of Physiology (SCI) 副主編 (2013~迄今)
獎勵事項	國立中興大學「優秀年輕學者獎助計畫-懷璧獎」(2015) 國立中興大學傑出青年教師 (2009、2011、2013) 國立中興大學產學績優獎 (2010) 國科會傑出學者養成計畫 (2007)
研究方向及興趣	1 各種腫瘤之細胞內訊息傳遞及藥物作用(包括攝護腺癌，乳癌，肺癌，子宮頸癌，膀胱癌，甲狀腺癌等) 2 內分泌之細胞內訊息傳遞 3 探討神經分化之分子機制 (包括神經退化性疾病) 4. 雄性激素生物合成機轉及受到藥物的影響與內分泌有關之癌症與神經退化性疾病之分子病因與診斷治療的研究

楊尚訓 教授

國立成功大學 生理學科暨研究所



專長學科	神經退化性疾病、神經科學、分子生物學、繁殖生理學、胚胎學
學經歷	美國艾默里大學遺傳與分子生物學程博士 成功大學生理學科暨研究所教授 (現職) 成功大學醫學院生理所 助理教授、副教授
獎勵事項	成杏優秀論文獎 (2014、2018、2019) 科技部未來科技突破獎 (2017) 科技部吳大猷先生紀念獎 (2016) 李國鼎研究獎 (2016) 第 10 屆永信李天德醫藥青年醫藥科技獎 (2015) 國立成功大學教學傑出教師 (2014)
研究方向及興趣	1. 利用新穎基因轉殖技術建立人類疾病動物模式 2. 探討 microRNA 於神經退化性疾病的影響，及其對神經保護的調控機制與應用於基因治療的可行性 3. 應用胚胎及幹細胞探討神經發育及保護機制 4. 利用基因轉殖小鼠探討 microRNA 的基因調控機制

何應瑞 教授

中山醫學大學 心理學系

專長學科	生理心理學
學經歷	台灣大學醫學院 生理學研究所博士 中山醫學大學心理系 教授 中山醫學大學 學務長 中國生理學會理事、秘書長 台中市政府 防毒委員 中山醫學大學教師會理事長 瑞金抗齡研究教育基金會 創會董事
獎勵事項	科技部特殊優秀人才獎 (2012~ 2020) 中山醫學大學教學優秀教師 台中市 SUPER 教師 Aristotle Research Award
研究方向及興趣	神經藥理學 神經退化性疾病 巴金森氏症 癲癇 失智症

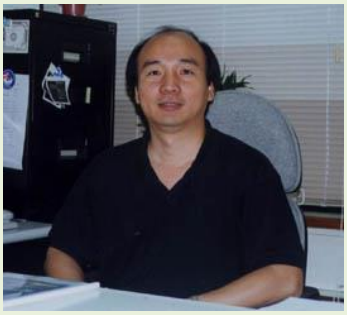
吳鈺琳 教授

國立陽明大學 生理學科暨研究所

專長學科	分子生物學、分子內分泌、生殖生理、腫瘤生物、發炎反應、訊息傳遞。
學經歷	國立陽明大學生理所教授（2017~） 國立陽明大學生理所副教授（2010~2017） 國立陽明大學生理所助理教授（2004~2010） 美國北卡羅萊那大學教堂崗校區博士後研究（2001~2004） 美國威斯康辛大學馬迪遜校區內分泌及生殖生理學博士（2001） 台灣大學醫學院微生物學研究所碩士（1992）
研究方向及興趣	本實驗室“細胞訊息傳遞實驗室”藉由細胞生物學，生化學，分子生物學及生理學的角度來探討內分泌系統及免疫系統功能（發炎反應）相關之分子調控機轉，及其在生理、病理或癌症進程中之角色。另外，我們也研究日常生活中所攝取之營養補品在生理上所扮演的角色及其在活體以及細胞內的標的分子與基因群等等。主要研究課題包含有: (1)分子內分泌之訊息傳遞在生殖系統之角色及其相關之生理、病理反應機制。(2) G protein-coupled receptors (GPCRs)在內分泌系統中分子作用機制。(3) 新穎抗發炎分子在生理病理狀態的角色定位及其對疾病的療效和作用機制。(4) 葡萄糖胺(Glucosamine)改善生理功能之角色及對疾病及腫瘤的療效。

蔡少正 教授

國立成功大學 生理學科暨研究所



專長學科	內分泌學、細胞及分子生物學、生物資訊學
學經歷	科技部生科司司長，2014-2017 加入成功大學教職，1998; 特聘教授，2008- 美國威斯康辛大學博士，1997 美國東密西根大學碩士，1992 成功大學學士，1986
獎勵事項	2020:美國生殖研究學會國際傑出科學家獎 2018:美國實驗生物及醫學學會會士(fellow) 2018:科技部傑出研究獎 2014:美國實驗生物及醫學學會傑出科學家獎 2012:國科會傑出研究獎 2005-2008:國科會優秀主持人獎 2003:成大醫學中心基礎醫學研究獎 1998:國科會研究獎(甲種)
研究方向及興趣	利用生物資訊學、細胞/分生的技術，從事與臨床疾病相關之生理及病理的探討 目前主要的研究方向有三個重點： 1. 探討子宮內膜異位(endometriosis)成因之分子機制：本實驗室過去十幾年的研究，在這個領域居於國際領先的地位，目前著重在整合性基因功能上的研究，希望能找到治療的方法。 2. 缺氧誘導因子(HIF-1α)基因調控網路與疾病之研究：我們利用生物資訊學、功能性基因體學、及系統生物學的方法，建構受缺氧誘導因子調控之基因網路，並研究其在生理、病理上的功能。 3. 癌細胞產生抗藥性機制之探討：癌症是目前人類的頭號殺手，癌症的發生常出於不可預期的因素。然而人類在癌症的治療上，雖窮盡一切之力，卻因為癌細胞產生抗藥性而功虧一簣。我們利用最先進的概念及方法，探討癌細胞產生抗藥性的機制，包括癌症幹細胞的產生、癌症表觀基因的修飾等。針對大腸直腸癌及胰臟癌做深入的研究，希望找出治療的契機。

華 瑜　講座教授

高雄長庚醫院

生物醫學轉譯研究所

專長學科	神經科學、訊息傳遞、心臟血管生理學、氧化壓力
學經歷	美國華盛頓州立大學神經科學研究所博士 美國華盛頓州立大學神經科學研究所博士後研究員 高雄長庚醫院生物醫學轉譯研究所 講座教授 高雄長庚醫院醫研部主任 第 20-21 屆中國生理學會理事長 亞太生理學聯盟(FAOPS)主席(2011~2015) 世界生理學會聯盟(IUPS)主席 (2017~2021) Journal of Biomedical Science 期刊副主編 (2005~迄今) Biomedicine Hub 期刊副主編 (2015~迄今) Frontiers in Physiology 期刊副主編 (2017~迄今) IBRO Reports 期刊副主編 (2015~迄今)
獎勵事項	科技部傑出獎(2004、2009、2012 共三次) 科技部神經科學專案計畫辦公室主持人 (2017~) 行政院退輔會傑出研究成果獎 (2010) 長庚醫療財團法人講座教授(2010~)
研究方向及興趣	中樞神經系統調節心臟血管功能生理與病理角色之研究，包括氧化壓力、粒線體與內質網功能、細胞自噬之角色；近期研究延伸上述各訊號探討親代相關機轉失調如何影響子代心臟血管功能之發育及異常，以及基因表觀遺傳學與代謝體學之參與。

阮琪昌 教授

國立陽明大學 生理學科暨研究所

專長學科	代謝生理學、分子內分泌學、脂肪細胞生理學
學經歷	國立陽明大學生理學博士（1998） 國立陽明大學生理學研究所所長 國立陽明大學醫學院醫學系 副系主任 (2009~2016) 國立陽明大學總務處 副總務長 (2016~2018) 國立陽明大學生理學研究所 教授 兼所長 (2008~2009) 國立陽明大學生理學研究所 副教授 (2005~2007) 國立陽明大學生理學研究所 助理教授 (2003~2005) 台北榮民總醫院教學研究部 博士後研究 (1998~2003)
研究方向及興趣	主要的研究興趣是（1）代謝症候群病理機轉的探討、（2）具血管活性因子對新陳代謝的影響、（3）重組蛋白質的表現及活性探討、（4）脂肪組織分泌之細胞因子在肥胖及胰島素抗拒症致病機轉中的角色探討、（5）藥物及保健食品的開發。實驗對象包括細胞、組織、小鼠以及大白鼠等。研究方法則涵蓋細胞生理學、內分泌學、生物化學以及分子生物學等相關技術的操作。

余佳慧 教授

國立台灣大學 生理學研究所

專長學科	腸胃生理，黏膜免疫
學經歷	Professor, Graduate Institute of Physiology, National Taiwan University College of Medicine, Taipei, Taiwan ROC Post-doctoral fellow, University of Calgary, Calgary, Alberta, Canada Ph.D., Department of Medical Sciences, McMaster University, Ontario, Canada M.Sc., Department of Biology, University of Waterloo, Ontario, Canada B.Sc., Department of Veterinary Medicine, National Taiwan University, Taipei, Taiwan
獎勵事項	2019 The 16th National Innovative Award 2016-21 Innovative Research Grant, National Health Research Institute (NHRI) 2013-16 Outstanding Young Investigator Project Grant, Ministry of Science and Technology 2011 Young Investigator Award, The 7th Federation of the Asian and Oceanian Physiological Society Congress (FAOPS) 2011 2009 Young Investigator Award, Asia Pacific Digestive Week (APDW) 2009
研究方向及興趣	Our research interest is focused on host-microbe interaction for regulation of epithelial barrier function and tumorigenesis in the gastrointestinal tract. The intestinal epithelium acts as the first line of defense against commensal bacteria and microbial pathogens. Epithelial dysfunction and microbiota dysbiosis are involved in the pathogenesis of various gastrointestinal diseases, including inflammatory bowel disease, irritable bowel syndrome, and colorectal cancers. Gene modified animal models, intestinal epithelial cell lines, primary organoids and spheroid cultures, as well as molecular techniques of site-directed mutagenesis, gene silencing and knockout are utilized in our laboratory to investigate the pathophysiological mechanisms of intestinal diseases. Current research projects: • Molecular mechanisms of regulation of epithelial tight junctions and bacterial transcytosis • Identification of specific intestinal bacteria with barrier-breaking and pro-tumorigenic ability • Cytoprotection and death resistance mechanisms by glucose on intestinal epithelium and colorectal carcinoma

林佑穗 教授

台北醫學大學 生理學科

專長學科	呼吸生理
學經歷	2000 陽明大學生理所博士 2009 迄今台北醫學大學生理學科副教授 2010 肯塔基大學醫學中心訪問學者 2002-2009 台北醫學大學生理學科助理教授 2000-2002 肯塔基大學醫學中心博士後研究員
獎勵事項	104 學年度台北醫學大學 教學傑出教師 100/102/104 學年度 台北醫學大學校級優良教師
研究方向及興趣	呼吸道與肺感覺神經過度敏感的形成機轉與藥物治療 簡報與教學技巧

謝坤叡 教授

慈濟大學 醫學系

專長學科	時間生物學、神經內分泌學、神經科學、胃腸生理學
學經歷	國立陽明大學生理學研究所博士 慈濟大學生理學科 教授 慈濟大學醫學系 副主任 慈濟大學醫學院 副院長 慈濟大學 學生事務長 考選部典試委員 行政院原子能委員會核能研究所諮詢顧問 中華民國基礎神經科學學會秘書長(2007-2009)
獎勵事項	日本生理學會 2012 年會之優秀學術壁報獎 日本生理學會第 83 屆年會之青年旅行獎 (2006) 國際腦研究組織旅行獎，IBRO 2003 王世濬院士青年旅行獎 (1999)
研究方向及興趣	探討生物時鐘 (biological clock)的日變節律 (circadian rhythm)現象，結合神經化學、神經內分泌學與生殖內分泌學的範疇，探討下視丘多巴胺神經元系統的日變週期，及其發育源由 (ontogeny)、神經間的交互作用、甚至到性別差異 (sexual difference)等獨特現象。進一步地由基因層面，並佐以動物行為觀測，我們發現哺乳類中從小鼠到人類相似性極高的生物時鐘相關基因 (circadian-clock genes)並非僅存於中樞神經系統，而是遍佈全身器官，透過相關獨特研究成果，希望能早日撥起日變節律現象的神秘面紗，更希望未來能進一步地提供有睡眠障礙。此外，個人的研究方向亦探討在中樞神經系統的飲食相關因子對飲食行為的調控，希望進而了解肥胖的成因；並探討現代文明病之一的精神壓力的形成原因的背後可能機轉，並探討抗精神藥物減壓的療效與評估。

林天南 研究員

中央研究院

生物醫學科學研究所

專長學科	腦中風的病理、生理機轉
學經歷	Since 1992 Assistant, Associate and Research Fellow, IBMS, AS 1990~1992 Post-doctoral Fellow, Baylor College of Medicine, TX 1985~1990 PhD in Biochemistry, UM-Columbia
獎勵事項	2015 Bronze medal Poster Award - The 25th ISN/13th APSN Satellite meeting, Singapore.1998 (4th) Shih-Chun Wang Memorial Young Investigator Award. 2010 5th TienTe Lee Biomedical Foundation - Outstanding Paper Award (Dr. Jui-Sheng Wu). 2002 The Robert KS Lim and Shih-Chun Wang Fund: Advanced Study Award. 2001 (3rd) Dr Shih-Bin Tu Memorial Excellent Paper Award. 2000 (1st) CVDPTF Excellent Paper Award.
研究方向及興趣	腦中風是造成死亡及成人殘疾的主因。 但除了血栓溶解劑，並無其他方法可侷限腦的損傷。 本實驗室的主要研究是要解開腦中風後神經傷害與功能復元的細胞與分子機制。 我們的研究顯示，利用 COX-1 基因轉殖可增加 PGI_2 (促進血管新生)與 PGD_2 (血管舒張)，進而刺激 PPAR-γ，降低發炎反應和抑制 Bad/ROS 所造成的細胞凋亡。 因此增強細胞的存活、並有效的降低梗塞體積。 所以，促進血管新生可作為是治療腦中風的方法之一。 其他研究方向還包含: 血管新生的機制及中樞神經的基因與細胞治療。

李憶菁 教授

輔仁大學 醫學系

專長學科	生理學、神經生理學、神經科學、眼科生理學
學經歷	英國倫敦大學 生理學 博士 輔仁大學研究發展處 副研發長 輔仁大學研究倫理中心 主任 輔仁大學人體研究倫理委員會 執行長 輔仁大學研發處研究管理中心 主任 輔仁大學醫學系 助理教授、副教授 英國劍橋大學藥理學 博士後研究員
獎勵事項	1. 獲科技部 103~105 年度優秀年輕學者研究計畫。 2. 獲 the 15th Annual Conference of the Forum for Ethical Review Committees in Asia and the Western Pacific Region (FERCAP) 的最佳海報論文獎 擔任美國眼科醫學會國際學術期刊 American Journal of Ophthalmology (SCI)、美國眼科與視覺學會國際學術期刊 Investigative Ophthalmology & Vision Science (SCI)、歐洲眼科與視覺醫學會國際學術期刊 Acta Ophthalmologica (SCI)、分子視覺國際學術期刊 Molecular Vision (SCI) 論文審查委員。
研究方向及興趣	視網膜生理學 幹細胞治療視網膜疾病之應用 視網膜退化性疾病之致病機制與治療 黃斑部病變疾病模式 糖尿病視網膜病變疾病模式 視網膜影像學 痛覺神經之調控機制

盧主欽 助理教授

長庚大學 生理暨藥理學科

專長學科	內分泌學
學經歷	2009~present Assistant Professor, Department of Physiology and Pharmacology, College of Medicine, Chang Gung University 2007~2009 Postdoctoral Fellow, Institute of Molecular Biology, Academia Sinica, Taipei, Taiwan 2003~2007 Postdoctoral Fellow, Division of Endocrinology and Metabolism, Department of Medicine, University of California, San Diego, California, USA 2002~2003 Research Associate, Department of Comparative Biosciences, University of Wisconsin- Madison, Wisconsin, USA Ph.D.: 2002, Endocrinology-Reproductive Physiology Program, University of Wisconsin-Madison, Wisconsin B.S.: 1992, Department of Plant Pathology and Entomology (Division of Plant Pathology), National Taiwan University
研究方向及興趣	肥胖為開發國家的一個重要健康問題。肥胖所引起的相關疾病，例如胰島素阻抗、糖尿病及心血管病變等，早已成為現代社會的醫療負擔。談到肥胖，脂肪細胞扮演重要的角色，因為肥胖即是脂肪細胞堆積脂肪而增大的結果；而脂肪細胞的增大與其功能異常有莫大的關聯。脂肪細胞的功能受到荷爾蒙、神經系統及飲食的調控，例如胰島素可促進脂肪細胞之脂肪合成、抑制脂肪分解、並促進血糖的吸收。在過去，脂肪細胞僅僅被認為只是儲存過多的能量之儲藏室。然而近年的研究結果發現，其實脂肪細胞分泌許多重要的物質，包括蛋白質與脂質因子，能夠主動調控身體其他組織或器官的生理功能。身體沒有脂肪細胞，過多的脂肪將被儲存在其他器官如肌肉與肝臟，造成這些器官的病變；同時缺乏脂肪細胞所分泌的重要物質，亦造成其他器官的功能異常。因此了解脂肪細胞的生理功能及其調控，將可幫助我們了解其與新陳代謝疾病的關係，並提供治療疾病之方法。

吳莉玲 助理教授

國立陽明大學 生理學科暨研究所

專長學科	消化道生理學、感染免疫學、分子病毒學、微生物學
學經歷	國立臺灣大學臨床醫學研究所博士後研究員(Sep,2014-Jul,2019) 國立臺灣大學生理學科暨研究所助教(Jan,2002-Feb,2008) 康寧護專兼任講師(Jan,2002-Feb,2004) 台北榮民總醫院兼任及專任研究助理(Jan,1998-Dec,1999) 國立台灣大學生理學科暨研究所博士(2008-2014)
獎勵事項	國際 B 型肝炎會議研討會『傑出海報獎』第一名，義大利，2018。國際 B 型肝炎會議研討會『傑出海報獎』第一名，美國，2017。 國立台灣大學『優秀研究生優秀著作獎』佳作，臺灣，2015。 第三十屆全國生物醫學聯合學術年會『口頭論文競賽』傑出論文獎，臺灣，2015。 第三十屆全國生物醫學聯合學術年會『口頭論文競賽』第一名，臺灣，2015。 第二十九屆全國生物醫學聯合學術年會『口頭論文競賽』第一名，臺灣，2014。
研究方向及興趣	本實驗室研究領域是以腸胃道生理學為主，目前研究計畫主要探討腸道菌透過腸-肝軸如何影響 B 型肝炎病毒持續性或清除感染之預後，主要目的是釐清其於免疫系統中 innate myeloid cell 和 lymphoid cell 所扮演的角色，以及腸道屏障功能的完整性，以期望了解腸道菌及其代謝產物如何影響肝臟免疫反應的完整訊息及機制，藉由此研究將有助於開發預防和治療先天免疫細胞相關肝病新穎和創新的策略，以期望未來可藉由細胞免疫療法來重新改編宿主的抗病毒免疫力，進而達成全面清除 B 型肝炎病毒，對未來 B 型肝炎的預防與治療提供新契機及新發現。

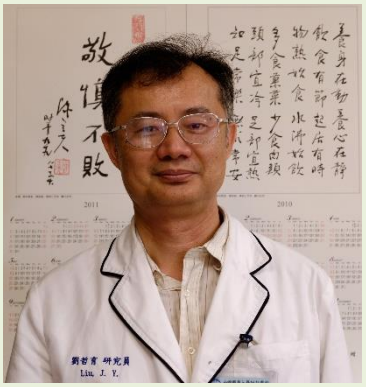
廖娟妙 副教授

中山醫學大學 醫學系生理學科

專長學科	生理學、生命科學
學經歷	中興大學生命科學所博士 現任中山醫學大學醫學系生理學科主任 第 26 屆中國生理學會理事 第 24-25 屆中國生理學會秘書長 中山醫學大學助教、貴儀室技士、講師、助理教授、副教授
研究方向及興趣	1. 利用電針刺激方式及系統電氣生理方法研究糖尿病鼠之膀胱平滑肌收縮功能。 2. 利用電針刺激方式及系統電氣生理方法研究基因肥胖大鼠之下泌尿道功能。 3. 以分子生物技術方法探討基因肥胖大鼠膀胱平滑肌收縮功能。 4. 糖尿病鼠遭受急性缺血性中風傷害的神經保護作用之探討。 5. 調控腸道微生物對牙周致病菌 P. gingivalis 惡化缺血性中風傷害之影響。

劉哲育 教授

中國醫藥大學 生物醫學研究所



專長學科	癌瘤生化學、分子內分泌學 Cancer Biology; Endocrine; Traditional Chinese Medicine
學經歷	中國醫藥大學 醫學院癌症生物學研究所 教授(980201~) 中山醫學大學 生物化學研究所 副教授(820801~ 900731) 中山醫學大學 生化暨生物科技研究所 副教授(900801~ 930630) 中山醫學大學 生化暨生物科技研究所 教授(930701~ 980131) 台灣大學 生化科學 博士 陽明大學 生理所 碩士
研究方向及興趣	專長是研究兩個轉錄基因 (MZF-1/ELK-1) 互相結合與癌症惡化的機制研究，目前正在申請抗癌藥物篩選專利。 研究中草藥七層塔水萃取物的功效，尤其對於減肥美白、降低膽固醇、抗癌和降低抗癌藥物毒性等功效的研究，目前已有市售產品 (名稱為七層塔茶)。

蔡克勵 副教授

高雄醫學大學 醫學系生理學科

專長學科	細胞生理學、細胞內離子運輸與調控、呼吸生理學
學經歷	高雄醫學大學生理學科 助理教授 臺灣大學醫學院 博士後研究員 臺灣大學醫學系生理學科 助教 英國牛津大學生理學系 哲學博士 臺灣大學醫學院生理學研究所 碩士
研究方向及興趣	1. 陰離子運輸機轉參與神經元分化之角色。 2. 細胞磷脂酶對於細胞增殖與分化之效應。 3. 胞器參與細胞程式性死亡之機制。

許諄諄 助理教授

臺北醫學大學 呼吸治療學系

專長學科	呼吸生理、呼吸神經電生理學、呼吸治療
學經歷	2016-present 臺北醫學大學 呼吸治療學系 助理教授 2013-2016 美國 University of Kentucky Medical Center Post-doctoral Researcher 2008-2013 臺北醫學大學 醫學院醫學科學研究所 博士 2010-2011 美國 University of Kentucky Medical Center Visiting Scholar 2003-2007 臺北醫學大學 呼吸治療學系 學士
獎勵事項	106 學年度校級新進教學優良教師獎
研究方向及興趣	肺迷走感覺神經於發炎引發呼吸道過度敏感中的角色

黃菁英 助理教授

國立中興大學
食品暨應用生物科技學系

專長學科	腸胃道生理學、腸黏膜免疫、營養學
學經歷	2016/08~2017/07 國立陽明大學生化暨分子生物研究所-博士後研究員 2016/02~2017/02 銘傳大學生物醫學工程學系-兼任助理教授 2013/08~2015/07 國立台灣大學醫學院生理學研究所-博士後研究員究員 2006/09~2013/06 國立台灣大學醫學院 生理學研究所(博士) 2002/09~2004/06 國立台灣大學醫學院 生理學研究所(碩士) 2002/09~2004/06 台北醫學大學保健營養學系
獎勵事項	科技部補助大專院校延攬特殊優秀人才 2018 Japan Digestive Disease Week, Travel Award speaker (Kobe, Japan)
研究方向及興趣	單一營養素或飲食型態組成在生理與病理狀態中於腸道之微觀與巨觀之角色。

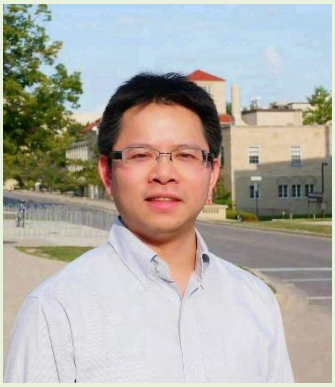
李昆澤 教授

國立中山大學生物科學系

專長學科	呼吸神經生理學、脊髓損傷醫學、神經科學
學經歷	國立中山大學生物科學系教授 國立臺灣師範大學博士
獎勵事項	國立中山大學學術研究績優(2015-2020) 國立中山大學特聘年輕學者(2016-2017) 亞太神經化學學會年輕學者獎(2016) 亞太生理學會年輕學者獎(2011)
研究方向及興趣	頸部脊髓損傷對於呼吸神經管制之研究。 間歇性低氧促進頸椎損傷後呼吸功能恢復之研究。 非侵入式磁刺激調控頸椎損傷後呼吸功能之研究。

張原翊 助理教授

國立陽明大學 生理學科暨研究所



專長學科	表觀遺傳學、造血與血癌幹細胞分子生理
學經歷	國立陽明大學醫學院生理學科暨研究所助理教授 (2014-迄今) 美國威斯康辛大學麥迪遜分校 McArdle Laboratory for Cancer Research 博士後研究 (2011~2014) 國立陽明大學生物藥學所碩士(1998)、博士 (2010)
獎勵事項	指導博士生林長億榮獲亞太生理學會 Young Scientist Travel Awards (2019) 指導碩士生陳筱雯榮獲第 33 屆生醫年會中國生理學會海報論文競賽佳作(2018) 中華民國血液病學會青年優秀論文獎首獎 (2016) 財團法人沈力揚教授醫學教育講學紀念基金會研究與進修獎勵 (2015) American Association for Cancer Research Scholar-in Training Award (2014) 榮獲國立陽明大學優良教師共九次
研究方向及興趣	終其一生，造血系統持續產生各種血球與免疫細胞，運送氧氣，參與凝血，調節體內免疫功能等等，藉以參與在各種病生理過程中。表觀遺傳學 (Epigenetics) 為在不改變 DNA 序列情況下，調控基因表現的機制，進而影響細胞生理、個體發育與疾病發展 (例如癌症生成)。其機制包括 DNA 甲基化 (DNA methylation)、組蛋白修飾 (histone modifications)、非編碼 RNA (non-coding RNA) 調控。基於 DNA 甲基轉移酶 DNMT3A 為血癌中常見的基因突變，過去我們的研究揭露 DNMT3A 劑量效應與致癌基因 RAS 誘導的不同型態血液腫瘤生成之關連。目前，本實驗室有兩大研究主軸: 第一、我們依然專注於 DNMT 家族對血球分化與血癌的形成或轉型之影響研究。尤其對分化過程，DNMT 與細胞能量代謝變化之關連感到興趣。第二，我們也探討在代謝症候群中，DNMT 與 RANTES-CCR5 訊息路徑兩者交互作用，對具免疫調節能力之 myeloid derived suppressor cell (MDSC) 形成之影響，及其對胰島素作用器官之發炎作用影響。同時我們也嘗試發展 MDSC 為基礎之細胞治療，用於代謝症候群或移植物對抗宿主疾病 (Graft-versus-host disease，GvHD) 治療。

吳偉立 助理教授

國立成功大學
生理學科暨研究所

專長學科	神經精神疾病、 腸腦軸研究、神經免疫學、腸道微生物與宿主交互關係、行為生理學
學經歷	成大生理所 助理教授 2018-迄今 加州理工學院 訪問學者 2018-迄今 加州理工學院 博士後學者 2012-2018 中研院 博士後研究 2011-2012 國防醫學院 生命科學所博士 2005-2011 中山大學 碩士 2001-2003
獎勵事項	最佳壁報論文 National Institutes of Health (NIH) Silvio O. Conte Centers for Basic and Translational Mental Health Retreat, Denver, CO, USA (2012)
研究方向及興趣	1.探討腸道微生物對自閉症的影響 2.探討腸道微生物代謝物對壓力的影響 3.利用化學遺傳學探討腸-腦軸的關聯

黃炯瑋 助理教授

高雄醫學大學 醫學系生理學科

專長學科	神經生理學
學經歷	高雄醫學大學醫學系生理學科助理教授 國立台灣大學醫學院生理學研究所博士後研究員(2014/02~2017/07) 國立台灣大學醫學院生理學研究所哲學博士(2005/08~2013/01) 高雄醫學大學醫學研究所基礎醫學組理學碩士(2000/08~2002/06)
獎勵事項	(1). 中華民國科技部 105 年度博士後研究人員學術著作獎 (Jun 2016) (2). 國立台灣大學醫學院研究生優秀著作獎; 博士組優等獎 (Mar 2016). (3). 國立台灣大學醫學院研究生優秀著作獎; 博士組佳作著作獎 (Mar 2015). (4). 中華民國第 30 屆生物醫學年會暨中國生理學會口頭論文競賽; 新科博士組口頭論文競賽第三名 (Apr 2015). (5). 中華民國第 30 屆生物醫學年會; 大會優秀論文獎 (Apr 2015). (6). 國立台灣大學醫學院研究生優秀著作獎; 博士組佳作著作獎 (Mar 2014). (7). 中華民國第 29 屆生物醫學年會暨中國生理學會口頭論文競賽; 新科博士組口頭論文競賽佳作獎 (Mar, 2014). (8). 財團法人行天宮資優生獎學金; 丙組:生物醫學組 (Nov, 2005- Jun, 2010).
研究方向及興趣	離子通道、細胞電生理學、突觸生理學、神經生理學

張雅雯 教授

國立成功大學

生理學科暨研究所

專長學科	藥理學、神經科學、毒理學、循環生理學、腦損傷
學經歷	國立成功大學生理所 教授，2014- 高雄醫學大學藥理所 合聘教授，2010-2015 國立中山大學生科系 合聘教授，2009- 長庚醫療財團法人生物醫學研究中心 教授級研究員兼任神經科學研究組主任，2009-2014 國立中山大學生科系暨神經科學研究中心 教授，2005-2009 國立中山大學生科系暨神經科學研究中心 副教授，2001-2005 國立中山大學生科系暨神經科學研究中心 助理教授，1998-2001 國立陽明大學 博士，1993-1998
獎勵事項	2009-2014：長庚醫療財團法人研究獎勵 1999-2001:國科會甲種獎 2008：國立中山大學資深優良教師 2007：國立中山大學研究績優獎 2006：台灣藥理學會傑出研究獎 2001：國立中山大學年輕學者獎 1999-2001:國科會甲種獎
研究方向及興趣	1. 研究有機磷中毒機制及解毒契機 2. 解析中風後誘發腦部運作與調控異常導致神經系統與心血管反應失能 3. 探究癲癇及其誘發之損傷機制 4. 論證成癮藥物中毒致死機制 5. 檢測神經細胞與膠細胞交互作用在神經疾病之重要角色 6. 科研表觀遺傳基因在食道癌致病機轉

賴亮全 教授

台大醫學院 生理學研究所

專長學科	基因體與細胞調控、癌症生物學、吸呼生理學
學經歷	國立臺灣大學生理學研究所 教授 國立臺灣大學基因體系統生物學學位學程 教授 中央研究院國際研究生學程生物資訊學程 教授 國立臺灣大學基因體暨精準醫學中心 生物資訊與生物統計核心實驗室 共同主持人 美國伊利諾大學香檳校區 分子與整合生理學研究所 博士 美國伊利諾大學香檳校區 分子與整合生理學研究所 碩士 國立臺灣大學生理學研究所 碩士
獎勵事項	年輕學者研究獎，第七屆亞太生理學聯盟學術大會 (Young Investigator Award, 7th Federation of the Asian and Oceanian Physiological Societies Congress, 2011)
研究方向及興趣	個人研究的興趣為利用基因體的技術，包括微陣列(microarray)及次世代定序(next generation sequencing)，來探討癌症產生的分子機轉，及尋找與診斷、預後或治療癌症相關的生物標記。為達此目的，個人的研究內容是於數種癌細胞（包括肺癌及乳癌）中，探討 DNA 結構的改變(例如：單核苷酸多型性(single nucleotide polymorphism; SNP)、拷貝數變異(copy number variation))，基因表現量的差異，及表觀遺傳(epigenetics)的調控(例如：DNA 甲基化(methylation)、微小 RNA(microRNA)及長片段非轉譯 RNA (long non-coding RNA) 等)。近年來特別著重於探討非轉譯 RNA (noncoding RNA)的調控機轉。

林惠菁 教授

國立陽明大學
生理學科暨研究所

專長學科	動物行為、電生理學、神經訊息傳遞
學經歷	國立陽明大學生理學科暨生理學研究所副教授(2016.8-2019.7) 國立陽明大學生理學科暨生理學研究所助理教授(2011.8-2016.7) 國立成功大學藥理所博士後研究員(2008-2011) 法國馬賽國家衛生暨醫學研究院(INSERM)訪問博士後研究員(2011) 美國德州貝勒醫學中心小兒研究部門訪問博士後研究員(2010) 國立成功大學基礎醫學所博士(2008) 國立成功大學藥理所碩士(2000)
獎勵事項	105 年度科技部傑出學者養成研究計畫 108 年度科技部優秀青年學者研究計畫 陽明大學教師學術卓越獎勵 第五屆永信李天德醫藥基金會傑出論文獎
研究方向及興趣	1. 自閉症是一種神經發育性的疾病，主要病徵為社交能力障礙與溝通能力的缺乏，且常出現反覆行為與過度焦慮反應。因此運用表現類似自閉症行為之齧齒類動物模型來探討自閉症相關大腦異常是一種具挑戰性但可能成果豐碩的方法。我們研究主要探討自閉症動物其大腦神經細胞突觸興奮性和抑制性間平衡改變的機制，並尋找藥物改善自閉症所導致之神經與行為異常。 2. 現今因社經環境壓力導致人類精神疾患人口日益增多。我們研究主題著重於探討精神疾患所引起大腦功能改變的因素,並尋找調控情緒反應的關鍵蛋白。研究策略結合神經藥物、分子生物學、動物行為模式和神經電生理技術，剖析精神創傷或生理性腦血管疾病造成憂鬱，焦慮情緒障礙表現的神經生理機制。

彭賢祐 教授

馬偕醫學院 醫學系

專長學科	感覺神經生理學、泌尿生理學、神經塑性、疼痛機轉
學經歷	馬偕醫學院醫學系教授 馬偕學院研發處研發長 馬偕醫學院教務處課務組組長 馬偕醫學院醫學系副教授 馬偕醫學院醫學系助理教授 中國醫藥大學醫學系生理學科助理教授 中國醫藥大學附設醫院泌尿部助理研究員 國立台灣大學附設醫院外科部博士後研究員 國立中興大學博士 中山醫學大學碩士
獎勵事項	108 年度科技部吳大猷先生紀念獎
研究方向及興趣	止痛與致痛機轉

謝博軒 教授

國防醫學院

生理及生物物理學研究所

專長學科	肥胖、代謝症候群、糖尿病
學經歷	美國凡登堡大學哲學博士 國防醫學院 生理及生物物理學研究所 教授 生理學科副教授 、生理學科教授兼主任、預防醫學研究所所長 國防生理學科助教、講師及助理教授、生物及解剖學科助理教授 中國生理學會常務理事 (105-109) 中國生理學會理事長(101- 105) 中國生理學會理事(96-101) 期刊主編 Journal of Medical Science (2011-2020) 期刊主編 Chinese Journal of Physiology (SCI, 2007-2020)
研究方向及興趣	1. The pathological links between inflammation and the development of metabolic syndrome and T2DM 2. The impact of chronic liver diseases on the development of T2DM 3. The relationship of COX2-mediated inflammation and the development of insulin resistance in diet-induced non-obesity and obesity-related insulin resistance 4. The role of chemokines and their receptors in the regulation of energy metabolism and the development of obesity-associated insulin resistance. 5. The novel mechanism underlying the control of adaptive thermogenesis

陳景宗 教授

長庚大學 生物醫學研究所

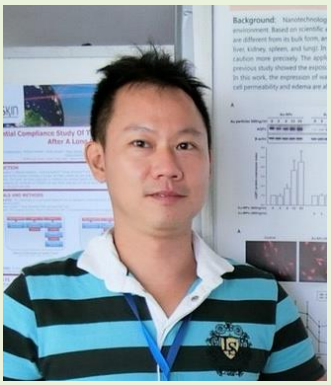
科技部生科司形態及生理醫學學門

召集人

專長學科	神經化學、神經藥理、老化醫學、藥物成癮、精神醫學、行為科學
學經歷	2008～迄今　長庚大學生物醫學研究所所長 2007～迄今　國立政治大學神經科學研究所兼任教授 2005～迄今　長庚大學生理暨藥理學科教授 2003～2008　長庚大學生理暨藥理學科主任 1992～1993　美國 QCB, Inc.研究員 1990～1992　美國麻州大學醫學院講師 1987～1990　美國波士頓大學醫學院博士後 1983～1987 美國伊利諾大學博士
研究方向及興趣	神經藥理研究室主要探討多巴胺酬償系統及神經胜肽(NPFF)在中樞神經系統上所扮演的角色，也探討若干中樞神經與精神病變，包括成癮與復犯機制（甲基安非他命及 K 他命）、精神分裂症、帕金森氏症、疼痛與憂鬱共病的中樞作用機轉。研究導向為利用藥物或基因轉殖鼠建立病變的動物模式，進行活體和離體的腦部神經化學、訊號傳遞與基因表現等的定性與定量分析，以神經藥理作驗證或以細胞培養模式探究其作用機制。在這些疾病研究中著重於數個神經傳導系統：包括多巴胺、興奮性 (glutamate) 和抑制性(GABA)氨基酸以及 HPA axis，希望經由多層次的整體性研究，確定特定神經系統在特殊病理時期的行為和生化反應，以利對疾病的瞭解或開發新型藥物。本實驗室開發先進的光遺傳學技術(optogenetics)，利用病毒 AAV5 所攜帶對光敏感的離子通道蛋白(ChR2 或 NpHR)感染具神經傳導物質專一性的 Cre mice 特定腦區，再利用植入的光纖維(optic fiber)激發或抑制神經活性，藉以在活體動物瞭解特定神經迴路對特定行為的影響。 本實驗室長期的合作夥伴包括:林口長庚醫院神經科學中心(NRC)、基隆長庚醫院、國衛院與德國的合作團隊。

李青澔 副教授

台北醫學大學 生理學科



專長學科	毒理學、環境毒理學、奈米毒理學
學經歷	2000-2005 國立台灣大學毒理學研究所博士 2006-2008 進階生物科技臨床前試驗中心副理 2009-2012 台大醫學院博士後研究員 2012-2017 台北醫學大學醫學系生理學科助理教授 2017-迄今 台北醫學大學醫學系生理學科副教授 2020.06-迄今 中國生理學會秘書長
研究方向及興趣	1. 奈米毒理學研究、管理政策與風險教育。奈米技術已廣泛應用於電子、醫療、食品、民生工業，影響層面也擴及民眾之消費行為、生態環境與健康。本實驗室針對奈米物質在體內的吸收分佈、及對內皮細胞障壁的穿透能力等主題進行系統性的研究。近期的研究發現經鼻吸入奈米顆粒會促進 aquaporin 1 表現並導致腦水腫，這可能是吸入空氣中細懸浮微粒導致腦病變的原因。 2. 多環芳香烴受體(AHR)之新穎功能。典型的 AHR 路徑係經由配體活化後進入細胞核作為轉錄因子並參與代謝活化。而我們近年來探討非典型的 AHR 功能，我們發現 AHR 在細胞質中具有泛素連接酶功能，並藉此調控多種蛋白質(包括: vimentin、BNIP-3 等)的表現與活性。我們利用基因剔除技術研究 AHR 缺陷型細胞的行為改變和重啟細胞多能特性的機轉。 3. 藍光對視網膜的損害。手持行動裝置的普及導致罹患眼腦疾病人口的比例快速攀升，眼睛有為數眾多且綿密的血管網絡，我們發現內皮細胞對藍光刺激特別敏感，我們正在研究神經血管單元使眼疾更趨惡化之機轉。

Poster 1

LncRNA *MALAT1* Inhibits miRNAs to Promote Cellular Functions of Breast Cancer.

Chung-Hsien Shih[1], Mong-Hsun Tsai[2], Eric Y. Chuang[3], Liang-Chuan Lai[1]
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[3]Graduate Institute of Biomedical Electronics and Bioinformatics, National Taiwan University

Abstract

Hypoxia, a common process during tumor growth, can lead to tumor aggressiveness and is tightly associated with poor prognosis. Long noncoding RNAs (lncRNAs) are ribonucleotide (>200 base) with limited ability to translate proteins, and lncRNAs are known to affect many aspects of cellular functions. One of the regulatory mechanisms is functioned as microRNA (miRNA) sponge to modulate the biological functions. Previously, we used next-generation sequencing (NGS) technology to identify oxygen-responsive lncRNAs in breast cancer MCF7 cells, and identified MALAT1 in the top five up-regulated lncRNAs under hypoxia. However, the regulatory mechanism and function of MALAT1 in breast cancer were still unclear. Therefore, the aim of this study is to explore whether MALAT1 can regulate the functions of breast cancer cells through miRNA. Endogenous expression levels of MALAT1 in MCF7 grown in different oxygen concentrations were examined by quantitative RT-PCR. Cellular proliferation and migration of breast cancer cells were examined by MTT assays and wound healing assays. To identify miRNAs affected by MALAT1, next-generation sequencing was performed in MALAT1-knockdown cells, and followed by RNA-binding protein immunoprecipitation (RIP) assays using antibody against AGO2 protein, as essential components of the miRNA-induced silencing complex, and by quantitative RT-PCR. Our data showed that expressions of MALAT1 were significantly upregulated under hypoxia. Overexpression of MALAT1 was able to promote migration and proliferation of breast cancer cells. Five differentially expressed miRNAs, including miR-3150a-3p, miR-4325, miR-378c, miR-3064-5p and miR-7855-5p, were identified using NGS and validated by qPCR when MALAT1 was knocked down under hypoxia. Lastly, the binding between MALAT1 and AGO2 enhanced in cells over-expressing MALAT1, indicating MALAT1 may function as miRNA sponge. These preliminary results suggest that MALAT1 might regulate cellular migration and proliferation of breast cancer cells via miRNAs. Therefore, MALAT1 may be a target candidate for inhibiting breast cancer progression.

Poster 2

Effects of mouse 5/6 nephrectomy on liver-infiltrating γδT cells and hepatic expression of lipid metabolism-, inflammation- and fibrosis-related genes

Yi-Ting Tu[1,2], Yi-Ju Wu[2], Wei-Yu Chen[1], Lung-Chih Lee[3,] Ben-Chung Cheng[3], Sheng-Ying Chung[4], Kuang-Tzu Huang[1,2], Ya-Wen Chiang[2], Li-Feng Bu[2], Yu-Fan Cheng[5], Chih-Chi Wang[2], Chao-Long Chen[1,2], Ding-Wei Chen[1,2,*]

涂怡婷[1,2]、巫奕儒[2]、陳威宇[1]、李隆志[3]、鄭本忠[3]、鍾昇穎[4]、黃廣慈[1,2]、蔣雅文[2]、卜莉鳳[2]、鄭汝汾[5]、王植熙[2]、陳肇隆[1,2]、陳定濰[1,2,*]

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高雄長庚紀念醫院[1]生物醫學轉譯研究所[2]外科部肝臟移植中心[3]內科部腎臟科[4]內科部心臟科[5]放射診斷科

Abstract

For the cause of adverse cardiovascular events resulting from end-stage renal disease (ESRD), uremic dyslipidemia is thought to be a major driving force indicating a critical role of hepatic disorders in ESRD. However, the pathogenesis has yet to be fully elucidated. Adenosine is a key extracellular signaling molecule that is involved in inflammation, immuno-modulation, and oxidative stress. Increased levels of CD73, the major enzyme of extracellular adenosine production is positively associated with induction of fibrosis in various organs. In this study, we examined whether liver-infiltrating CD73-expressing T lymphocytes and CD73 expression by hepatic cells were altered, and investigated hepatic transcriptional profiles associated with lipid metabolism, inflammation and fibrosis in a mouse model of ESRD. Mice with laboratory-induced renal failure by 5/6 nephrectomy were sacrificed at 8 weeks after the surgery. We found that in ESRD, an “unconventional” T cell subset bearing γδT cell receptors (γδTCR) exhibiting $CD73^+$, $CD25^+$ and $Foxp3^+$ phenotypes were drastically induced. Furthermore, expression of CD73 and a series of ectonucleotidases for purinergic signaling were significantly increased in liver cells. The expression of lipid metabolism–, inflammation–, and fibrosis-related genes were also skewed towards metabolic disorders. We will further examine the potential role of $CD73^+$ γδT cells in hepatic dysfunction in response to renal failure.

Poster 3

miR-524-5p suppresses the progression of the BRAF inhibitor-resistant melanoma

Mai-Huong Thi Nguyen[1a], Chen-Huan Lin[1a], Szu-Mam Liu[1a], Azusa Miyashita[2], Hironobu Ihn[2], Hsuan Lin[3,4], Chi Hou Ng[4,5], Jen-Chieh Tsai[1], Ming-Hong Chen[6], Mu-Shiun Tsai[7], In-Yu Lin[1], Shu-Chen Liu[1], Long-Yuan Li[8], Satoshi Fukushima[2*], Jean Lu[4,5,9,10,11*] and Nianhan Ma[1*]

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[a]These authors contributed equally to this work. [*]Corresponding authors

Abstract

BRAF inhibitors were approved for the treatment of BRAF-mutant melanoma. However, most patients acquire the resistance to BRAF inhibitors after several months of treatment. miR-524-5p is considered as a tumor suppressor in many cancers, including melanoma. In this study, we investigated the biological functions of miR-524-5p in melanoma with acquired resistance to BRAF inhibitor and evaluated the endogenous miR-524-5p expression as a biomarker for melanoma. The results showed that the expression of miR-524-5p was 0.481-fold lower in melanoma tissues (n = 117) than in nevus tissues (n = 40). Overexpression of miR-524-5p strongly suppressed proliferative, anchorage-independent growth, migratory and invasive abilities of BRAF inhibitor-resistant melanoma cells. Moreover, the introduction of miR-524-5p led to a reduced development of BRAF inhibitor-resistant melanoma in vivo. Remarkably, the MAPK/ERK signaling pathway was downregulated after treatment with miR-524-5p. Furthermore, next-generation sequencing analysis implied that the complement system, leukocyte extravasation, LXR/RXR activation and cAMP-mediated signaling may be related to miR-524-5p-induced pathways in the resistant cells. Most interestingly, the miR-524-5p level was higher on average in complete response and long-term partial response patients than in progressive disease and short-term partial response patients treated with BRAF inhibitors. Our results suggested that miR-524-5p could be a therapeutic target in BRAF inhibitor-resistant melanoma. In addition, miR-524-5p could serve as a prognostic marker in the response of patients to BRAF inhibitors for melanoma.

Keywords: Melanoma, miRNA, BRAF inhibitor, resistance, miR-524-5p

Poster 4

Elucidating the functional neural circuits of hypothalamic SF-1 neurons in exploratory social behaviors

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[Ψ]國立台灣大學生命科學院生命科學系

Abstract

Steroidogenic factor 1 (SF-1) expressing neurons in the ventromedial nucleus of the hypothalamus (VMH) are known for their essential roles in maintaining energy homeostasis and initiating innate behaviors that are essential for the individual survival. Artificial stimulation of the SF-1 neurons in the VMH elicits defensive behaviors towards conspecific competitors and triggers various anxious-like responses against predator cues; nevertheless, the underlying neural mechanisms remain elusive. In this study, we aimed to construct the input-output neural circuits of the hypothalamic SF-1 neurons and to establish the causal relationship between the SF-1 neurons and the defensive behaviors. To determine the targeting regions of the hypothalamic SF1-neurons, we conducted Cre-dependent anterograde viral tracing in the SF1-cre mice. We found that the SF1-neurons projected to multiple social-interaction-associated brain regions, including the periaqueductal gray, bed nucleus of stria terminalis and paraventricular nucleus etc. Such results indicated that the SF-1 neurons might engage in initiating or modulating innate behaviors that are associated with social interactions. Next, we performed *in vivo* calcium imaging to monitor the SF-1 neuronal activities under exposure to various social and non-social cues in free-roaming mice. We found that, while the conspecific social interactions robustly activated SF1 neurons, other non-conspecific stimulations, including predator-associated cues and unsociable objects, only modestly evoked SF-1 neuronal activities. Our detailed behavioral analysis further suggested that, among all the social behavior subtypes, the SF-1 neural activity strongly correlated with exploratory behaviors. In conclusion, our study has identified the VMH SF-1 neurons as a critical neural component in initiating social behaviors.

Poster 5

Cardioprotective effect of Fisetin on rats subjected to myocardial ischemia-reperfusion injury

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Abstract

Fisetin, a promising novel antioxidant, is a bioactive flavonol molecule found in fruits and vegetables. Several studies indicate that Fisetin can interact with diverse redox-related signaling pathways when transmitted through cellular membranes. However, cardioprotective effects of Fisetin remain unknown. In our study, the potential cardioprotective effects and the signaling pathways involved in Fisetin-inhibited expressions of inflammation mediators and Fisetin-induced antioxidant capacity were investigated in rats subjected to myocardial ischemia-reperfusion (I/R) injury. The left main coronary artery of anesthetized rats was subjected to 1 h occlusion and 3 h reperfusion. The animals were treated with/without Fisetin and the severities of I/R-induced arrhythmias and infarction were compared. Fisetin inhibited I/R-induced arrhythmias and reduced mortality, as well as decreased the lactate dehydrogenase, Troponin I and CPK levels in carotid blood. Fisetin increased the enhancement of expressions of endothelial nitric oxide synthase (eNOS) and Succinate Dehydrogenase (SDH). Our study so far confirms that Fisetin possesses the anti-arrhythmic effect and also has a valuable contribution of decreasing myocardial infarct size and plasma enzyme activity associated with cell damage in rats after myocardial I/R injury. In the future, we will explore the mechanism of cardioprotective effects of Fisetin and the signaling pathway associated with eNOS and SDH.

Poster 6

Identification of the Roles of Different Semaphorin 5A Domains in Lung Cancer Cells

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Abstract

Semaphorin proteins, a large family of membrane glycosylphosphatidylinositol-anchored, transmembrane, or secreted proteins, were initially identified as an axon guidance molecule in nervous systems. In recent years, several studies have revealed that semaphorins also involved in immune, cardiovascular, respiratory, musculoskeletal systems, and tumor progression. In our previous studies, we identified SEMA5A, a transmembrane protein, functioning as a tumor suppressor in lung adenocarcinoma, but the underlying mechanism was still unknown. Since different domains of transmembrane protein might have different functions in cell signaling pathways, here we investigated whether SEMA5A domains had different effects on cellular functions of lung cancer cells. Therefore, we constructed extracellular (5A-ECD) and cytosolic domains (5A-ICD) of SEMA5A and overexpressed them separately in A549 and H1299 cells. As compared to the wild-type control, the 5A-ECD-overexpressing groups significantly decreased cell proliferation, cell migration and reduced colony formation, but no significant difference in cell apoptosis was observed. In addition, functional analysis using Ingenuity Pathway Analysis (IPA) revealed that *SEMA5A* regulated genes were enriched in interferon signaling pathway. However, no significant expression levels changed among the interferon-related proteins by western blotting. Therefore, in order to systemically identify the downstream signaling mechanism of inhibiting tumor growth, co-immunoprecipitation and mass spectrometry to discover the interacted proteins are currently undergoing, and will be followed by western blotting for validation. Once we better understand the regulatory mechanism of SEMA5A, we hope to contribute in developing a more specific therapeutic regime to treat lung adenocarcinoma.

Poster 7

17β-Estradiol Increases the Bone Mass and Biomechanical Properties in a Mouse Model of Complete Androgen Insensitive Syndrome

Ya-Fen Liu[1, 2], Kuo-Chung Lan[1], Meng-Yin Tsai[1, 2], ChawnShang Chang[3], Ko-En Huang[1 *], and Hong-Yo Kang[1, 2 *]

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Introduction: Complete androgen insensitivity syndrome (CAIS) is the most common frequent causes of 46,XY female disorder of sex development and caused by X-linked androgen receptor (AR) mutations, which completely inactivate androgen-mediated sexual differentiation. Gonadectomy after puberty is generally recommended to avoid the risk of gonadal tumors but leads to reduce bone mineral density (BMD) in CAIS patients. Hormone replacement therapy (HRT) is required after gonadectomy to maintain secondary sexual characteristics, however, whether appropriate HRT is sufficient to maintain bone health remains unclear. Here we aimed to evaluate the effects of sex hormone treatments on bone microarchitectures in gonadectomized mice with inactivation of AR.

Methods: These global androgen receptor knockout (ARKO) mice were classified according to their sex gender, AR status, and types of surgery and sex hormone implants. Sex hormone supplements, including dihydrotestosterone (DHT), dehydroepiandrostendione (DHEA), or 17β-estradiol (E2), were given to gonadectomized control and ARKO mice at 18 weeks old and the effects were evaluated at 30 weeks old by microcomputed tomography.

Results: The results showed bone mass decreased in ARKO mice at 6, 18, and 30 weeks old. Gonadectomy did not further worse the bone microstructure in ARKO mice at 18 weeks of age. Bone strength and stiffness decreased in female and male ARKO mice. While none of the hormones significantly increased bone strength, E2 but not DHT or DHEA treatment rescued trabecular bone mass and increased bone stiffness in gonadectomized ARKO mice.

Conclusion: Given that the prevalence of osteoporosis and osteopenia was significantly higher in the CAIS patients, HRT was prescribed for 91% patients with CAIS after gonadectomy to increase BMD. Our ARKO mice can serve as a mouse model of CAIS to recapture the clinical observation of bone loss in CAIS patients. E2 supplementation can rescue the trabecular bone loss in mice with AR deficiency, reinforcing the importance of adequate hormonal treatment for women with CAIS.

Poster 8

LDLR-Mediated Lipidome-Transcriptome Reprogramming Impulses to Cisplatin Insensitivity

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Institutions:[1]Department of OBS/GYN, Sex Hormone Research Center, Research Center for Tumor Medicine, and Chinese Medicine Research and Development Center, China Medical University Hospital, Taichung, 40403, Taiwan. ; [2]Department of Medicine, Graduate Institute of Biomedical Sciences, and Graduate Institution of Cancer Biology, School of Medicine, China Medical University, Taichung, Taiwan 40403
Correspondence: Wen-Lung Ma, PhD., email: maverick@mail.cmu.edu.tw

Abstract

Platinum-based therapy remains the cornerstone for cancer patient management; however, its efficacy varies. This study demonstrates that expression of low-density lipoprotein receptor (LDLR) in subtypes of epithelial ovarian carcinoma (EOC) determines cisplatin sensitivity. It's sensitive in serous EOCs (low LDLR), where insensitive in endometrioid and clear cell EOCs (high LDLR). Meanwhile, knocked-down or overexpressed LDLR in EOC could reversed the chemosensitivity pattern both in vitro and in vivo. Mechanistic dissection with trans-omics (transcriptome and lipidome) analyses elucidated the LDLR-->LPC (Lyso-PhosphotidylCholine)-->FAM83B (phospholipase-related)-->FGFRs (cisplatin sensitivity and phospholipase-related) regulatory axis in cisplatin insensitivity. Implementing LPC-liposome encapsulated cisplatin could facilitate DNA-adduct formation via decreasing lipid droplets (LDs) deportation of drugs. Furthermore, Bioinformatics analyses found that the LDL/R-->LD homeostasis alteration is critical for therapeutic prognosis. Lastly, using LPC-liposome-cisplatin (LLC) drug improved sensitivities for gastric cancer, renal cell carcinoma, hepatocellular carcinoma, cholangiocarcinoma, and pancreatic adenocarcinoma cells. In conclusion, this report discovered a LDL/R-reprogrammed transcriptome-lipidome network, by which impulses platinum insensitivity and disease outcome. The drug specific lipidome for liposome manufacture might be an efficienct pharmaceutics strategy for chemoagents.

Keyword: LDLR, EOC, LPC, Cisplatin, Lipid Droplet

Poster 9

Endothelin (ET)-3 stimulated 3T3-L1 preadipocyte proliferation differently from ET-1 through the PKC/ERK-independent pathways

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[3] 聯新國際醫院腸胃肝膽科

Abstract

The endothelin (ET) family consists of three small (21-amino acid) peptides: ET-1, ET-2, and ET-3. ET-1 has been extensively reported to regulate the growth, development, adipogenesis, and metabolism of fat cells, while ET-3 has been reported to stimulate the growth of gastrointestinal epithelial cells in a region-specific manner and much less information to know its signaling pathways to mediate fat cell growth. This study investigated the pathways involved differently between ET-1 and ET-3 modulation of 3T3-L1 preadipocyte proliferation. We found that ET-1 and ET-3 were both able to stimulate preadipocyte growth, as indicated by increases in both cell number and BrdU incorporation. Further Western blot indicated that ET-1 stimulated phosphorylations of signal transducer and activator of transcription (STAT)-3, c-JUN, AMP-activated protein kinase (AMPK), protein kinase C alpha (PKCα/βII), and mitogen-activated protein kinase (MAPK) pathway proteins, ERK, but not JNK or p38. ET-3 did not alter phosphorylations of PKC and MAPK pathway proteins, but stimulated phosphorylations of STAT3, c-JUN, and AMPK proteins. Pretreatment with specific inhibitors of either Janus kinase 2 (JAK2)/STAT3, or JNK, or AMPK prevented ET-1-increased and ET3-increased levels of cell proliferation and phosphorylations of STAT, c-JUN, and AMPK, respectively. Pretreatment with specific inhibitors of either ERK or PKC inhibitors blocked ET-1-stimulated but not ET-3-stimulated cell growth, respectively. Interestingly, pretreatment with an ETAR inhibitor, such as BQ610, but not ETBR antagonist BQ788, blocked the ET-1-induced or ET-3-induced increases in both cell number and BrdU incorporation. BQ610 suppressed ET-1-induced increases in phosphorylated levels of STAT3, AMPK, PKC, ERK, and c-JUN proteins, and it blocked ET-3-increased STAT3, AMPK, and c-JUN, phosphorylations. These data suggest that ET-3 exhibits somewhat different signals from ET-1 to stimulate preadipocyte proliferation through modulations of ERK and PKC independent pathways. Results of this study may help explain how different endothelin isoforms mediate fat cell activity and fat cell-associated disease.

Poster 10

Protective effects of an active component of Hericium erinaceus on neuron-glia coupling in hypoxia ischemia-induced brain injury mouse model

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Abstract

Hypoxia-ischemia (HI) disrupted the brain function and is the leading cause of death worldwide. Reducing HI-induced brain injury and mortality has been the common interest in preventive medicine. Recent studies suggested that the Hericium erinaceus (HE) may exert neuroprotective effects in brain ischemia animal model; however, the cellular mechanism regarding whether HE affects the neuroglia-mediated support to neuronal survival in injured brain remained unclear. In this study, we investigated the effects of Erinacine A (EA), an active component of HE, and HE mycelia crude extract in transient HI (tHI) mouse model in vivo and oxygen-glucose deprivation (OGD)-challenged glia-neuron mix culture in vitro. Our results show that in the tHI mouse model, pre-treatments of EA or mycelia by daily oral gavage for 7 days did not affect the tHI-induced infarct volume and brain edema at 24 hr after tHI as evidenced by TTC staining. Notably, post-tHI daily intranasal delivery of EA in mice with pretreatments show significant improvement of the impaired grip strength accompanied with the reduction of infarct volume as evidenced by Nissl staining 72 h after the injury. Immunohistochemical staining of microtubule-associated protein 2 (MAP-2), a neuronal dendrite marker, further revealed that EA strengthened dendrite integrity of surviving neurons in the posterior cortex and dentate gyrus. Furthermore, glutamate transporter 1 (GLT-1)-labeled perineuronal astrocyte processes that indicate the glutamate uptake capacity show that EA attenuated tHI-induced astrocytic damage in the posterior cortex and dentate gyrus. In primary glia-neuron mix culture, EA also showed protective effect against OGD-induced neuronal and astrocytic damages. EA also decreases OGD-induced JNK phosphorylation, a signaling pathway critical for glial plasticity and cell death. Thus, these results suggest that the action mechanism of EA on ischemic brain injury may involve structural and functional protection of neuron-glia interaction.

Poster 11

Hypoxia-responsive circ*AAGAB* sponges *miR-378h* and reduces tumor malignancy of breast cancer

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Abstract

Breast cancer is the most common malignant tumor in women, accounting for almost one third of all malignant tumors. In the progress of tumors, insufficient blood supply to the tumor could result in hypoxic environment. Cancer cells needed to adjust the molecular mechanism under hypoxia in order to grow continuously. Recent reports indicated that circular RNAs (circRNA) affect the functions of cancer cells and regulate the development of carcinogenesis. However, the underlying biological functions of circular RNAs under hypoxia in breast cancer are still ambiguous. Therefore, the purpose of this study is to investigate the oxygen-responsive circular RNA *circAAGAB* and its effects on breast cancer. First, we used RNA-sequencing data to identify circular RNAs that were differentially expressed under different oxygen conditions, and validated these circRNA candidates by PCR. Among these oxygen responsive circRNAs, *circAAGAB* was chosen for further investigation. First, *circAAGAB* was up-regulated under hypoxia in five breast cancer cell lines, especially in MCF-7 and MDA-MB-231 cells. Therefore, we overexpressed *circAAGAB* in these two cell lines, and found that it significantly inhibited cell migration and invasion, and increased apoptosis, implying the tumor suppressor role of *circAAGAB*. Next, since circRNAs localize to their sites of action within the cell, the nuclear and cytoplasmic extraction method were used to explore its regulatory role. Results revealed that *circAAGAB* was mostly distributed in the cytoplasm, implicating the role of post-transcriptional regulation of *circAAGAB*. Also, an expanding evidence reveals that circRNAs can work with miRNAs to mediate gene expression via complex post-transcriptional mechanisms. We continued to explore whether *circAAGAB* could adsorb microRNA as a sponge. By using bioinformatics tools, quantitative RT-PCR and luciferase reporter assays, we found that *circAAGAB* could bind with miR-378h. Lastly, by using bioinformatics tools and RNA pull-down assays, we found that *circAAGAB* might directly interact with the RNA binding protein FUS. In the future, we will analyze whether the functions of cancer cells affected by *circAAGAB* are via the involvement of miR-378h or FUS protein.

Poster 12

Androgen Receptor in Periosteum-Derived Progenitor Cells Promotes Bone Fracture Repair in Mice

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Introduction: Periosteum is a specialized connective tissue that envelops bone surfaces and the sources of periosteum derived progenitor cells (PDCs) that contribute to bone development and fracture healing. While androgen receptor (AR) has been shown to lead to an increase in periosteal bone formation in male mice, the roles of AR in PDCs during fracture repair remain unclear.

Methods: We elucidated the actions of androgen/AR on the PDCs and evaluated its potential of exploiting androgenic bone building effects for fracture repair in mice.

Results: We first observed that AR was highly expressed and co-localized with the paired-related homeobox protein 1 (Prx-1; a mesenchymal progenitor cell marker) in the cells of periosteum during bone fracture repair. Deletion of AR gene in Prx1-cre expressing mesenchyme (Prx-1 ARKO) leads to significant impairments of callus volume and new bone volume in mice during fracture healing. Notably, the testosterone promote bone fracture repair only in wild-type mice, but not Prx-1 ARKO mice. Microarray expression profiling revealed that the mRNA levels of various type collagens and integrins were downregulated in PDCs from Prx-1 ARKO mice. *In vitro* gain/loss-of-function experiments demonstrated that AR promoted PDCs cell migration via collagen-integrin α2β1 activation. DHT/AR induced the phosphorylation of focal adhesion kinase (FAK) and the formation of FAK/AR/ARA55 complexes to promote the rearrangement of the actin cytoskeleton and subsequently regulate cell migration. *In vitro* overexpression of AR increased PDCs differentiation into osteogenesis and *in vivo* delivery of AR overexpressing PDCs accelerated bone fracture repair in mice.

Conclusion:

In conjunction with current therapeutic regimens, targeting activation of androgen/AR axis in periosteum may provide potential approaches to improve fracture-healing outcomes.

Poster 13

The intercellular communication involves in BRAF inhibitor -induced tumorigenesis

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Abstract

Vemurafenib (PLX4032) is a small molecule inhibitor of the V600E mutant form of BRAF gene (BRAFV600E) used in the treatment of melanoma. The treatment of BRAFV600E inhibitor in metastatic melanoma with BRAF mutation ensures the clinical improvement of the cancer. However, previous studies showed that 15-30% of the patients with BRAFV600E inhibitor treatment developed secondary benign or malignant skin tumors after an average of ten weeks from start of treatment. Cutaneous squamous cell carcinoma (cSCC) and keratoacanthomas (KA) were the majority of skin tumors presenting in the patients. The mechanism of BRAFV600E inhibitor-induced secondary tumor development is not well defined. Therefore, we asked whether the intercellular communication especially factors released from melanoma treated with BRAFV600E inhibitors affects epidermal cells to promote the formation of the secondary tumors. In this study, we utilized conditioned medium collected from melanoma cells treated with BRAFV600E inhibitor to investigate our hypothesis. The proliferation of epidermal cells treated with the conditioned medium was detected by AlamarBlue and colony formation assays. In addition, the signaling transduction pathways in the epidermal cells treated with the conditioned medium were studied by western blot. Our results found that the epidermal cells increased cell proliferation by the upregulation of MAPK/ERK signaling pathway. The detail mechanism will be dissected further.

Poster 14

In situ administration of enteral glucose protects remote gut against ischemia-induced barrier dysfunction through inflammation suppression

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Abstract

Intestinal ischemia/reperfusion (I/R) causes mucosal barrier damage and bacterial translocation (BT), leading to septic complications. Our recent studies have clarified the distinct cytoprotective role played by anaerobic glycolytic metabolites in the ischemia gut. Beyond the cytoprotective effects, we also found that enteral administration of glucose in the ischemia jejunal loop prevents bacterial translocation from the entire intestinal tract. The current study was aim to investigate whether the intestinal epithelial cell-derived anaerobic mediator plays a role in regulating the remote intestinal function via indirect contact with glucose in ischemic rats. Two 10 cm jejunal loops were created in rats, one was filled with Krebs buffer containing glucose (*in situ* loop), the other with Krebs buffer alone (remote loop). Rats then received sham operation of I/R challenge by occlusion of the superior mesenteric artery for 20 mins and reperfusion for 1 hr. Data showed that enteral glucose ameliorated I/R-induced gut permeability rise in the remote jejuna loop without direct glucose-enterocyte contacting as evidenced by magnetic resonance imaging (MRI)- and 4 kDa fluorescein isothiocyanate-conjugated dextran (FD4)- based gut permeability assay. Intestinal goblet cells were stained by Periodic acid Schiff (PAS), and the cell numbers per crypt or villus were quantified. Ischemic challenge caused the reduction of goblet cells in both *in situ* and remote loops. The number of goblet cells returned to the control level when enteral administration of glucose prior to intestinal I/R. Enteral glucose lowered the levels of mucosal myeloperoxidase (MPO) activity, macrophage inflammatory proteins (MIP)-1 and tumor necrosis factor (TNF) alpha production in the remote loop without direct interaction between glucose and remote mucosa. In conclusion, these results indicated that enteral administration of glucose in I/R gut may change the composition of epithelial cells, partly by altering the percentage of goblet cells. Furthermore, the protective effects mediated by enteral glucose are not limited in epithelial level but may act indirectly through suppression of inflammation via increments on goblet cells population.

Poster 15

Repression of COUP-TFII by proinflammatory cytokines contributes to endometriotic lymphangiogenesis

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Abstract

Endometriosis is a common gynecological disease that affects 8-10% women of reproductive age. It is characterized as the presence of endometriotic lesions outside the uterine cavity and often causes symptoms in patients such as pelvic pains, dyspareunia, and even infertility. Greater angiogenic and lymphangiogenic processes have been found in ectopic lesions. However, the underlying mechanism is still uncharacterized. In the present study, we found VEGF-C is highly secreted by endometriotic stromal cells. Elevation of VEGF-C in endometriotic stromal cells is mediated by derepression of chicken ovalbumin upstream promoter- transcription factor II (COUP-TFII) dependent transcriptional regulation. Further investigation reveals that level of COUP-TFII is suppressed by proinflammatory cytokines. Additionally, we also demonstrated that functional VEGF-C can be transported by exosomes, a 30-100 nm-size extracellular vesicles derived from the endosomal compartments, to enhance the tube formation abilities of lymphatic endothelial cells. Blockage of VEGF-C signaling by a highly selective inhibitor for VEGFR-2/3, Lenvatinib, abolished loss of COUP-TFII-mediated lymphangiogenesis in endometriosis both in *in vitro* and *in vivo* models. Herein, we have unveiled the novel mechanism of VEGF-C transportation by extracellular vesicles to communicate between endometriotic cells and lymphatic endothelial cells and demonstrate the regulatory status of VEGF-C involved in the pathophysiology of endometriosis.

Poster 16

The regulatory mechanism and function of hypoxia-induced long non-coding RNA *NDRG1-OT1* in triple-negative breast cancer cells

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Abstract

Breast cancer is the *most common* cancer in *women* worldwide. Triple-negative breast cancer (TNBC) is the most aggressive and the most difficult to treat in all breast cancers. Hypoxia has been known to be a critical factor in the malignant progression of many cancers. In recent years, many studies indicated that long noncoding RNAs (lncRNA) were involved in a variety of biological functions and hypoxia induced the expression of many lncRNAs in breast cancer cells, including *NDRG1-OT1* identified in our lab. However, their regulatory mechanism and biological functions remain largely unknown. Therefore, the purpose of this study is to investigate the regulation and function of lncRNA *NDRG1-OT1* in TNBC cells. Using Luciferase reporter assays and chromatin immunoprecipitation, we found that the hypoxia-inducible factor α (HIF-1α) bound at the *NDRG1-OT1* promoter and enhanced the expression of *NDRG1-OT1*. Next, using bioinformatics prediction and experimental validation, the results showed that *NDRG1-OT1* could bind with miR-875-3p. Furthermore, many recent studies reported that some lncRNAs were able to translate small peptides to participate a wide range of biological processes. Our study also found that *NDRG1-OT* could translate small peptides. Lastly, overexpression of *NDRG1-OT1* enhanced tumor malignancy by promoting cell proliferation, colony formation, migration and invasion capacity of triple-negative breast cancer cells, angiogenesis in peripheral endothelial cells, and tumor growth in xenograft mouse models. Taken together, the hypoxia-induced lncRNA *NDRG1-OT1* was transcriptionally regulated by HIF-1α in nucleus, could translate small peptides and sponged with miR-875-3p in cytoplasm, and promoted cell malignancy of triple-negative breast cancer cells.

Poster 17

Synovial Fluid Interleukin-16 Contributes to Osteoclast Activation and Bone Loss through the JNK/NFATc1 Signaling Cascade in Patients with Periprosthetic Joint Infection

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Abstract

Because of lipopolysaccharide (LPS)-mediated effects on osteoclast differentiation and bone loss, periprosthetic joint infection (PJI) caused by Gram-negative bacteria increases the risk of aseptic loosening after reimplantation. Synovial fluid interleukin-16 (IL-16) expression was higher in patients with PJI than in patients without joint infection. Thus, we explored the effects of IL-16 on bone. We investigated whether IL-16 modulates osteoclast or osteoblast differentiation in vitro. An LPS-induced bone loss mice model was used to explore the possible advantages of IL-16 inhibition for the prevention of bone loss. IL-16 directly activated p38 and c-Jun N-terminal kinase (JNK)/mitogen-activated protein kinase (MAPK) signaling and increased osteoclast activation markers, including tartrate-resistant acid phosphatase (TRAP), cathepsin K, and nuclear factor of activated T cells 1 (NFATc1). IL-16 directly caused monocytes to differentiate into TRAP-positive osteoclast-like cells through NFATc1 activation dependent on JNK/MAPK signaling. Moreover, IL-16 did not alter alkaline phosphatase activity or calcium deposition during osteoblastic differentiation. Finally, IL-16 inhibition prevented LPS-induced trabecular bone loss and osteoclast activation in vivo. IL-16 directly increased osteoclast activation through the JNK/NFATc1 pathway. IL-16 inhibition could represent a new strategy for treating infection-associated bone loss.

Poster 18

Deficiency of CCR5 Signaling Enhances Immunosuppressive Profile of MDSCs but Deteriorates Hepatocyte Steatosis in NAFLD

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Abstract

Non-alcoholic fatty liver disease (NAFLD) is a common liver disease that associates with dysregulated immune functions. The goal of our study is to determine whether and how myeloid-derived suppressor cells (MDSCs) and chemokine ligand 5 (CCL5) participate in the pathogenesis of NAFLD. We demonstrate here that either high fat diet-induced NAFLD or CCL5-deficiency alone is causative for the elevation of hepatic MDSCs. Nevertheless, CCL5-deficient obese mice manifest severe liver damage and microvesicular steatosis. Additionally, over-activation of STAT1/3/5 signaling in CCL5-deficient fatty liver insinuates a significant alteration of crosstalk between lipid metabolism and immunomodulation. Given that CCL5 receptor, CCR5, is highly expressed on hepatic MDSCs and hepatocytes, we narrow down putative targets for studying the molecular functions of CCR5 signaling in the liver. Our *in vitro* analysis of a murine hepatocyte cell line, AML12, suggests that null of CCR5 signaling strongly affects pivotal signals for lipid accumulation, such as STAT5 and NF-κB phosphorylation, in NAFLD micro-environment. Blockade of CCR5 also enhances critical MDSC expansion signal, STAT3, and up-regulates immunosuppressive features in primary MDSCs. In bone marrow transplantation experiments, deletion of hematopoietic CCL5 or CCR5 in wild-type C57BL/6 mice reverses liver injury. These findings lead us to hypothesize that CCR5 signaling may play distinct roles for hepatic MDSCs and hepatocytes. Experiments are ongoing to test our hypothesis.

Poster 19

Effect of Kefir Exopolysaccharide Extract on STZ-induced Type 2 Diabetes Mellitus Rats

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Abstract

Diabetes mellitus is a chronic metabolic disease, it makes a big issue that the diabetes population increase about 25,000 people per year in Taiwan, 95% of them has diagnosed as type 2 diabetes mellitus, which is the type has insulin resistance and can't use insulin effective to reduce the blood glucose. Kefir is a fermented dairy products made by kefir grain, it has been found in North Causasus and East Europe as a kind of beverage. Recently, many studys have showed that kefir has a lot of benefits on many ways, such as relieves osteoporosis, hypertension, inflammation etc. The aim of this study is using the exopolysaccharide extracted from kefir to improve type 2 diabetes mellitus, which was induced by streptozotocin (STZ) with high fat diet. Male Sprague-Dawley rats were randomly divided into four groups: control, diabetes untreated (mock), high-dose kefir exopolysaccharide (KEPS), low-dose KEPS treated diabetes rats. Every group fed high fat diet for four weeks, afterwards, diabetes were induced by STZ (35 mg/kg/bw, i.p.). The fasting blood glucose was measured after 72 hours to check whether it has modeling. Next day gave every group oral glucose tolerate test (OGTT) and measured the blood insulin for calculating the HOMA-IR. Treated KEPS for four weeks, we measured blood glucose, food intake, urine volume for every week and sacrificed at the forth week after treatment starting. The data of biochemistry analysis, section histological staining, OGTT and HOMA-IR, showed that KEPS can improve diabetes rat's insulin resistance and hypoglycemic. Kefir polysaccharide can reduce the pancreas damage from type 2 diabetes mellitus. We speculated that KEPS could be act as a normal approach for diabetes therapy in the future.

Keywords: Streptozotocin (STZ), kefir exopolysaccharide, type 2 diabetes mellitus, insulin resistance

Poster 20

Effects of an FKBP51 inhibitor on GR activity and glutamate homeostasis in astrocytes under excitotoxicity

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Abstract

Brain injury often causes excessive accumulation of extracellular glutamate due to impaired astrocyte-mediated glutamate homeostasis, which leads to excitotoxic neuronal death. Injury-induced stress response elevates endogenous glucocorticoids (GCs), such as corticosterone (CORT) and cortisol, in both periphery and the brain. FK506-binding protein 51 (FKBP51) encoded by Fkbp5 gene is a negative co-chaperone of glucocorticoid receptor (GR), and its overexpression has been linked to GC resistance in psychiatric disorders. We previously reported that c-Jun N-terminal kinase 1 (JNK1) signaling pathway regulates astrocytic plasticity under excitotoxicity. In this study, we further examined the effect of an FKBP51 inhibitor SaFit2, on GR sensitivity to GC, glutamate transporter expression, and neuroplasticity in primary rat astrocyte culture and glia-neuron mix culture subjected to excitotoxic glutamate and stress hormone CORT stimulations. Our data showed that CORT-induced GR and FKBP51 expressions in astrocytes can be significantly enhanced by high glutamate stimulation that mimics excitotoxic microenvironment. SaFit2 decreased GR levels without affecting FKBP51 expression in glutamate-CORT-cotreated astrocytes. SaFit2 also enhanced CORT-increased SGK1 expression, a GR target gene that was reported to downregulate glutamate transporter 1 (GLT-1) in astrocytes. However, SaFit2 did not affect the CORT effect on glutamate-induced GLT-1 downregulation. Yet, SaFit2 could facilitate CORT inhibition of glutamate-induced JNK1 phosphorylation that regulates astrocytic process formation for glutamate uptake capacity. Immunocytochemistry and morphometric analysis further revealed that CORT, JNK inhibitor, and SaFit2 can all attenuate glutamate-induced reduction of GLT-1+ astrocytic process density. Importantly, in glia-neuron mix culture, SaFit2 could attenuate the excitotoxicity-induced disruption of neuronal and glial processes. In summary, these results suggest that SaFit2 that inhibits FKBP51 to enhance GR sensitivity to CORT may facilitate astrocytic plasticity and glutamate uptake capacity by inhibiting JNK1 activation, thereby protecting neuron-astrocyte coupling against excitotoxicity.

大會須知及注意事項

一、 出發前的準備須知

1. 本活動以所有與會人員之安全與健康為第一優先考量。為配合政府相關防疫措施，**請於8/13前先填寫健康聲明書** (填寫連結：https://bit.ly/3ghO6xD)，並配合主辦單位相關防疫規定(請見第十點)。
2. 山區氣候不穩定，請務必準備**雨具**、涼鞋或拖鞋等，以備不時之需。
3. 惠蓀林場海拔約 770公尺，天氣較平地涼爽，晝夜溫差大，建議您可攜帶薄外套、遮陽防曬物品(如防曬乳液、遮陽傘、帽子)、個人常備藥品、**防蚊用品**、望遠鏡、穿著長褲及輕便衣物。山區夜間照明不足，山中步道因露水而時有濕滑，建議自備**手機照明或手電筒**，及穿著防滑休閒鞋。
4. 為簡化現場報到流程，秘書處將於第二次行前通知發送行前報到QR code, 以供與會人員核對個人之名牌內容及瀏覽議程。
5. 請勿攜帶寵物入園。
6. 於行前若臨時因故無法出席本活動，請務必致電本會秘書處李青澔秘書長（手機0911-158705）；徐松柏會務幹事（手機0955-995012），以便因應。

二、 交通須知 (前往惠蓀林場有一段山路較為蜿蜒，如果有必要，請先服用**暈車藥**)

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1. 惠蓀林場住址：南投縣仁愛鄉新生村山林巷1號 (http://huisun.nchu.edu.tw/home.php)。

2. 在園區大門收費站時，請告知櫃台人員您來參加「生理醫學研習會暨科技部研究成果發表會」，或出示活動海報當車輛識別證（請自行列印）即可入園，無須付任何費用，並請依照指示進入惠蓀林場。自行開車名單放置於第64頁。

3. 惠蓀林場因進行「明隧道旁邊坡處理工程」，周一(8/17)道路將進行交通管制(管制時間如下表)，因此8/17第二天的議程將於11:30結束，並發放便當，以便於12:00-13:00放行時段下山的車輛(含遊覽車)能於12:30以前出發下山，請各位與會者配合，造成不便，敬請見諒。

惠蓀林場明隧道旁邊坡處理工程 交通管制時間表 預計管制期間：109年3月10日至109年12月13日		
周一至周五	時間	管制情形
	08:00~10:00	管制開始，禁止通行
	10:00~10:20	放行
	10:20~12:00	管制開始，禁止通行
	12:00~13:00	放行
	13:00~15:00	管制開始，禁止通行
	15:00~15:20	放行
	15:20~17:00	管制開始，禁止通行
	17:00	管制結束
週六、週日 國定假日	不管制	

搭乘大會接駁車者:

8/16日去程: 共六輛接駁車。發車現場將有穿著生理學會藍色背心之工作人員，請主動告知工作人員您的大名及單位，以完成簽到及領取會議資料。為使交通車能準時於午餐時間以前抵達惠蓀(下午1:00會議準時開始)，各交通車均將準時發車，逾時礙難等候喔！（萬一無法趕上接駁車，請務必致電隨車人員，以便協助因應）。接駁車名單分配表放置於本通知第65頁，請您留意搭車時間、地點，若臨時有突發狀況，請與隨車人員聯繫。

台北-惠蓀 (北1車/北2車): 上午**8:00**前抵達**台北車站東三門**集合，**8:20準時發車**。

台中-惠蓀 (中1車/中2車): 上午**9:45**前抵達**高鐵台中站一樓6號出口**集合(高鐵到站後請由 5-6 號出口指示的手扶梯下樓至一樓的 6 號出口），**10:00準時發車**。

台南-惠蓀 (南1車): 上午**8:45**前抵達**成大醫學院(成杏校區小東路警衛室門口)**集合，**9:00**準時發車。

高雄-惠蓀 (南2車): 上午**8:00**前抵達**高雄新左營高鐵站(彩虹市集外)**集合，**8:20準時發車。**

8/17回程: 共四輛接駁車，接駁車名單分配表放置於第66頁。因配合惠蓀林場邊坡工程交通管制，接駁車將於12:45 從惠蓀林場遊客中心旁發車。

三、報到須知

1. 報到地點：將於惠蓀林場遊客服務中心辦理報到，發放名牌及手冊 (活動期間請全程配掛名牌)，報到時間為11:00 - 13:00。
2. 行李請先寄放在櫃枱。
3. 為維護報到時人流順暢，報到桌分為以下四類，請您依照分類進行報到手續。

   A. 演講及主持人嘉賓

   B. 教師

   C. 博後與研究助理

   D. 學生
4. 壁報張貼：壁報編號已於 email 個別通知論文作者。發表人請於13:00會議開始前，依照編號至會議中心大廳正確看板位置張貼壁報論文，現場備有雙面布膠帶供黏貼壁報，請勿使用圖釘或雙面膠，若有疑問請洽詢現場工作人員。壁報論文現場解說時段為19:00 - 20:00。並請於8/17會議結束後自行撤除攜回。壁報尺寸：直式/建議論文輸出尺寸：W90cm × H150cm。論文之圖表文字大小以在一公尺距離可清楚閱讀為原則。
5. 因配合惠蓀林場邊坡工程交通管制，第二天會議議程有所變動，請見手冊第2頁。
6. 請演講嘉賓協助配合，將演講PPT電子檔請於 8/15 前寄交李青澔秘書長(bros22@tmu.edu.tw)或徐松柏會務幹事(sphsu@tmu.edu.tw)，以利會議進行之時間掌控。

四、會場：

本次活動主要為於**惠蓀林場會議中心**，距離餐廳、遊客服務中心、和住宿小木屋區徒步約5-10分鐘。由於本次活動報名踴躍，小木屋區不敷使用，有部分嘉賓安排至研習中心住宿，研習中心距離會議中心約1.2公里，步行約20分鐘，秘書處將協助安排該區人員之車輛共乘。(可參考封面內頁惠蓀林場地圖)

五、用餐須知

第一天午餐和晚餐: 地點於惠蓀林場餐廳，為桌菜型式，請大家務必準時前往就坐，為促進與會者多交流，因此活動期間之用餐，將不安排固定座次，現場將有工作人員引導您入座。(第一天晚餐參與壁報展示的同學將特別安排與圓桌導師同桌，以增進交流)。素食桌將另有標示。

第二天早餐: 上午 7:00 可開始自由用餐，地點於惠蓀林場餐廳(遊客中心報到處旁)。

第二天午餐: 中午 11:30 於會議中心領取餐盒。

六、住宿須知：

1. 本活動依報名之訂房需求安排1-4人一間房，共用一把客房鑰匙(房號標示於名牌背面)。
2. 由於飯店作業所需，住房鑰匙會於18:00會議結束後於會議中心統一發放。如有家眷想提早領取鑰匙回房休息，可於 15:00~15:30 攜帶名牌至遊客中心報到桌領取，逾時因大會作業，將統一於 18:00 後發放。
3. **熱水供應山莊住宿區雖無限制，但建議盡量晚上12點前使用；研習中心因設備受限則有17:00-24:00的限制。**
4. 退房須知：為避免影響會議進行，建議8/17上午 **09:00**前完成check out，將房間鑰匙交還櫃枱，行李寄放在櫃枱。如有家眷仍需使用房間設備，惠蓀最晚退房時間為11:00。

七、林場探索：

於 8/17上午 **07:30** 於**餐廳門口**集合出發，需時約一小時。

八、緊急連絡電話:

生理學會承辦人：李青澔秘書長(0911-158705)；徐松柏會務幹事(0955-995012)

九、若因天災或其他不可抗力之因素時，會以 email 通知與會者最新消息並同步公佈於中國生理學會網頁及FB。

十、活動防疫建議

依據「嚴重特殊傳染性肺炎中央流行疫情指揮中心」監測資料顯示，目前國內之確診病例以境外移入為主，除從事醫療照護工作或與確診病例曾有密切接觸者外，一般大眾於社區感染之風險相對較低。由於集會活動通常人潮擁擠，長時間且近距離可能增加呼吸道傳染病之傳播風險，最基本且最重要的防疫措施仍是：**落實勤洗手、呼吸道衛生與咳嗽禮節，及生病在家休息**。

1. 落實自主健康管理，**請於活動前三日填寫風險評估表**。
2. **室內集會活動場所務必配戴口罩**。
3. 加強衛教宣導。透過明顯告示(如:海報等)宣導「呼吸道衛生與咳嗽禮節」。
4. **有呼吸道症狀者，應配戴口罩儘速就醫後在家休養，避免參加集會活動。**有發燒者，直至退燒後至少 24 小時才可參加集會活動，如集會活動辦理當日未達此標準，應避免參加。
5. **維持良好個人衛生習慣，落實正確勤洗手、呼吸道衛生及咳嗽禮節(如:咳嗽時以面紙或手帕遮口鼻)，避免觸摸眼睛、鼻子、嘴部，如不得已需碰觸，則一定要事先確實清潔手部，以減少感染與傳播疾病的機會。**
6. **維持社交距離。保持室內 1.5 公尺、室外 1 公尺以上之社交距離，否則請正確佩戴口罩。**
7. 由於車站、候車地點及大眾運輸工具等屬公眾使用且多為密閉空間，長時間且近距離接觸不特定人士，可能增加呼吸道傳染病之傳播風險，依據交通部 109 年 4 月 15 日交航字第 1095004256 號公告，自公告日起至指揮中心解散之日止，**搭乘指定大眾運輸工具時，務必佩戴口罩**，未佩戴者不得搭乘。且搭乘具體溫監測設備之大眾運輸工具時，應配合體溫測量措施。

8. 用餐時務必落實公筷母匙，進食時避免近距離交談。大會將視防疫指揮中心的最新指引，必要時調整用餐型式。

9. 工作人員可能經常直接面對面接觸人群者，建議配戴口罩。主辦單位備有足量個人清潔及防護用品。

10. 若工作人員或參加者在集會活動期間出現呼吸道症狀者，應讓其戴上口罩，暫時留置非人潮必經處且空氣流通之安置空間，直至其返家或就醫。如前開患者出現嚴重不適症狀(如高燒不退、吸呼困難、呼吸急促、胸痛暈眩、抽搐、嚴重腹瀉等)，集會活動之主責人員應協助其儘速就醫

自行開車名單

李青澔、詹燕茹、溫宏諾、陳竑愷	12○○-QM	王家義、鄭惠玲、王司道、王思亮	BEX-10○○
蔡少正、鍾懿柔、湯宜禎、黃美鳳	BAF-○○15	陳紹寬、朱佩文	AHK-59○○
何應瑞	3○○3-QX	張原翊、阮琪昌、林長億、陳碩文	AKF1○○9
廖娟妙		顏賢章	BEX-10○○
劉哲育、徐娟珍	63○○-YV	呂史提	AJD-9○○2
陳威宇、張俐盈	29○○-SJ	蕭貴陽、林雅琪、蕭謙碩	88○○-V3
陳美鳳、林盈宏	1○○6-G7	蔡靜宜	AQY-5○○5
黃炯瑋、劉永筑	70○○-N3	賴政遠	AXG-76○○
吳莉玲	AGJ-○○25	阮子軒、陳瑾儀、簡友琳、楊宗珉	AMX-57○○
陳嘉晏、廖堉甄、初昭瑄、陳妍君	AGJ-○○25	高永旭、姜碧如、高芝恩	8○○2-RV
陳倫魁	ATQ-○○26	彭瑞銘	BDY-0○○8
陳珮君	AJX-○○81	李佳陽、李祖維、李祖頤、董瓊盛、王述綺	ADB-9○○3
汪雅雲	○○36-G7	林赫、林巧、林翰	AFS-6○○9
陳美智	AFS-6○○9	林士傑、李敏嘉、林以哲、林以恩	ATK-3○○1
吳承修	AMK-3○○2	陳俊霖	5○○0-K3
何盧勳	92○○ ZE	連正章、陳倩	ANA-○○56
宋瑞敏	AXE-○○07	詹佩穎、詹雅智	9○○2VD
吳偉立、黃曉寧、吳泓賢	BAJ-7○○2	陳鴻震司長及夫人	5○○0-YL
林恒、詹鳳妮		郭宜盈	

＊ 本表依報名資料整理

8/16 交通車集合及名單分配表

集合地點	集合時間	發車時間	隨車人員 (手機)	搭乘人員 (* 表示車長)
台北車站 東三門	08：00	08：20	北 1 辛予蕎 (0928-797573) 游博凱 (0936-716976)	徐松柏*、林佑穗*、許諄諄、林天南、賴亮全、莊麗玲、陳景宗、宋品樺、甘育菱、黃昱傑、方雅菁、游博凱、陳彥碩、藍以傑、辛予蕎、盧主欽、莊淑惠、吳明恒、李宗玄、李學德、趙偉廷、蘇詩涵、林妤家、王凱立、高嫩涵、林惠菁、陳思元、廖子傑 (共 28 人)
			北 2 張皓程 (0975-319192) 劉大維 (0922-330404)	林君樺*、洪家琪*、吳鈺琳、蔡思綺、高珮瑜、初銘家、吉翔、謝宗翰、李旂緯、林心荃、廖詠維、吳澯宇、陳姵蓁、李冠儀、鄭伊純、趙幸華、蔡羽庭、尤正昀、石忠賢、白宇辰、張皓程、劉大維、郭倍佳、陳玟樺、劉冠緯、欒玖霖 (共 26 人)
台中烏日 高鐵站 (高鐵站 一樓 6 號出口 處)	09：45	10：00	中 1 周心喬 (0976-770557)	李昆澤*、王羿忻*、余佳慧、李怡萱、謝博軒、馬文隆、華瑜、李憶菁、謝昀容、康宏佑、魏國鼎、劉雅棻、謝坤叡、許美鈴、田履黛、楊世斌、周心喬、余盈君、黃菁英、高祖仁、黃祺真、張絲珍、林榮興 (共 23 人)
			中 2 張主龍 (0910-395221)	林雅婷*、李宜釗、莊健盈、郭亭攸、Nguyen Thi Mai Huong、鍾沛容、黃予庭、鄭靜宜、徐宗溢、張主龍、宋軒瑋、黃玲茹、黎喻暄、Trayee Dhar、游椀媜、鄭語喬、許璷文、劉芳伶、Ayse Celik、G M Shazzad Hossain Prince、Muhammet Oner、鄭邦廷、陳建瑋、蕭安淇、蔡忠良、黃婉婷、侯俊宏 (共 27 人)
成大醫學 院 (成杏校 區小東路 警衛室門 口)	08：45	09：00	南 1 許 筠 (0988-155133)	張雅雯*、楊尚訓*、李婉寧、林新智、黃允辰、黃于軒、陳幸儀、許筠、曾秀珍、黃品優、楊琇羽 、楊晴雅、楊詠云、鄭佩薰、葉儀君、洪珮娥、許佩玲、蕭雅心、林逸宣、李純純、彭怡禎、鍾懿柔、湯宜禎、黃美鳳 (台南共 24 人)
高雄新左 營高鐵站 (彩虹市 集外)	08：00	08：20	南 2 吳慶龍 (0910-846130)	蔡克勵*、楊政霖*、李枝芳、蔡秉真、陳宜芳、徐志文、劉佩芬、葉宇軒、曾鴻泰、陳定濰、王于倫、黃廣慈、孫昭玲、鍾智聆、張姿淳、林士哲、吳慶龍 (高雄共 17 人)

8/17 交通車集合及名單分配表

集合地點	集合/發車時間	隨車人員 (手機)	搭乘人員 (*表示車長)
惠蓀林場 遊客服務 中心	12:30 集合 12:45 發車	北 1 辛予蕎 (0928-797573) 游博凱 (0936-716976)	徐松柏*、林佑穗*、許諄諄、林天南、賴亮全、莊麗玲、陳景宗、宋品樺、甘育菱、黃昱傑、方雅菁、游博凱、陳彥碩、藍以傑、辛予蕎、盧主欽、莊淑惠、吳明恒、李宗玄、李學德、趙偉廷、蘇詩涵、林妤家、王凱立、高媺涵、林士哲、陳思元、廖子傑、陳建瑋、張雅雯 (共 30 人)
		北 2 (經台中高鐵站) 張皓程 (0975-319192) 劉大維 (0922-330-404)	林君樺*、洪家琪*、吳鈺琳、蔡思綺、高珮瑜、初銘家、吉翔、謝宗翰、李旂緯、林心荃、廖詠維、吳潑宇、陳姵蓁、李冠儀、鄭伊純、趙幸華、蔡羽庭、尤正昀、石忠賢、白宇辰、張皓程、劉大維、郭倍佳、陳玟樺、劉冠緯、欒玖霖 (共 26 人) 黎喻暄、Trayee Dhar、游椀媜、鄭語喬、許璨文、劉芳伶、Ayse Celik、G M Shazzad Hossain Prince、Muhammet Oner、鄭邦廷、蕭安淇、蔡忠良(共 12 人)
		中 1 周心喬 (0976-770-557)	李昆澤*、王羿忻*、余佳慧、李怡萱、謝博軒、馬文隆、華瑜、李憶菁、謝昀容、康宏佑、魏國鼎、劉雅棻、謝坤叡、許美鈴、田履黛、楊世斌、周心喬、余盈君、黃菁英、高祖仁、黃祺真、張絲珍、林榮興、林惠菁 (共 24 人) 郭亭攸、Nguyen Thi Mai Huong、鍾沛容、黃予庭、林雅婷、鄭靜宜、李宜釗、莊健盈、徐宗溢、宋軒瑋、黃玲茹、黃婉婷、侯俊宏 (共 13 人)
		南 1 (經成大醫學院~終點站高鐵左營站) 許　　筠 (0988-155133) 吳慶龍 (0910-846130) 張主龍 (0910-395-221)	楊尚訓、李婉寧、林新智、黃允辰、黃于軒、陳幸儀、許筠、曾秀珍、黃品優、楊琇羽、楊晴雅、楊詠云、鄭佩薰、葉儀君、洪珮娥、許佩玲、蕭雅心、林逸宣、李純純、彭怡禎、吳慶龍、鍾懿柔、湯宜禎、黃美鳳(台南共 24 人) 蔡克勵*、李枝芳、蔡秉真、楊政霖、陳宜芳、徐志文、劉佩芬、葉宇軒、曾鴻泰、陳定濰、王于倫、黃廣慈、孫昭玲、鍾智聆、張姿淳、張主龍 (回程)、承洛業務*2 (高雄共 18 人)