編號 No.	投稿學會 Society	研究領域 Topic	題目Title	投稿者 Name	作者 CO-Author	作者(Co-Author)	單位(Affliation)	關鍵字(Keywords)	poster number
20200728161043	台灣基礎神經科學學會	基礎	Effects of dim light at night on metabolic status and intestinal gene expressions in mice	Mr. 單禹堯	李亦騏、單禹堯和陳示國	I-Chi Lee, Yu-Yau Shan, and Shih-Kuo Chen	Department of Life Science, National Taiwan University, Taipei 106, Taiwan	circadian rhythm,light at night,metabolic disorders,intestinal gene expressions,	1
20200802151713	台灣基礎神經科學學會	基礎	Light modulates oxytocin release and socio-sexual behavior in mice through ipRGCs	Mr. Yu-Fan Huang	黃宇凡、廖柏喻、游若嫻、陳示國	Yu-Fan Huang, Po-Yu Liao, Jo-Hsien Yu, Shih-Kuo Chen	Department of Life Science, National Taiwan University	ipRGCs,Oxytocin,social interaction,Light	2
20200809124938	台灣基礎神經科學學會	基礎	In vivo neuron activity study of the suprachiasmatic nucleus through gradient-index lenses: a novel aspect of the mammalian circadian rhythms	Mr. 葉柏廷	葉柏廷、鄭志帆、陳示國	Po-Ting Yeh, Chih-Fan Jeng, Shih-Kou Chen	Department of Life Science, National Taiwan University	suprachiasmatic nucleus,In vivo calcium imaging,circadian rhythm,gradient-index lens	3
20200630195936	台灣基礎神經科學學會	基礎	Light Pattern is Important for Circadian Photoentrainment	Ms. 蕭亦聆		I-Ling Hsiao Shih-Kuo Chen	Department of Life Science, National Taiwan University	Central circadian pacemaker,Suprachiasmatic nucleus (SCN),Intrinsically photosensitive retinal ganglion cells (ipRGCs),Circadian photoentrainment,Phase shift	4
20200727212221	台灣基礎神經科學學會	基礎	Light-Dark Cycle Mediates Diurnal Oscillatory Rhythms in Gut Microbiota	Ms. 梁 風	梁風; 陳示國	Feng Liang; Shih-Kuo Chen	Department of Life Science, National Taiwan University	Light-Dark Cycle,Diurnal Oscillatory Rhythms,Gut Microbiota	5
20200729104803	台灣基礎神經科學學會	基礎	Investigation of daily and tidal behavioral rhythm in Shuttles hoppfish (Periophthalmus modestus)	Ms. Yan-Min Chiu	邱妍敏，廖柏喻	Yan-Min, Chiu, Po-Yu, Liao, Shih-Kuo, Chen	College of Life Science, National Taiwan University	circatidal rhythm,circadian rhythm,mudskipper,Periophthalmus modestus,intertidal zone	6
20200730103538	台灣基礎神經科學學會	基礎	The impact of early-life antibiotic exposure on the neurodevelopmental outcomes and gut microbiota development in mice	Ms. 林元元	林元元, 吳偉立	Yuan-Yuan Lin, Wei-Li Wu	Department of Physiology, College of Medicine, National Cheng Kung University	Neurodevelopmental disorders,Gut microbiota,Environment-environment interaction,Peripubertal stress,Early-life challenge	7
20200730110734	台灣基礎神經科學學會	基礎	The level of COUP-TFI governs the septo-temporal region patterning during hippocampal development	Dr. Ching-San Tseng	曾慶三 周申如	Ching-San Tseng Shen-Ju Chou	Institute of Cellular and Organismic Biology, Academia Sinica, Taipei	NR2F1 gene,dorso-ventral axis,neuronal specification,embryonic neurogenesis,hippocampal hyperplasia	8
20200730144509	無	基礎	COUP-TFI specifies entorhinal cortex and determines the location and integrity of its border through cell affinity mechanisms	Dr. Wen-Hsin Hsu		Jia Feng, Wen-Hsin Hsu, Denis Paterson, Ching-San Tseng, Zi-Hui Zhuang, Hsiang-Wei Hsin, Yi-Ting Huang, Jonathan Touboul and Shen-Ju Chou	Institute of Cellular and Organismic Biology, Academia Sinica, Taipei, Taiwan	COUP-TFI,entorhinal cortex,cell fate specification,border integrity,cell affinity	9
20200808003730	無	基礎	Lhx2 regulates cortical neuronal excitability to maintain thalamus development	Dr. Chia-Fang Wang		Chia-Fang Wang, Jenq-Wei Yang, Hsin-Yo Chen, Heiko Luhmann and Shen-Ju Chou	Institute of Cellular and Organismic Biology, Academia Sinica, Taipei, Taiwan Institute of Physiology and Pathophysiology, University Medical Center of the Johannes Gutenberg University Mainz, Mainz, Germany	Lhx2,barrel cortex,thalamus,neuronal excitability	10
20200803145519	無	基礎	Contribution of Dbx1 lineage cells to the Piriform Cortex	Mr. Thando Wizzy Shabangu		Thando W. Shabangu	Taiwan International Graduate Program in Molecular Cell Biology	Piriform cortex,Dbx1,orbitofrontal cortex	11
20200725201324	台灣基礎神經科學學會	基礎	Generating hiPSC-derived cerebral organoids to model human brain development and primary microcephaly	Mr. Hsiao-Lung An	安小龍, 郭紘志, 唐堂	Hsiao-Lung An, Hung-Chin Kuo, Tang K. Tang	Program in Molecular Medicine, National Yang-Ming University, Taipei, Taiwan Institute of Biomedical Sciences, Academia Sinica, Taipei, Taiwan	CPAP/Cenpj,Centriole,Microcephaly,oRG,hiPSC-derived brain organoid	12
20200708063320	台灣基礎神經科學學會	基礎	Investigating the roles of centriolar protein Cep120 during cerebellar development	Dr. Chia-Hsiang Chang		Chia-Hsiang Chang, I-Ling Lu, Jhih-Jie Tsai and Tang K. Tang	Institute of Biomedical Sciences, Academia Sinica	Centriolar protein Cep120,Granule neuron progenitors,Primary cilium,Cerebellar development,Joubert syndrome	13
20200724181408	台灣基礎神經科學學會	其他	Cap Methyltransferase 2 (CMTR2) is required for cerebellar development and function	Ms. Sayma Azeem		Sayma Azeem 1,2 , Imelda Margaretha A2, Yi-Shuian Huang1,2*	1 Taiwan International Graduate Program in Interdisciplinary Neurosciences, National Yang-Ming University and Academia Sinica, Taipei, Taiwan 2 Institute of Biomedical Sciences, Academia Sinica, Taipei, Taiwan	CMTR2,Cerebellar development,cerebellar function,Motor Coordination	14
20200725210926	台灣基礎神經科學學會	基礎	Knockout of a Splicing Regulator Causes Cerebellar Vermis Hypoplasia	Mr. 沈久倫		Dhananjaya D, Chiu-Lun Shen, Ching-Yen Tsai, Wataru Kakegaw, Michisuke Yuzaki, Woan-Yuh Tarn	Institute of Biomedical Sciences, Academia Sinica, Taipei, Taiwan Institute of Molecular Medicine, College of Medicine, National Taiwan University Department of Physiology, Keio University School of Medicine, Tokyo, Japan	alternative splicing,cerebellar foliation,vermis hypoplasia	15

20200729105338	台灣基礎神經科學學會	基礎	Dopamine D2 receptor antagonist trifluoperazine declines HFD-induced inflammation and gliosis	Dr. Hui-Ting Huang	黃暉庭, 陳珮君, 陳柏熹, 郭余民, 曾淑芬	Hui-Ting Huang1, Pei-Chun Chen2, Po-See Chen3, Yu-Min Kuo4, Shun-Fen Tzeng1*	1 Department of Life Sciences, College of Bioscience and Biotechnology, National Cheng Kung University, Tainan, Taiwan 2 Department of Physiology, College of Medicine, National Cheng Kung University, Tainan, Taiwan 3 Department of Psychiatry, College of Medicine, National Cheng Kung University, Tainan, Taiwan 4 Department of Cell Biology and Anatomy, College of Medicine, National Cheng Kung University, Tainan, Taiwan	Trifluoperazine,Neuroinflammation,Obesity,Microglia,	16
20200731165026	台灣基礎神經科學學會	基礎	The detrimental impacts of proteasome over-activation on acute intracerebral hemorrhage in rats	Dr. Hock-Kean Liew	廖學健、胡瑋芬、林伯謙、蔡伯宜、張般誠、方若茵、羅明仁、張增岷、馮清榮、陳宗鷹	Hock-Kean Liew 1,2,3, Wei-Fen Hu 3, Peter Bor-Chian Lin 4, Andy Po-Yi Tsai 4, Yin-Cheng Chang 1, Jo-Yin Fang 1, Shaik Ismail Mohammed Thangameeran 1,5, Tseng-Min Chang 1, Cheng-Yoong Pang 1,2,5,*, Tsung-Ying Chen 6,7,8,*	1 Department of Medical Research, Hualien Tzu Chi Hospital, Buddhist Tzu Chi Medical Foundation, Hualien, Taiwan 2 Neuro-Medical Scientific Center, Hualien Tzu Chi Hospital, Buddhist Tzu Chi Medical Foundation, Hualien, Taiwan 3 PhD Program in Pharmacology and Toxicology, Tzu Chi University, Hualien, Taiwan 4 Indiana University School of Medicine, Indianapolis, IN, USA 5 Institute of Medical Sciences, Tzu Chi University, Hualien, Taiwan 6 Department of Anesthesiology, Hualien Tzu Chi Hospital, Buddhist Tzu Chi Medical Foundation and Tzu Chi University, Hualien, Taiwan 7 School of Medicine, Tzu Chi University, Hualien, Taiwan 8 Department of Medical Education, Hualien Tzu Chi Hospital, Buddhist Tzu Chi Medical Foundation and Tzu Chi University, Hualien, Taiwan	unfolded protein response,neuroinflammation,neuroprotection,proteasomal inhibition,ER stress	17
20200810165649	台灣基礎神經科學學會	基礎	Sleep deprivation exacerbates neuroinflammation and impairs hippocampal neurogenesis in heterozygous Disc1 mutant mice	Ms. Chih Yu Tsao	曹志瑜, 段立珩, 李俊賢, 劉智民, 胡海國, 李立仁	Chih-Yu Tsao , Li-Heng Tuan, Lukas Jyuhn-Hsiarn Lee, Chih-Min Liu, Hai-Gwo Hwu and Li-Jen Lee	Graduate Institute of Anatomy and Cell Biology, National Taiwan University, Taipei, Taiwan	sleep deprivation,psychiatric disorder,microglia,neurogenesis,proinflammatory cytokine	18
20200724145600	台灣基礎神經科學學會	基礎	The role of CCL5 in hippocampal memory function and antioxidant activation after mild TBI	Mr. Manhau Ho	何文孝 1,2; 周思怡 2,3	Man-Hau Ho1,2; Szu-Yi, Chou2,3	1 Graduate Institute of Medical Sciences, College of Medicine, Taipei Medical University, Taipei, Taiwan 2 Ph.D. Program for Neural Regenerative Medicine, College of Medical Science and Technology, Taipei Medical University and National Health Research 3 Graduate Institute of Neural Regenerative Medicine, College of Medical Science and Technology, Taipei Medical University, Taipei, Taiwan	CCL5,mild traumatic brain injury,reactive oxygen species,NADPH oxidase,antioxidant	19
20200730120847	台灣基礎神經科學學會	基礎	Voluntary Exercise As a Preventative Strategy for Microglia-mediated Synaptic Pruning Defects on Sleep-deprived Adolescent Mice	Dr. 段立珩	段立珩、曹志瑜、李立仁	Li-Heng Tuan, Chih-Yu Tsao and Li-Jen Lee	Graduate Institute of Anatomy and Cell Biology, College of Medicine, National Taiwan University	Sleep deprivation,Microglia,Adolescent,Exercise	20
20200809152009	台灣生物精神醫學暨神經精神藥理學會	臨床	Inflammatory cytokines and executive function may be correlated with outcomes of substance use disorder	Dr. 王姿云	王姿云，陳柏熹，陸汝斌	Tzu-Yun Wang, Po See Chen, Ru-Band Lu	1 Department of Psychiatry, National Cheng Kung University Hospital, College of Medicine, National Cheng Kung University, Tainan, Taiwan 2 Institute of Behavioral Medicine, College of Medicine, National Cheng Kung University, Tainan, Taiwan 3 Yanjiao Furen Hospital, Hebei, China	cytokines,brain-derived neurotrophic factor,substance use disorder,Wisconsin card sorting test	21
20200810113242	台灣基礎神經科學學會	基礎	The deficient Clec5a ameliorates the neuroinflammation-induced neurodegeneration in Alzheimer's disease	Mr. Yu-Yi Lin		Yu-Yi Lin, Pei-Ling Hsieh, Wen-Han Chang, Han-Juo Cheng	Institute of Brain Science, National Yang-Ming University, Taipei 112, Taiwan	CLEC5A,Alzheimer's disease,Neuroinflammation	22
20200724103927	台灣基礎神經科學學會	基礎	Study the Effects of Zn2+ in Dopamine-induced Pyroptosis in Primary-cultured Rat Embryonic Cortical Neurons	Ms. Hui-Chiun Tseng	曾惠群 廖怡芬 潘建源	Hui-Chiun Tseng Yi-Fen Liao Chien-Yuan Pan	Department of Life Science, National Taiwan University, Taipei, Taiwan, ROC	dopamine,neurodegeneration,neuroinflammation,pyroptosis,Zn2+	23
20200810113652	台灣計算神經科學學會	工程	From neuroscience to engineering: a spiking neural network model of dynamic vision based on fruit fly visual system	Prof. Cheng-Te Wang	王誠德、葉宸甫、姚皇宇、高暐哲、白安雷、呂仁碩、謝志成、鄭桂忠、羅中泉	Cheng-Te Wang, Chen-Fu Yeh, Huang-Yu Yao, Wei-Tse Kao, Alexander White, Ren-Shuo Liu, Chih-Cheng Hsieh, Kea-Tiong Tang, Chung-Chuan Lo	Institute of Systems Neuroscience Department of Electrical Engineering	Obstacle detection,Neuromorphic network,Drosophila vision system	24

20200727153608	台灣計算神經科學學會	其他	A coupled neural circuit and Markov process model of spatial orientation in Drosophila melanogaster	Mr. Hsuan-Pei Huang	黃宣霈、韓睿、羅中泉	Hsuan-Pei Huang, Rui Han, Chung-Chuan Lo	Institute of Systems Neuroscience, National Tsing Hua University	central complex,navigation,attractor network,spatial orientation,angular path integration	25
20200810115812	台灣計算神經科學學會	基礎	The Advanced construction of FlyCircuit and analysis of neuron images	Prof. Hsiu-Ming Chang	張修明(1)，林敬堯(2)，莊朝鈞(2)，江安世(1)	Hsiu-Ming Chang(1), Ching-Yao lin(2), Chao-Chun Chuang(2) and Ann-Shyn Chiang(1)	(1) Brain Research Center, National Tsing-Hua University, (2) National Center for High-performance computing.	brain,circuit,image,database	26
20200810144235	台灣計算神經科學學會	基礎	Self-similarity of neurons in Strahler order analysis	Ms. Pin Ju Chou		Pin-Ju Chou, Ching-Che Charng, Harrison Ku, Chung-Chuan Lo	Department of Life Science, National Tsing Hua University	Strahler number,FlyCircuit,neuron morphology,fractals	27
20200722122937	台灣基礎神經科學學會	基礎	Spatiotemporal dynamics of astrocytic Ca2+ signalling in three-dimension in vivo	Mr. Pingyen Wu	吳秉彥 吳玉威	Ping-Yen Wu Yu-Wei Wu	Institute of Molecular Biology, Academia Sinica, Taipei 115, Taiwan	astrocyte,calcium imaging,3-dimension,in vivo,2-photon microscopy	28
20200727152730	台灣基礎神經科學學會	基礎	Ripple frequency emerges from coordinated activation of CA1 parvalbumin interneurons	Mr. Yi-Chieh Huang		Yi-Chieh Huang, Huei-Ching Chen, Szu-Ting Lin, Yu-Ting Lin, Ahmed S. Abdelfattah, Eric R. Schreiter, Bei-Jung Lin and Tsai-Wen Chen	Institute of Neuroscience, National Yang-Ming University, Taiwan	ripple,parvalbumin interneuron,voltage imaging	29
20200810200802	台灣基礎神經科學學會	基礎	Glutamate/GABA co-transmission modulates hippocampal neuron activity and long-term potentiation	Mr. Musa Iyiola Ajibola		Cheng-Chang Lien	Institute of Neuroscience, National Yang-Ming University, Taipei, Taiwan	Co-transmission,Supramammillary nucleus,Co-transmission,Dentate gyrus,Long-term potentiation	30
20200721232124	台灣基礎神經科學學會	基礎	Morpho-physiological Properties of Hippocampal Dentate Granule Cells in the Blm-s Knockout Mice	Mr. George Chia-Wei Yeh	葉家維1, 王凱誼1, 伊木夏1, 威漢1, 程淮榮2,5, 黃佩欣3,4, 連正章1,5*	Chia-Wei Yeh1, Kai-Yi Wang1, Musa Iyiola Ajibola1, Wahab Imam Abdulmajeed1, Hwai-Jong Cheng,2,5 Pei-Hsin Huang,3,4 and Cheng-Chang Lien1,5*	1 Institute of Neuroscience, National Yang-Ming University, Taipei 112, Taiwan 2 Institute of Molecular Biology, Academia Sinica, Taipei 115, Taiwan 3 Graduate Institute of Pathology, National Taiwan University, Taipei 100, Taiwan 4 Department of Pathology, National Taiwan University Hospital, Taipei 100, Taiwan 5 Brain Research Center, National Yang-Ming University, Taipei 112, Taiwan	BLM-s,dentate granule cells,patch-clamp recording,hippocampal curcuits,morphology	31
20200810192819	台灣基礎神經科學學會	基礎	Hilar Mossy Cells Differentially Regulate Dentate Gyrus Activity via Distinct Synaptic Mechanisms	Mr. Wahab Imam Abdulmajeed		Jei-Wei Wu, Cheng-Chang Lien	1Taiwan International Graduate Program in Interdisciplinary Neuroscience, Academia Sinica, Taipei, Taiwan 2Institute of Neuroscience, National Yang-Ming University, Taipei, Taiwan 3Brain Research Center, National Yang-Ming University, Taipei, Taiwan	Dentate gyrus,Mossy cells,Spike-timing	32
20200810115532	無	基礎	Gene Expression Profiles in Parvalbumin+ and Somatostatin+ Interneurons	Ms. Tzu-Hsuan Huang	黃子瑄(1)，蕭巧婉(1,2)，林宜賢(3)，吳品柔(3)，陳曜中(3)，陳卓逸(3)，鄭茵若(1)	Tzu-Hsuan Huang(1), Chiao-Wan Hsiao(1,2), Yi-Sian Lin(3), Pin-Jou Wu(3), Yao-Chung Chen(3), Cho-Yi Chen(3), Irene Han-Juo Cheng(1)	1, Institute of Brain Science, National Yang-Ming University, Taipei, Taiwan 2, Taiwan International Graduate Program in Molecular Medicine, National Yang-Ming University and Academia Sinica, Taipei, Taiwan 3, Institute of Biomedical Informatics, National Yang-Ming University, Taipei, Taiwan	RiboTag,Parvalbumin-positive interneurons,Somatostatin-positive interneurons,Hippocampus	33
20200810173212	台灣基礎神經科學學會	基礎	Function of Fringe localized Golgi outposts in dendrite arborization of neuron	Dr. Hsun Li		Hsun Li, Hsin-Ho Sung, Ying-Ju Cheng, Hai-wei Pi, Cheng-Ting Chien	Institute of Molecular Biology, Academia Sinica	Golgi outpost,Fringe,dendrite	34
20200810160617	台灣基礎神經科學學會	基礎	Galectins Crouching tiger and Hidden dragon function through N-glycosylated Draper/Ced-1 receptor in neuronal pruning	Dr. Hsin-Ho Sung		Hsin-Ho Sung1, Hsun Li1, Yi-Chun Huang1, Yu-Ju Peng2, Hsien-Ya Lin2, Chih-Hsuan Yeh2, Shu-Yu Lin2, Chuan-Fa Chang3,4, Chun-Hung Lin2, Khoong Hong Khoo2, Cheng-Ting Chien*1,5	1Institute of Molecular Biology, Academia Sinica, Taipei, Taiwan 2Institute of Biological Chemistry, Academia Sinica, Taipei, Taiwan 3Department of Medical Laboratory Science and Biotechnology, College of Medicine, National Cheng Kung University, Tainan, Taiwan 4Center of Infectious Disease and Signaling Research, National Cheng Kung University, Tainan, Taiwan,	Galectin,neuronal pruning,Draper/Ced-1,N-glycosylation	35
20200727131315	台灣基礎神經科學學會	基礎	Systematic investigation of gamma-TuRC function in cerebral cortical development	Dr. Jia-Long Chen	陳嘉隆，魏妊亘	Jia-Long Chen and Jen-Hsuan Wei	Institute of Molecular Biology, Academia Sinica, Nankang, Taipei, Taiwan	Gamma-TuRC,Development,Neuron,,	36

20200721101554	台灣基礎神經科學學會	基礎	An optogenetic approach to examine the effect of Ran GTPase in regulating non-centrosomal microtubules in neurons	Prof. 黃兆祺		Chih-Hsuan Hsu, Yung-An Huang, Ho-Chieh Chiu, Chris T. Ho, Wei-Lun Lo, Eric Hwang	Department of Biological Science and Technology, National Chiao Tung University	Growth cone-like waves,Optogenetics,Neuronal development,+TIPs,	37
20200806060411	台灣認知神經科學學會	基礎	Differences between family- vs. individual-level processing of objects: a cross-site fMRI study	Ms. Meiselina Irmayanti Abdul	度米蒂	Meiselina Irmayanti	Principles and Implication of Mind Sciences, National Cheng Kung University, Tainan, Taiwan	Ziggerins,Multi Voxel Pattern Analysis,Ventral Occipital Cortex,Medial Occipital Cortex,PsychoPhysiological Interaction	38
20200810201940	無	基礎	Using ERP to Measure Learners' Extraneous Cognitive Load During the Simple Mathematics Addition Task	Prof. Tzu-Hua Wang		Chao-Chih Wang, Peter Kuan-Hao Cheng, Wei-Jun Liao, Sih-Yu Huang and Tzu-Hua Wang	Research Center for Education and Mind Sciences, National Tsing Hua University	cognitive load,extraneous cognitive load,even-related potential	39
20200809230313	台灣基礎神經科學學會	基礎	Chronic Elevation of Indoxyl Sulfate Causes Glutamate Uptake Impairment via Aryl Hydrocarbon Receptor in Chronic Kidney Disease Mouse Brain	Mr. 黃昱傑	黃昱傑, 盧佳琛, 林惠菁, 許珮蒨, 唐德成, 李怡萱	Yu-Jie Huang, Chia-Jing Lu, Hui-Ching Lin, Pei-Chien Hsu, Der-Cherng Tarng, Yi-Hsuan Lee	1. Department and Institute of Physiology, National Yang-Ming University, Taipei, Taiwan 2. Brain Research Center, National Yang-Ming University, Taipei, Taiwan 3. Division of Nephrology, Taipei Tzu Chi Hospital, Buddhist Tzu Chi Medical Foundation, Taipei, Taiwan 4. Institute of Clinical Medicine, National Yang-Ming University, Taipei, Taiwan 5. Division of Nephrology, Department of Medicine and Immunology Research Centre, Taipei Veterans General Hospital, Taipei, Taiwan	Aryl Hydrocarbon Receptor,Chronic Kidney Disease,Glutamate transporter 1,Cognitive impairment	40
	台灣基礎神經科學學會	基礎	The functional role of post-translational modification in ASIC4	Mr. Ya-Chih Chien	簡雅致, 林星宏, 陳志成	Ya-Chih Chien, Shing-Hong Lin, Chih-Cheng Chen	Institute of Biomedical Sciences, Academia Sinica, Taipei, Taiwan	Anxiety, ASIC1a, ASIC4	41
20200810111552	台灣基礎神經科學學會	基礎	A remote-control mechanism for sensing pHo in TALK1 channels	Mr. Wen-Hao Tsai	蔡文豪 和 楊世斌	Wen-Hao Tsai and Shi-Bing Yang	1Taiwan International Graduate Program in Molecular Medicine, National Yang-Ming University and Academia Sinica, Taipei, Taiwan 2Institute of Clinical Medicine, National Yang-Ming University, Taipei, Taiwan 3Institute of Biotechnology in Medicine, National Yang-Ming University, Taipei, Taiwan	Channel,K2P,diabetes	42
20200809210709	台灣基礎神經科學學會	基礎	The Genetic Mapping of Kir6.2 in Whole-Body Expression Pattern	Ms. Athena Hsu Li		Athena H. Li, Shi-Bing Yang	Institute of Biomedical Sciences, Academia Sinica Taiwan International Graduate Program in Interdisciplinary Neuroscience, National Yang Ming University and Academia Sinica	Kir6.2,KCNJ11,genetic mapping,energy homeostasis	43
20200806151936	台灣基礎神經科學學會	基礎	Investigation of Novel Long Noncoding RNA Litchi Regulating Spinal Activities during Development	Mr. 徐聖平		Ho-Chiang Hsu, Sheng-Ping Hsu, Ting-Yu Kuo, Ya-Lin Lu, Joye Li, Jui-Hung Hung and Jun-An Chen	Institute of Molecular Biology, Academia Sinica, Taipei, Taiwan	spinal cord,long non-coding RNA,motor neuron development,neurite outgrowth,calcium imaging	44
20200729132941	無	基礎	miR-34/449 mediates precise interneuron assembly to exert proper core sensory-to-motor spinal network	Ms. Shih-Hsin Chang		Shih-Hsin Chang, Yi-Ching Su, Mien Chang, Ya-Yin Tsai, and Jun-An Chen	TIGP-INS	miR-34/449,Satb2 interneurons,Sensory	45
20200808164401	台灣計算神經科學學會	基礎	Analysis of brain images of Drosophila melanogaster acquired by x-ray synchrotron	Mr. 強敬哲		Ching-Che Charng, Ting-Yuan Wang, Nan-Yow Chen, Chao-Chun Chuang, ChunChung Chen, Chi-Tin Shih, Ting-Kuo Lee, Chung-Chuan Lo	Institute of Systems Neuroscience, National Tsing Hua University	A Drosophila connectome,AXON,neuronal classification,neuronal bundles	46
20200810154551	無	基礎	A Novel Genetic X-Ray CT Mapping of Animal Brain	Dr. An-Lun Chin		An-Lun Chin, # Yu-Han Hsieh, Yeukuang Hwu,* Ann-Shyn Chiang	Brain Research Center, National Tsing Hua University	X-ray computed tomography,Drosophila,brain imaging	47
20200731103332	台灣基礎神經科學學會	基礎	Constructing Neuron-neuron Interaction Graph from Calcium Imaging Data	Dr. 蔡郁偉	蔡郁偉、陳瑋鑫、陳建璋、施純傑	Yu-Wei Tsay 1, Chien-Chang Chen 2, Wei-Hsin Chen 2, Arthur Chun-Chieh Shih 1	1. Institute of Information Sciences, Academia Sinica, Taipei, Taiwan 2. Institute of Biomedical Science, Academia Sinica, Taipei, Taiwan	Calcium Imaging,Mini Microscope,Paraventricular Thalamus	48
20200730154552	台灣基礎神經科學學會	基礎	Identify the role of hippocampus plays in fear memory retrieval during sleep through calcium imaging	Mr. 張晉源		Ching-Yuan Chang, Ting-Yen Lee, Wan-Ting Liao, Yi-Tse Hsiao	Department of Veterinary Medicine, School of Veterinary Medicine, National Taiwan University, Taipei, Taiwan	Calcium imaging,Hippocampus,Miniscope,Fear memory	49

投稿學會：台灣基礎神經科學學會 | 編號:1

Effects of dim light at night on metabolic status and intestinal gene expressions in mice

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Abstract

Environmental light modulates physiological functions of mammals via intrinsically photosensitive retinal ganglion cells (ipRGCs) in retina and their projections to the hypothalamus. Since the electric lights were invented in 19th century, culture of human being has improved tremendously. However, concern about artificial light during night time has been raised that these kind of anomalous light information may cause disruption on circadian rhythms and furthermore result in several metabolic disorders, such as obesity and hyperglycemia. On the other hand, the exact mechanism behind how these metabolic diseases are brought about under dim light at night (dLAN) condition remain unknown. In this study, we analyzed the transcriptome of intestinal epithelial cells from the mice kept in dLAN environment and identified several potential genes, such as *Mmp10*, *Nfil3* and *Nod2*, which may account for dLAN-induced metabolic disorders. Moreover, when sympathetic nerve system was eliminated by 6-OHDA, we found that the changes of *Mmp10* and *Nfil3* expressions and obesity raised by dLAN vanished. Together, we could suggest that dLAN may alter the expressions of these genes in small intestine and lead to obesity and hyperglycemia, providing a novel insight for future research in metabolic disorders induced by aberrant luminance information.

投稿學會：台灣基礎神經科學學會 | 編號:2

Light modulates oxytocin release and sociosexual behavior in mice through ipRGCs

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Abstract

Over the past decades, oxytocin has been extensively studied for its complex role as a neurohormone that modulates wide aspects of social behavior and conventional physiological functions. In addition to its correlation with stress, how and whether environmental stimulation influence oxytocin release in the plasma and cerebrospinal fluid remain unclear. In the present study, we investigate if light stimuli could influence oxytocin and regulate the downstream sociosexual behavior with mice. We confirmed that a one-hour light pulse can reduce the concentration of oxytocin in the blood. Furthermore, light exposure could also reduce the sociosexual behavior in WT mice, but not in ipRGC genetically eliminated mice. Although the circuit which relays the light signal to regulate the oxytocin release is still under investigation, our results suggest that light can influence the oxytocin level and potentially modulate social behaviors.

Keywords

ipRGC, oxytocin, sociosexual behavior.

投稿學會：台灣基礎神經科學學會 | 編號:3

In vivo neuron activity study of the suprachiasmatic nucleus through gradient-index lenses: a novel aspect of the mammalian circadian rhythms

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Abstract

The suprachiasmatic nucleus (SCN) in mammal generates spontaneous daily oscillation and lesion of the SCN severely interrupts the rhythmicity of animals. The characters of and computation through SCN neurons have been studied for more than two decades. However, previous *ex vivo* SCN studies showed inconsistency results when different tissue preparation methods were applied, indicates that intact SCN connection is necessary for its normal operation. To study neuron activities of SCN in live animals, we combined GCaMP6s calcium imaging and gradient-index (GRIN) lens implantation techniques that minimized the interference to the SCN structure. Our results exhibited distinct cell clustering patterns in SCN with and without acute light stimulus to retinae, demonstrating a novel aspect of SCN network study and mammalian circadian rhythms.

投稿學會：台灣基礎神經科學學會 | 編號:4

Light Pattern is Important for Circadian Photoentrainment

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Abstract

In mammals, the central circadian pacemaker suprachiasmatic nucleus (SCN) in the hypothalamus plays an important role to synchronize the peripheral clocks in a roughly 24-hour cycle and influence many physiological functions such as sleep-wake cycle. In addition, the SCN receives environmental luminance information from the intrinsically photosensitive retinal ganglion cells (ipRGCs) to mediate circadian photoentrainment. However, the neural connection from ipRGCs to the SCN and the photoentrainment mechanism remain poorly understood. Anatomical studies showed that the ventrolateral part of the SCN (core region) is the primary recipient region photic input. Intriguingly, recent studies showed that the dorsal part of the SCN (shell region) may receive synaptic inputs from ipRGCs directly despite the physiological function is still unclear. Using different light pattern and c-Fos staining, here we showed that ipRGCs may innervate and activate the dorsal and ventral region of the SCN neurons separately. Furthermore, light from above could generate larger phase shift response than light from below under same luminance. These results indicate the possibility of distinct populations of SCN neurons which could be activated by different light pattern, and lead to different endogenous circadian phase response.

投稿學會：台灣基礎神經科學學會 | 編號:5

Light-Dark Cycle Mediates Diurnal Oscillatory Rhythms in Gut Microbiota

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Abstract

Nearly all tissue in mammals can adapt to changing light in the environment, even the microbiome in the gut, where isn't directly received the environmental light dark cycle. Recently studies have show that the deletion of Bmal1 abolished rhythmicity in the fecal microbiota composition, which means the host circadian clock has responsibility with it. However, how does the circadian clock modulate the rhythmicity of gut microbiota remain unclear. Using genetic mouse models and modulate different light-dark cycle, here we showed that tissue specific deletion of Bmal1 cannot disrupted the oscillation of gut microbome. However, its oscillating microbome are partially different from the control group.

投稿學會：台灣基礎神經科學學會 | 編號:6

Investigation of daily and tidal behavioral rhythm in Shuttles hoppfish (*Periophthalmus modestus*)

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Abstract

Most organism used internal circadian clock to regulate various physiological functions throughout the daily light dark cycle. However, animals live in intertidal zone not only experience daily light-dark cycles, they also face rapid and severe environmental changes such as salinity or water level fluctuation caused by tidal cycle. Although some circatidal rhythmic behaviors have been observed in many intertidal invertebrates, whether there is a universal driving mechanism for circa-tidal rhythm remains unclear. As a unique amphibious vertebrate inhabits intertidal zone, mudskippers have been found to have different activity levels and regulate ion channels phenotype in osmoregulatory organs between high and low tide. Therefore, they are suitable for the circatidal study. Here, we recorded and analyzed the activity of shuttles hoppfish (*Periophthalmus modestus*) under constant dark and 12:12 LD cycles with constant fresh water. We found potential close to 24 hour and 12 hour behavior in traveling distance and staying preference on land or water respectively. Such preliminary experiments prefaced the research of behavioral rhythm in intertidal vertebrates.

投稿學會：台灣基礎神經科學學會 | 編號:7

The impact of early-life antibiotic exposure on the neurodevelopmental outcomes and gut microbiota development in mice

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Abstract

Preterm infants develop incomplete neural development, immature immune system, and altered gut microbiome. Several studies indicated that preterm infants have a high prevalence of developing autism spectrum disorder (ASD). Clinically, the administration of antibiotics in preterm infants is a common practice to combat bacterial infections. However, whether the antibiotics exposure at the early-life stage leads to neurodevelopmental disorders in a direct manner is still unclear. We hypothesize that early-life antibiotic exposure in infants is a major risk factor for adverse neurobehavioral outcomes associated with the dysbiosis of gut microbiota. To characterize the neurobehaviors in mice after exposure to antibiotics at the early-life stage, we treated first-line antibiotics widely used clinically in mice at the post-weaning period and test for their ASD-like behaviors. We found that the antibiotics-treated group displayed the decrease trending of anxiety-like behavior, increase of sociability, and decrease of social novelty. To further test whether peripubertal stress produces environment-environment interaction worsen the neurodevelopmental behavior outcomes, *we* challenged the antibiotics-treated mice with sleep deprivation during the peripubertal stage and test for behaviors at the adult stage. Interestingly, sleep deprivation increased the anxiety-like behavior in antibiotics-treated mice but did not produce an effect on the increase of sociability and decrease of social novelty found in antibiotics-treated mice. To understand whether the antibiotics and sleep deprivation challenges alter the gut microbiota composition, we collected the fecal samples from mice and analyzed the microbiota composition in fecal DNA by qPCR. The result showed that the relative abundance of major phyla shifted in antibiotics and sleep deprivation groups. Specifically, we found that early-life antibiotics exposure decreased *Firmicutes* and increased *Bacteroidetes* at the phyla level. Co-administered antibiotics and sleep deprivation led similar shifts in microbiome composition at the phyla level but increased in *Actinobacteria*. Furthermore, we also detected the changes in *Bifidobacterium* at the genus level, *Lactobacillus reuteri*, *Segmented filamentous bacteria,* and *Akkermansia muciniphila* at the species level. These results strongly suggest that early-life antibiotic exposure impacts neurobehavior outcomes and microbiota development.

投稿學會：台灣基礎神經科學學會 | 編號:8

The level of COUP-TFI governs the septo-temporal region patterning during hippocampal development

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Abstract

While the hippocampus is well-known for its function on cognitive processes such as spatial learning, it is also involved in interoceptive behaviors, including fear and anxiety. The hippocampus can be divided into two functional domains along the septo-temporal axis: the septal hippocampus consists of many place cells and processes the spatial representation, while the temporal hippocampus is composed of abundant anxiety cells for the anxiety and fear memory. It remains mostly unknown how to establish these functionally distinct domains during hippocampal development. COUP-TFI (chick ovalbumin upstream transcription factor I, or NR2F1) has been proposed to play a key role in hippocampal growth, as deleting COUP-TFI leads to significant hippocampal hypoplasia. Here, I defined the function of COUP-TFI for hippocampus patterning by altering COUP-TFI expression levels during cortical development. By comparing the hippocampal cytoarchitecture among wild type (WT), COUP-TFI-conditional knockout (cKO) and COUP-TFI-conditional transgenic mice (cTG), I showed that hippocampal volume was greatly reduced in the cKO, while the hippocampus was expanded in the COUP-TFI-cTG mice. This finding suggested that COUP-TFI expression level correlates with hippocampal neuronal numbers. To demonstrate this link, I performed EdU birthdating analyses to trace neuronal production at different developmental stages and found COUP-TFI overexpression increased hippocampal neuronal production at E13.5 and E15.5. I further analyzed CA1 neuronal compositions and hippocampal regional makers and demonstrated that the hippocampus is septalized in the cKO and is temporalized in the cTG. Further, the overexpression of COUP-TFI impaired CA1 pyramidal layer organization. I am currently exploring the molecular mechanism underlying COUP-TFI regulating hippocampal patterning along the septo-temporal axis and examining the outcome of the overgrowth and disorganization of the CA1 region in COUP-TFI-cTG mice.

投稿學會： 無	編號:9

COUP-TFI specifies entorhinal cortex and determines the location and integrity of its border through cell affinity mechanisms

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Abstract

The mammalian cerebral cortex is a remarkably complex organ, which is comprised of several cortical regions. Each cortical region has distinct unique cytoarchitecture, connections and functions. As most of the cortical projection neurons are derived from progenitors in the dorsal telencephalon, of the Emx1 lineage, it is interesting to investigate how distinct neuronal fates arise from the relatively uniform progenitor population. Orphan nuclear receptor COUP-TFI is expressed in cortical progenitors with a high caudo-lateral to low rostro-medial gradient during cortical development, and its highest expression level is detected where entorhinal cortex (EC) presumably originates. We found lowering COUP-TFI expression in the cortical progenitor of the *Emx1*-lineage decreased the size of EC while complementarily increased the size of neocortex (NC). Additionally, COUP-TFI overexpression rostrally expanded the EC, and generated ectopic EC-like structures, with similar laminar architecture and connectivity as endogenous EC, within the caudal NC. Using *in utero* electroporation to overexpress COUP-TFI in NC revealed that high level of COUP-TFI is able to cell-autonomously re-specify NC cells to adopt EC fate and induce cell clustering. Moreover, we used a new mathematical model to simulate the phenotypes observed in COUP-TFI mutant mice, and discovered that differential cell affinity plays a key role in the emergence of ectopic domains and supporting boundary regularity. Experimentally, we demonstrated that NC and EC cells have differential cell affinities. Further, we identified that Pcdh19, a cell adhesion molecule that shows a high caudal to low rostral expression gradient, is critical for the ability of COUP-TFI to induce cell clustering. In summary, we discovered that the patterning transcription factor COUP-TFI plays a critical role in EC fate specification and controls the location and integrity of NC/EC border through cell affinity mechanisms.

投稿學會： 無	編號:10

Lhx2 regulates cortical neuronal excitability to maintain thalamus development

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Abstract

To understand mechanisms regulating neuronal circuit formation during development, we study the development of barrel cortex, the rodent primary somatosensory cortex. Barrel cortex is a topographic map for processing stimuli from the whisker hairs, where each barrel is a neuronal module with axonal inputs coming from the ventral posteromedial nucleus (VPM) of the thalamus and recipient cortical neurons in the layer 4 (L4). With thalamocortical axon (TCA) as a presynaptic component and the L4 neurons as a postsynaptic component, we focus on how postsynaptic neurons feedback to regulate the development of presynaptic neurons. While previous studies had clearly demonstrated the impact of thalamocortical inputs on the development of somatosensory cortex, whether cortical neurons regulate thalamus development remains relatively unknown. Here we showed that Lhx2, a LIM homeodomain containing transcription factor, functions in the L4 neurons to regulate TCA arborization and maintain the structure of VPM. Further, deleting Lhx2 in cortical neurons showed functional defects in whisker-evoked responses, as well as spontaneous activities, in the developing barrel cortex. Mechanistically, we found that Lhx2 regulates the excitability of cortical neurons, in turn to influence TCA arborization and thalamus development.

Key word: Lhx2, barrel cortex, thalamus, neuron excitability

投稿學會：無	編號:11

Contribution of Dbx1 lineage cells to the Piriform Cortex

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Abstract

Piriform cortex (PC), the major olfactory cortex, consists of neurons derived from non-localized and disparate source origins. It is still unknown how distinct neuronal lineages contribute to PC. During cortical development, Dbx1 is expressed in the progenitors located in the pallial subpallial boundary (PSB) and preoptic area (POA). It was previously shown that many neurons from the Dbx1 lineage are located in the PC. Here, we used Dbx1-Cre to turn on the expression of reporter genes to investigate the birth-date and postnatal distribution of Dbx1-derived cells in the PC, their morphological landscape and output projections to the orbitofrontal cortex (OBC). We first-used the GAD67-GFP interneuron reporter line to distinguish the projection neurons and interneurons derived from Dbx1-lineage. Dbx1-derived interneuron population was only 36% with a majority of this population being located in the ventral region in layer 3. The projection neurons of the Dbx1 lineage are over-represented in layer 2 and have a high preference for the ventral relative to the dorsal PC in their distribution. Further, the peak of Dbx1 lineage cells were generated early at E11.5, where ventral Dbx1 cell-generation exceeded dorsal cell-generation by more than 3-fold. We then investigated the morphology of Dbx1-derived cells in the PC. Interestingly, Dbx1-lineage cells adopted the majority of described cell morphologies in the PC, including pyramidal, semilunar, interneuron, multipolar and bifuted. These findings suggest that Dbx1 lineage contributes to early born neurons in the PC, although Dbx1-lineage cells do not show cell type (morphology) preferences, they show a ventral-caudal preference in distribution. Lastly, we traced the output projections of the Dbx1 lineage and found that many of the ventrally located Dbx1 cells project to the orbitofrontal cortex (OFC). Collectively, our results show a difference of Dbx1 lineage cells located in dorsal and ventral PC in their distribution, timing of neurogenesis, and output projections and imply that these neurons derived from the Dbx1 lineage might serve specific functions in PC.

投稿學會：台灣基礎神經科學學會 | 編號:12

Generating hiPSC-derived cerebral organoids to model human brain development and primary microcephaly

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Abstract

Centrosome acts as a major microtubule organizing center (MTOC) in mammalian cells and plays an essential role on microtubule-related events, such as cell division and intracellular transport. Centrosome dysfunction has been linked to cancer and autosomal recessive primary microcephaly (MCPH). MCPH is a human neurodevelopmental disorder characterized by small brain size with mild to severe mental retardation. Interestingly, most MCPH gene-encoding proteins localize at the centrosomes, implying the centrosomal role of MCPH proteins in developing human brain, yet the underlining mechanism is unclear. Recent studies have identified a new type of neural stem cells (oRG, outer radial glia cells) in the developing neocortex of primates/humans, but generally not found in rodent brains. To study the centrosomal role of microcephaly protein CPAP that participates in neural stem cell division, particularly oRG cells, we have successfully established hiPSC-derived cerebral organoid as a model. Our results showed that hiPSCs-derived cerebral organoid can recapitulate many cellular events seen in developing human brain, including interkinetic nuclear migration (INM), neuronal cell differentiation, and the formation of cortical layers and oRG cells. Recently, a homozygous missense mutation (E1235V) was identified in the *CPAP/CENPJ* gene that causes primary microcephaly in humans. Using the CRISPR/Cas9 gene editing system, we have generated the disease-associated hiPSCs that carried the CPAP-E1235V mutation. Our results showed that hiPSC-CPAP-E1235V produces microcephalic brain comparing with control hiPSC-derived organoids. Importantly, hiPSC-CPAP-E1235V-derived brain organoids produce less number of oRG cells ($HOPX^+$, $SOX2^+$) than control brain organoids. Here, we demonstrate that the hiPSC-derived brain organoid coupling with the CRISPR/Cas9 gene editing technology was able to model human microcephaly and study the behavior of oRG cells during human brain development.

投稿學會：台灣基礎神經科學學會 | 編號:13

Investigating the roles of centriolar protein Cep120 during cerebellar development

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Abstract

The development of cerebellum requires the primary cilium, a microtubule-based organelle, for Sonic Hedgehog (SHH)-triggered proliferation of cerebellar granule neuron progenitor cells (GNPs). Loss of the primary cilium leads to an underdeveloped cerebellum, such as Joubert syndrome, a devastating ciliopathy represented as cerebellar hypoplasia. Hitherto, mutations in 35 genes have been identified in Joubert syndrome, including CEP120, a centriolar protein located at the basal body of cilia. Here, we took advantage of multiple modalities to study the roles of Cep120 during murine cerebellar development, including the *in vivo* cerebellar electroporation, *in vitro* GNP purification, and *ex vivo* organotypic slice culture for live cell imaging. We showed that loss of Cep120 not only resulted in a decrease of ciliated GNPs *in vivo* and *in vitro*, but to our surprise, it is required for the neuronal distribution toward the internal granular layer (IGL), the destination for GNPs. Knockdown Cep120 resulted in producing ectopic neurons possibly through a block of cell-cycle progression, thereby hindering the cell-cycle exit. This cellular mechanism illustrates that Cep120 is required for neuronal differentiation in advance of the radial migration in GNPs. Importantly, the replenish of wild-type CEP120 mitigated above defects, while the supplement of disease-associated CEP120 mutants still impeded the neuronal distribution *in vivo*. This finding suggests that the ectopic neuron frequently found in Joubert syndrome may result from the delay of neuronal maturation. Next, we screened and examined the roles of a number of Cep120-interacting proteins implicated in Joubert syndrome. Our findings confirm and extend the current understanding on our previously identified “the Cpap-Cep120-Talpid3/c2cd3 axis” for ciliogenesis in GNPs. Taken together, our data reveal that Cep120 regulates the differentiation of GNPs in cerebellar development, while the mutations on CEP120 may implicate in the pathogenesis of ectopic neurons observed in Joubert syndrome.

投稿學會：台灣基礎神經科學學會 | 編號:14

Cap Methyltransferase 2 (CMTR2) is required for cerebellar development and function

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Abstract

The 5′-end of mRNAs is capped with 7-methylguanosine (m7G) that is important for the stability, nuclear export and translation of mRNAs. This modification, known as cap or cap0 (m7GpppNpNp, N: any nucleotide), is catalyzed by RNA guanine-7 methyltransferase (RNMT) in all eukaryotic organisms. In some viruses and higher eukaryotes, further modifications occur to produce cap1 (m7GpppNmNp) and cap2 (m7GpppNmNm) structures by adding a methyl group at the 2′ position of ribose of the first (N1) and second (N2) nucleotides by cap methyltransferase 1 (CMTR1) and CMTR2, respectively. Although both N1 and N2 2′-O-ribose methylation (2′-O-Me) were discovered more than 40 years ago, their molecular and physiological functions remain undetermined, especially for CMTR2-catalyzed N2 2′-O-Me i.e., cap2). Thus, we generated *Cmtr2*-knockout (KO) mice. The KO mice die before embryonic day 11.5, so we generated mice with conditional knockout of *Cmtr2* (*Cmtr2*-cKO$^{Nestin\text{-}Cre}$) in the nervous system to investigate CMTR2 function. *Cmtr2*-cKO mice were born with similar body weight compared to their wild-type littermates but they showed postnatal growth retardation and motor dysfunction with cerebellar atrophy as they age. Our preliminary results indicate that reduced dendritic growth of Purkinje cells and thinner molecular layer were found in *Cmtr2*-cKO cerebellum. We also observed increasing axonal swellings (torpedoes/axonal varicosities) in Purkinje neurons of adult cKO mice from 2- to 5-month-old. Using the rotarod assay to examine motor learning and coordination, we found that cKO mice exhibited impaired motor coordination at higher speeds (30 and 40 rpm) at~ 4-month-old and afterward. Moreover, cKO mice also walked abnormally in a beam walk test. These findings suggest that the motor coordination is defected in cKO mice. Whether and how CMTR2-catalyzed N2 2′-O-Me in mRNAs affects posttranscriptional gene expression to regulate cerebellar development and function require further investigation.

投稿學會：臺灣基礎神經科學學會 | 編號:15

Knockout of a Splicing Regulator Causes Cerebellar Vermis Hypoplasia

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Abstract

The cerebellum has diverse functional roles from motor control to cognition. The splicing regulator RBM4 modulates neuronal cell differentiation and migration via its role in alternative splicing. Conventional *Rbm4* gene double knockout (dKO) mice were, however, alive and fertile. Nevertheless, the cerebella of *Rbm4*-dKO mice exhibited profound hypoplasia of vermian lobules VI-VII, which is a characteristic feature of juvenile autism spectrum disorder. Open-field test showed *Rbm4*-dKO mice exhibited hyperactive exploratory behavior. In the developing cerebellum, we observed that granular layer formation was delayed and dendritic arborization of Purkinje cells was impaired in *Rbm4*-dKO cerebella. Surprisingly, *Rbm4* knockout enhanced optokinetic response adaptation of mice, indicating improved motor learning. By examining the RBM4-associated mRNAs in brain, we identified several target candidates with a role in synaptic function; verification of RBM4 targets is currently underway. In addition, identification of splicing alteration in the *Rbm4*-dKO cerebellum is also on-going. This study provides a hint that RBM4 is critical for cerebellar foliation and perhaps synaptic function via its role in alternative splicing regulation.

投稿學會：台灣基礎神經科學學會 | 編號:16

Dopamine D2 receptor antagonist trifluoperazine declines HFD-induced inflammation and gliosis

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Abstract

Obesity is a critical health problem in recent society, which leads to several diseases, such as diabetes, hypertension, cancer and neural degenerative diseases. Peripheral inflammation and neuroinflammation are common symptoms in obese patients and obese animal models, which involves in the pathogenesis of obesity. Limit of neuroinflammation in hypothalamus have been reported to rescue high fat diet (HFD) -induced weight gain and abnormal metabolic index. Dopamine and its receptors participate in immune system and regulate inflammation. Trifluoperazine (TFP), an antagonist of dopamine D2 receptor (D2R), which is used to treat symptoms of schizophrenia. Here, we found the D2R mRNA expression was upregulated after lipopolysaccharide (LPS, 10 ng/ml) administration for 6 hours, and impaired by TFP (2 mg/kg) treatment in mouse hypothalamus. The increasing levels of D2R was found in several brain regions of obese mice with chronic HFD feeding. Moreover, TFP had anti-inflammatory effects on LPS -induced inflammation in plasma and hypothalamus. Chronic HFD-induced plasma inflammation and gliosis in hypothalamus were also declined after daily TFP administration for 1 month via intraperitoneal injection. We also found 1 month-TFP injection decreased chronic HFD-induced hyperglycemia, whereas TFP administration did not change the body weights of the obese mice. According to our results, TFP has the ability to suppress HFD-induced hyperglycemia, inflammation and gliosis in hypothalamus, but not able to improve HFD-induced body weight gain and other metabolic index.

投稿學會：台灣基礎神經科學學會 | 編號:17

The detrimental impacts of proteasome over-activation on acute intracerebral hemorrhage in rats

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Abstract

Objective: Misfolded proteins in the endoplasmic reticulum (ER) initiate unfolded protein response (UPR) to restore protein homeostasis. Studies have revealed the connections at multiple levels between UPR and inflammation. Neuroinflammation is a hallmark in intracerebral hemorrhage (ICH). In various neurological disorder, ER stress-triggered apoptosis and proteostasis disruption which plays an important role in causing neuroinflammation, but was not studied during ICH development.

Materials and Methods: ICH was induced by collagenase VII-S intrastriatal infusion. Rats were determined for body weight changes, hematoma volume, and neurological deficits. Brain tissues were harvested for molecular signaling analysis either for ELISA, immunoblotting, immunoprecipitation, RT-qPCR, protein aggregation, or histological examination. A non-selective proteasome inhibitor, MG132, was administered into the right striatum three hours prior to ICH induction.

Results: Our results demonstrated that ICH-induced acute proteasome over-activation causing proteostasis disturbance and neuroinflammation. The ER stress-associated GRP78 and inhibitory IκB protein was rapidly degraded at 3 hours after ICH accompanied by the elevation of pro-apoptotic CCAAT-enhancer-binding protein homologous protein (CHOP) protein expression and pro-inflammatory cytokines expression via NFκB signal activation. These detrimental impacts of the ICH-induced over-activation of the proteasome were abolished by proteasomal inhibition, exerting neuroprotective effects against ICH-induced ER stress/proteostasis disruption, pro-inflammatory cytokines, neuronal cells apoptosis, and neurological deficits.

投稿學會：台灣基礎神經科學學會 | 編號:18

Sleep deprivation exacerbates neuroinflammation and impairs hippocampal neurogenesis in heterozygous Disc1 mutant mice

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Abstract

Sleep/circadian rhythm disturbances are considered as environmental stress factors that might interact with genetic risk factors and contribute to the pathogenesis of psychiatric disorders. In this study, we evaluated the impact of 72-hour sleep deprivation (SD) on heterozygous Disc1 mutant (Disc1 Het) mice. We observed harmful consequences of 72-hour SD, including delayed maturation of newly generated dentate gyrus neurons and upregulation of proinflammatory cytokines, in both wild-type (WT) and Disc1 Het mice. However, the effects of SD seemed to be more deleterious in Disc1 Het mice. Increased microglial density and reduced neural proliferative activity were found in neurogenic niche of Het-SD mice. Interestingly, SD-induced Bdnf mRNA elevations were evident in both WT and Het mice, while only in WT-SD mice did we observe increased BDNF protein expression. Together, the present study demonstrated that sleep disturbance could be pathogenic especially in genetically predisposed subjects; SD deteriorates hippocampal neurogenesis and exacerbates inflammatory conditions in a Disc1 haploinsufficiency model. Our results propose an SD-induced stress-coping reaction featured by the elevation of BDNF protein expression and sufficient DISC1 is required in this process. Potential therapeutic strategies for psychiatric disorders targeting the mRNA translation machinery could be considered.

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The role of CCL5 in hippocampal memory function and antioxidant activation after mild TBI

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Abstract

Traumatic brain injury (TBI) is common head injury worldwide which increased the risk of neurodegeneration disease such as Alzheimer's disease (AD) and Parkinson's disease (PD). Increased reactive oxygen species (ROS) and inflammation chemokines after TBI induced serial secondary effects to damage neurons. Studies showed that C-C motif chemokine ligand 5 (CCL5) has neurotropic functions as promoting neuritis outgrowth and anti-apoptosis. CCL5 level in blood associates to the severity of TBI patients, but the function of CCL5 after brain injury is largely unknown. In the current study, study induced mild brain injury in C57B/6 (wildtype, WT) mice and CCL5 knockout (CCL5-KO) mice by a weight-drop model. The cognitive and memory function in mice were analyzed by Novel-object-recognition and Barnes Maze. The memory performance of both WT and KO mice were impaired after mild injury. The cognition and memory function in WT mice were quickly recovered after 7 days but which took more than 14 days CCL5-KO mice. FJC, NeuN and Hypoxyprobe staining revealed high amounts of neurons damaged by oxidative stress in CCL5-KO mice after mTBI. NADPH oxidase activity showed increased reactive-oxidative-species (ROS) generation with reduced GPX1 protein and GSH activity in CCL5-KO mice which was opposite in WT mice. CCL5 increased GPX1 expression and reduced intracellular ROS level which increased cell survival in both primary neuron culture system and overexpression model in SHSY5Y cell line. The memory impairment in CCL-KO mice induced by TBI could be successfully rescue by i.p. GSH precursor – NAC into mice after injury. In summary, CCL5 is an important factor for GPX1 antioxidant activation and neuron protection during post-injury day 4~7 which protects hippocampal neuron from ROS and improves memory function after brain trauma.

Keywords: CCL5, mild traumatic brain injury, reactive oxygen species, NADPH oxidase, antioxidant

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Voluntary Exercise As a Preventative Strategy for Microglia-mediated Synaptic Pruning Defects on Sleep-deprived Adolescent Mice

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Abstract

Adolescence is a critical period for brain development and adequate sleep during this period is essential for physical function and mental health. Emerging evidence has detailed the neurological impacts of sleep insufficiency on the developing brain. Our previous study demonstrated 72 h sleep deprivation (SD) disrupts microglia-mediated synaptic refinement in the dentate gyrus of adolescent mice. Physical exercise could counteract the harmful consequences in various stress or neurodegenerative models by modulating microglia. In this study, we tested the preventive effect of physical exercise on microglial function in an adolescent SD mouse model. In this study, adolescence C57/BL6 male mice (~P23) were randomly assigned to the Sedentary (S) or the Exercise (E) group, in which mice were housed in pairs and a running wheel was placed in each cage. After 11 days of voluntary exercise, each group of mice were further divided into the 72 h Sleep Deprivation (SD) or the Normal Sleep (NS) group. All subject groups (SNS, ENS, SSD, and ESD) were subjected to a short-term memory test or sacrificed directly for further examinations. Our results demonstrated that SD-induced impairment in short-term memory and increase of neural activity index was rescued by preceding voluntary exercise (VE). Furthermore, increased dendritic spine density in the SSD group was not observed in the ESD group, implying that VE prevents SD-induced synaptic pruning defect. We also observed greater microglial phagocytic ability, characterized by increased internalized postsynaptic materials and lysosomal structure within individual microglia, in the ESD group compared with the SSD group. mRNA expression of microglia-specific receptors and their ligands critical to developmental synaptic refinement was found upregulated in both ENS and ESD groups. Here, we provided evidence featuring an effective effect of VE that significantly alleviates the SD-induced defects in short-term memory and microglia-mediated synaptic pruning. Physical exercise could be a beneficial health practice for the adolescents that copes the adverse influence of inevitable sleep insufficiency.

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Inflammatory cytokines and executive function may be correlated with outcomes of substance use disorder

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Abstract

Background: Substance abuse, such as opioid or amphetamine type stimulants, may activate proinflammatory processes by increasing cytokine production and impairing neurotrophic factor expression, and possibly leads to impairments of executive function. Therefore, we investigated whether inflammatory markers, neurotrophic expression and performance in Wisconsin card sorting test (WCST) were different between patients with substance use disorders (SUD) and healthy controls (HC), and correlated with substance use severity.

Method: We assessed plasma tumor necrosis factor (TNF)-α, C-reactive protein (CRP), interleukin (IL)-8, transforming growth factor (TGF)-β1, brain-derived neurotrophic factor (BDNF) and WCST in SUD patients (opioid or amphetamine type stimulants use disorder) and HC. SUD patients were followed for 12 weeks and their urine morphine and amphetamine tests and cytokine levels were measured at initial screen phase, week 1, 4, 8, and 12.

Results: We enrolled 479 patients and 187 HC. Plasma levels of TNF-α, CRP, IL-8, and BDNF, and most subscales in WCST were significantly different between SUD patients and HC. Plasma TNF-α, CRP, IL-8, and BDNF levels were also significantly correlated with the performance in WCST. However, only TNF-α levels and total number correct (TNC) in WCST were significantly associated with urine morphine and amphetamine positive results (P=0.02 and 0.03).

Conclusion: Higher TNF-α levels and poor WCST performance were associated with more substance use. Studies on regulating inflammatory process and enhancing executive function to improve substance use behavior might be warranted.

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The deficient Clec5a ameliorates the neuroinflammation-induced neurodegeneration in Alzheimer's disease

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Abstract

Neuroinflammation is a critical factor of Alzheimer's disease (AD), which is the most common neurodegenerative disease in elderly. The pathological hallmarks of AD, amyloid-β (Aβ) and hyper-phosphorylated tau, triggers the microglia activation to induce the neuroinflammation. Aβ deposits activate the NACHT, LRR and PYD domains-containing protein 3 (NLRP3) inflammasome in microglia, which induce the mature interleukin-1β (IL-1β) release. C-type lectin domain family 5 member A (CLEC5A) is a microglial membrane receptor and was found to regulate the inflammatory signal in virus-infected microglia. However, the activity of microglial CLEC5A against Aβ-induced neuroinflammation remains unknow. In this study, the AD mouse model was used to cross with CLEC5A knockout (KO) mice. The results showed that the AD mice with CLEC5A KO showed ameliorative memory and cognition deficits compared with pure AD mice. Meanwhile, the hippocampal Aβ deposition and IL-1β mRNA level were reduced in AD mice with CLEC5A KO. To understand the mechanism of CLEC5A in AD pathology, we used the shRNA knockdown (KD) of CLEC5A in the microglia-like cell line, BV-2 cell. The results showed the decrease of NLRP3 expression and IL-1β release in CLEC5A KD group under Aβ stimulation. However, the anti-inflammatory markers, such as YM-1 and Arg-1, were not changed in the CLEC5A KD model. The results revealed that deficient CLEC5A improved memory function in AD mice and also decrease microglial-induced inflammatory signaling. This study implied the modulation of microglial-induced inflammatory signal may be a potential treatment of AD pathology.

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Study the Effects of Zn^{2+} in Dopamine-induced Pyroptosis in Primary-cultured Rat Embryonic Cortical Neurons

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Abstract

Neuroinflammation has attracted much attention recently in nervous system for its association with the pathogenesis of neurodegeneration disorders like Alzheimer's, Huntington's, and Parkinson's diseases etc. The activation of inflammation will induce the formation of inflammasomes which activates the caspase-1 resulting in the induction of pyroptosis signaling pathway and the release of matured inflammatory cytokines including IL-1β and IL-18 into the extracellular milieu. Our previous reports have shown that dopamine (DA) elevates intracellular Zn^{2+} concentration which is a perquisite for DA-induced cell death and enhances the mRNA level of IL-1β in cultured rat embryonic cortical neurons. However, it is unclear that neuroinflammation is involved in DA-induced cell death. In this report, we treated the primary cultured rat embryonic cortical neurons with DA and dihydrexidine (DHX), a dopamine D1 receptor agonist, to induce cell death. We pretreated the cultured neurons with TPEN, a cell permeable Zn^{2+} chelator, MCC950, inflammasome blocker, and VX765, caspase 1 inhibitor; the results showed that these blockers rescued the cell death induced by dopamine and DHX. The results of PCR and Western blot showed increments in the expression levels of IL-1β in cells treated with DA or DHX. In addition, pretreating the neurons with IL-1β suppressed the DA-induced cell death. These results suggest the involvement of pyroptosis in D1 receptor-Zn^{2+}-cell death pathway and the released IL-1β enhances the cell survival. Therefore, controlling the pyroptosis and IL-1β level is a new therapeutic strategy for neurodegenerative diseases.

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From neuroscience to engineering: a spiking neural network model of dynamic vision based on fruit fly visual system

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Abstract

In recently years, the technologies of unmanned aerial vehicle (UAV) and autonomous vehicle (AV) have advanced rapidly. However, the visual-guiding systems of most of these technologies are energy intensive and are not suitable for power-limited small vehicles. Inspired by fruit fly's highly efficient visual system, we designed a neuromorphic system for dynamic vision and combined it with the spatial orientation memory system and the decision network for navigation. The first component of the system is a retina-inspired optical flow algorithm. It is improved from the classic Hassenstein-Reichardt detector model by the inclusion of spatial filters. The improvement allowed us to significantly reduce the required number of detectors. The second component is a motion-detection neural network. It is based on the concept of T4/T5 neurons in lobula plate tangential cells (LPTC) of the fruit flies. In the network, every motion-detection neuron has a preferred motion state and detects the motion state of image sensor through a neural competition mechanism. The third component is an obstacle-detection neural network. It is inspired by the LC11 neurons of the fruit flies and is able to detect small objects in the foreground. The forth component is an object-tracking network that is inspired by the fruit fly spatial orientation memory mechanism in the central complex. It can memorize the object's motion state and reduce the computation when the motion state does not change. Additionally, we developed a new neuron model which is digital hardware friendly and is easily to be implemented in spiking neural network chip. It works on integrate but possesses some properties that are exhibited by complex nonlinear neuron models. Our work demonstrates how knowledge in neuroscience can help with designing energy-efficient engineering systems.

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A coupled neural circuit and Markov process model of spatial orientation in *Drosophila melanogaster*

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Abstract

To navigate in a complex environment, an animal needs to keep track of its orientation. Many studies revealed that a bump of activity in the Drosophila ellipsoid body (EB) represented head-direction. However, how the activity bump maintains stability while constantly updates its representation is not completed understood. To address this issue, we constructed a Markov-chain behavior model and combined it with a previously developed neural circuit model of protocerebral bridge (PB) and EB. We modified the neural circuit model based on the latest electron microscopy data and performed parameter tuning. The model was used to elucidate the results of behavioral experiments on the basis of a modified Buridan's paradigm. In the experiments, roles of specific neurons in orientation working memory were observed by silencing or optogenetically activating two types of inhibitory ring neurons: EIP-ring neurons and P-ring neurons. The model suggested that the EIP-ring neurons are crucial for maintaining the shape of the activity bump in EB, while the P-ring neurons are responsible for controlling the updating process for the bump location when a fly rotates its body.

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The Advanced construction of FlyCircuit and analysis of neuron images within

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Abstract

Recent progresses in FlyCircuit database present new aspects in collecting and analyze single neuron images. A new model brain was constructed from a fly brain kept within its skull so that the physiological morphology of the brain may be disrupted minimally. The neuron registration may become more reliable and structural fidelity kept. Then, algorithms to simplify neuron morphology from 3D configuration to 1D sequence were developed, similar neurons disregard their lateralization or gender differences may be found. Similar neurons may express different genes or sexual dimorphisms. Furthermore, investigate divergence, convergence, or connectivity among neurons is possible by sequence comparisons. Even more, collective cluster algorithms are developed to analyze the global structure of neural network within the brain. The criteria for clustering may be due to the randomness of the connection or the structural similarity of neurons. Thus the neural structure and possible functional connections may be linked. A website for statistically handling massive neuron image will be constructed.

FlyCircuit 進階版及其影像分析工具

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影像資料庫未來升級將包含數項改進:首先，所用的標準腦將位於果蠅腦殼內，以降低干擾其生理狀況下之形態，如此可提高神細胞對位的形態及位置正確度。其次，我們也開發新的計算法，將三維的神經空間結構降成一維的序列，可以藉此找尋位於不同腦側或不同性別組織中的相似神經，也可找出相同神經中可能表現的不同基因。此外，神經走向的發散式匯聚也可由其空間分佈序列的比對來判讀。更進一步的分析則包含利用群聚分析法來判讀非隨機連結的神經及相似結構的神經集結，從而推斷可能的功能／結構關係。如此統計分析的工具將隨網頁上線提供研究者使用。

投稿學會：台灣計算神經科學學會 | 編號:27

Self-similarity of neurons in Strahler order analysis

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Abstract

Owing to the mass variation of neuron morphology, universal similarities among neurons are obscure. Meanwhile, it is important to examine the mutual laws of neuron growth underlying the diversiform appearance of neurons. Former research made use of the Strahler ordering system and shed light on the correlation of path length and branch number with Strahler order. The fact is also found in FlyCircuit whole data neurons, and this research steps further, we used the system as a metric to evaluate the symmetry of a neuron and investigated how the correlation between morphology features and Strahler order changes with different classifications of symmetry. Furthermore, this correlation variation can be transformed into formulas, describing branch number, path length, and the average path length of each Strahler order. To conclude, we provide a new method to classify the symmetry of neurons in a concise way.

投稿學會：台灣基礎神經科學學會 | 編號:28

Spatiotemporal dynamics of astrocytic Ca^{2+} signalling in three-dimension *in vivo*

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Abstract

Astrocytes are a major type of glial cells in the central nervous system, and they function as neuronal support by interacting with vascular system. In addition to homeostatic roles, astrocytes can also modulate neuronal functions via the release of gliotransmitters, which is controlled by astrocytic Ca^{2+} transients. Therefore, decoding astrocytic Ca^{2+} signalling is essential for full understandings of brain functions. However, the hugely divergent morphology of astrocyte makes conventional 2D imaging and analysis a source for inconsistent results. Imaging only on a single focal plane is incompetent to represent the whole cell. Here, we employ two-photon fluorescent microscopy and scanning-based time series in three-dimension to map the spatial-temporal Ca^{2+} activity in the astrocytic territory. By combining astrocyte-specific Cre line *Aldh1l1-Cre*^{ERT2}, and genetically encoded calcium indicators (GECIs), we demonstrate the three-dimensional and temporal distribution of astrocytic Ca^{2+} in the motor cortex of awake mice. The method also demonstrates the potential for studying the brain signals decoded by the dynamics of astrocytic Ca^{2+} activity in behavioral tasks.

投稿學會：台灣基礎神經科學學會 | 編號:29

Ripple frequency emerges from coordinated activation of CA1 parvalbumin interneurons

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Abstract

Hippocampal ripple is a high-frequency oscillation of the CA1 local field potential (LFP) critical for memory consolidation. Ripple oscillation requires inhibition mediated by interneurons. However, the firing rates of single interneurons during ripples rarely reach the ripple frequency. It is still unknown how ripple oscillation is generated by the CA1 circuit. Using a novel voltage indicator, 'Voltron', we investigated the spiking and the subthreshold activity of a population of parvalbumin (PV) interneurons while simultaneously recorded LFP ripple oscillations in vivo. Voltron revealed voltage-dependent fluorescence signal of labeled PV neurons. The spike half width and the firing rate of PV neurons were distinct from those of pyramidal neurons. Although the firing rates of PV neurons were highly variable, virtually all of them increased their firing rates during ripples. Strikingly, the summed activity of simultaneously recorded PV neurons displayed prominent ripple frequency oscillations during LFP ripple events. Such reliable oscillations were abolished by disrupting the spike timing between neurons or by synchronizing the spike patterns of all cells to that of a single cell. During ripples, PV neurons were strongly phase locked to the population oscillation. The ensemble of PV neurons active during single oscillation cycles were dynamic and their activation followed a specific temporal sequence. Moreover, the subthreshold fluctuation of PV neurons predicts the strength and the phase of their spiking modulation. Overall, our results reveal the spiking and subthreshold coordination among PV neurons underlying the generation of rhythmic inhibition during LFP ripple.

投稿學會：台灣基礎神經科學學會 | 編號:30

Glutamate/GABA co-transmission modulates hippocampal neuron activity and long-term potentiation

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Abstract

Subcortical inputs participate in corticohippocampal information processing. The neurons of the supramammillary nucleus (SuM) in the hypothalamus innervate the dentate gyrus (DG) and co-release two contrasting fast neurotransmitters, glutamate and GABA. However, the synaptic mechanisms by which SuM neurons regulate the DG activity are not well understood. The DG comprises excitatory granule cells (GCs), which are the major principal cells, as well as local inhibitory interneurons (INs). Using a combination of optogenetic, electrophysiological, and pharmacological approaches, we demonstrate that the SuM input differentially regulates the activities of different types of DG neurons via distinct synaptic excitation/inhibition (E/I) balances. Specifically, SuM-mediated synaptic excitation of GCs and soma-targeting INs is weak, and the E/I balances at these synapses are less than 1. Conversely, SuM excitation of dendrite-targeting INs is strong, and the E/I balances at the respective synapses are consistently greater than 1. Notably, SuM excitation of GCs, although weak, can enhance the spike precision of GCs and reduce their latencies in response to excitatory drives. Moreover, the SuM input as temporally co-activated with the cortical input can enhance the GC activities and facilitate long-term potentiation (LTP) at the cortical-GC synapses. Collectively, these findings provide novel insight into the co-transmission of glutamate/GABA by SuM neurons in the DG network.

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Morpho-physiological Properties of Hippocampal Dentate Granule Cells in the *Blm-s* Knockout Mice

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Abstract

BLM-s, abbreviates for BCL-2-like molecule short isoform, is a newly identified member of the *Bcl-2* family. It is expressed in postmitotic immature neurons of the developing mouse brain and acts as an apoptosis sensitizer/derepressor in regulating developmental neuronal apoptosis. However, the role of BLM-s protein in the adult brain is totally unknown. Our preliminary study showed that the adult *Blm-s* knock-out (*Blm-s*$^{-/-}$) mice exhibited heightened anxiety- and depression-like behaviors. In the adult mouse brain, *Blm-s* is expressed in the hippocampal dentate granule cells (GCs). Increased GC excitability is associated with higher susceptibility to stress-induced anxiety and depression disorders. Therefore, we investigated the electrophysiological and morphological properties of GCs in *Blm-s*$^{-/-}$ mice as a first step to understand the function of BLM-s in the adult hippocampus.

Here, we used whole-cell patch-clamp recording and *post-hoc* morphological reconstructions to investigate the electrophysiological and morphological properties of the mature GCs in male *Blm-s*$^{-/-}$ mice. Compared to those in their wild-type (WT) littermates, the *Blm-s*$^{-/-}$ GCs exhibited the following distinct features. First, the *Blm-s*$^{-/-}$ GCs had more hyperpolarized resting membrane potential and exhibited more action potentials (APs) in response to sustained depolarizing current injection. Second, the *Blm*-s$^{-/-}$ GCs generated APs with higher rising rates and shorter duration. Third, the *Blm*-s$^{-/-}$ GCs received both enhanced spontaneous excitatory and inhibitory synaptic transmissions. Finally, the complexity of the distal dendrites of *Blm*-s$^{-/-}$ GCs was reduced. Collectively, the enhanced excitability of mature *Blm-s*$^{-/-}$ GCs may account for the affective phenotypes of male *Blm-s*$^{-/-}$ mice.

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Hilar Mossy Cells Differentially Regulate Dentate Gyrus Activity via Distinct Synaptic Mechanisms

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Abstract

The hippocampus is a long, curved brain structure, which runs along a dorsal (septal)-to ventral (temporal) axis in rodents, corresponding to a posterior-to-anterior axis in humans. It is unclear how information is distributed along the hippocampal long axis. Hilar mossy cells (MCs), the excitatory principal cells of the dentate gyrus (DG), establish extensive longitudinal projections along the septotemporal axis. Therefore, MCs are thought to integrate convergent local inputs from granule cells (GCs) in the DG and in turn relay that information to distant GCs. Here, we investigated MC connections with GCs along the longitudinal axis and assessed how MC activation regulates both local and distant GC activities. Using the optogenetic approach, selective activation of MC associational fibers reduced spike latency of both local and distant GCs, but only enhanced the spike-timing precision of GCs located in the same lamella. Furthermore, MCs directly excited and indirectly inhibited GCs. Thus, MC excitation caused a low excitation/inhibition (E/I) balance in GCs in the same lamella, leading to timing-dependent enhancement or suppression of GC firing. In contrast, MC excitation resulted in a high E/I balance in GCs located in distant lamellae, thereby facilitating GC firing. The collective findings reveal that MCs can differentially regulate the activities of GCs along the hippocampal longitudinal axis through distinct synaptic mechanisms.

投稿學會：無	編號:33

Gene Expression Profiles in Parvalbumin$^+$ and Somatostatin$^+$ Interneurons

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Abstract

The two major types of inhibitory interneurons, Parvalbumin-positive (PV$^+$) and Somatostatin-positive (SST$^+$) interneurons are critical for information processing and memory formation in the hippocampus. These two types of inhibitory interneuron provide distinct contributions to feedforward and feedback inhibition pathways. PV$^+$ interneurons preferentially acting in a feedforward manner on excitatory neurons; and SST$^+$ interneurons preferentially acting via feedback pathways to excitatory neurons. These interneurons activity represents an important causal mechanism for fear memory. Although many studies investigate their physiological functions, the gene expression profiling of PV$^+$ and SST$^+$ interneurons involved in the learning and memory process remained unclear. To investigate the molecular details between them, we utilized the RiboTag and Cre-loxP-dependent recombination method to measure the transcriptome of these neurons in the hippocampus. Ribosome-bound mRNA was isolated from transgenic mice expressed HA-tagged ribosomal subunits (RiboTag) specifically in PV$^+$ and SST$^+$ interneurons and analyzed by using RNA-sequencing and RT-qPCR. Our results indicated that PV$^+$ and SST$^+$ interneurons highly expressed their specific marker genes and GABAergic related genes. The gene expression profile of PV$^+$ and SST$^+$ interneurons can contribute to the comprehension of the molecular mechanism of inhibitory interneurons under different conditions.

投稿學會：台灣基礎神經科學學會 | 編號:34

Function of Fringe localized Golgi outposts in dendrite arborization of neuron

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Abstract

Among exquisite autonomous and non-autonomous regulations crucial for the dendrite arborization of neuron, Golgi outposts (GOPs) are specialized organelle in neuron, which are potential for being functional platforms involved in dendrite arborization. Whether GOPs are responsible for multiple roles in dendrites yet remained elusive. In a candidate-based screen, we found that Fringe (Fng), the *Drosophila* beta-1,3-N-acetylglucosaminyltransferase, located to a subset of GOPs in *Drosophila* class IV dendritic arborization (C4da) neurons. and *fng* negatively regulates dendrite arborization. Interestingly, Fng increases distributions in dendrites only after mid 3rd instar larval (M3) stage that causes increase of Fng-localized GOPs in distal dendrites. Concomitantly, the overall dendrites plasticity transits from the outgrowth status to retracting status in M3 stage. Therefore, Fng-localized GOPs is the temporal factor contributing to dendrite retraction during the later development stage of C4da neurons after M3. In addition, the endopeptidase, *Furin 2* (*Fur2*), could be a gatekeeper for Fng before M3 stage, since *Fur2* expression is declined in M3 and the level of *Fur2* is anti-correlated with the amount of Fng-localized GOPs to affect the dendrite arborization. Furthermore, since Fng is known to modify the receptor Notch and enhance the interaction with its ligand Delta, the downstream effectors of Fng are also investigated in this study. Except that the *Delta* increased expression in epidermal cells adjacent to C4da neurons, epidermal Delta and neuronal Notch negatively regulate the dendrite arborization, which is in consistent with the function of Fng. Taken together, Fur2 regulates the abundance of Fng-localized GOPs, while Fng, Notch, and Delta coordinately affect the plasticity of dendrite arborization during neuronal development. Therefore, we conclude that dendritic GOPs indeed could be the pluripotent platforms of local molecular machinery for dendrite plasticity and arborization, since the Fng-localized GOPs, which are regulated spatiotemporally, is one significant repertoire of the versatile GOPs function in dendrite arborization.

投稿學會：台灣基礎神經科學學會 | 編號:35

Galectins Crouching tiger and Hidden dragon function through N-glycosylated Draper/Ced-1 receptor in dendrite pruning

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Abstract

Pruning is the important process to remove unnecessary synapses and strength the connections for neuron maturation. Drpr/Ced-1, a conserved scavenger receptor, is expressed in phagocytes to clear the neuron debris during pruning. However, the regulatory mechanism of Drpr/Ced-1 is still not well-known. We use *Drosophila* da neuron as a model to search regulators of Drpr/Ced-1 during dendrite pruning. From a genetic screen, we identified two genes in the N-glycosylation process in epithelial cells regulated dendrite pruning. Drpr/Ced-1 in epithelial cells has been reported to regulate dendrite pruning in *Drosophila*. From glycosidase treatment and mass spectrum analysis, we identified that Drpr/Ced-1 is the substrate of N-glycosylation. Moreover, we also found out that the complex or Hybrid type of N-glycosylation modification are important for Drpr/Ced-1 plasma membrane localization and function. Furthermore, we also identified two tandem-repeat type Galectins, *crouching tiger (ctg) and hidden dragon (hdg)*, genetically acted in the *Drpr/ced-1* pathway and physically interacted with N-glycan on Drpr/Ced-1. Next, Ctg and Hdg in hemocyte are induced by injury and secreted to bind Drpr/CED-1 on dendrites. Taken together, Galectins, Ctg and Hdg, in hemocyte regulate dendrite pruning through Drpr/Ced-1 receptor pathway.

投稿學會：台灣基礎神經科學學會 | 編號:36

Systematic investigation of γ-TuRC function in cerebral cortical development

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Abstract

The γ-tubulin ring complex (γ-TuRC) is a multi-subunit protein complex composed of γ-tubulin and γ-tubulin complex proteins (GCPs). γ-TuRC promotes microtubule assembly by serving as a template that allows efficient nucleation and elongation of α/β-tubulins into straw-shaped microtubule filaments, thus playing a crucial role in various cellular processes including cell division, cell differentiation, cell polarization, and cell migration. Recent studies revealed that mutations in the tubulin superfamily lead to cortical dysgenesis with a wide spectrum of neurodevelopmental defects, collectively termed tubulinopathies. In particular, mutations in γ-tubulin, the core component of γ-TuRC, and its activator Cdk5Rap2 cause brain developmental disorders known as malformations of cortical development (MCD) and microcephaly (MCPH). However, it remains elusive how γ-TuRC functions in cerebral cortex formation during embryonic brain development. In this study, we set out to systematically dissect the roles of γ-TuRC in this process and illuminate the molecular mechanisms underlying clinical deficits using mouse model. Our preliminary data indicate that knockdown of individual γ-TuRC subunits by *in utero* electroporation severely delays neuronal migration in developing brain. In particular, in the GCP2-knockdown embryonic brain, cells are almost completely locked at the intermediate zone. Surprisingly, loss of GCP2 neither affects progenitor proliferation nor induces neuronal cell death. Rather interestingly, the GCP2-knockdown cells fail to differentiate into neuronal lineages. Furthermore, using live brain-slice imaging, we found that GCP2 knockdown induces depolarization of radial glial cells (RGCs) and compromises their radial migration. These findings suggest that GCP2 is indispensable for establishing neuronal polarity that guides neuronal maturation and migration during cortical development.

投稿學會：台灣基礎神經科學學會	編號:37

An optogenetic approach to examine the effect of Ran GTPase in regulating non-centrosomal microtubules in neurons

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Abstract

Microtubules (MTs) are the most abundant cytoskeleton in neurons, and control multiple facets of their development. While the MT-organizing center (MTOC) in mitotic cells is typically located at the centrosome, the MTOC in neurons switches to non-centrosomal sites. A handful of cellular components have been shown to promote non-centrosomal MT (ncMT) formation in neurons, yet the regulation mechanism remains unknown. Here, we demonstrate that the small GTPase Ran is a key regulator of ncMTs in neurons. Using an optogenetic tool that enables light-induced local production of RanGTP, we demonstrate that RanGTP promotes ncMT plus-end growth along the neurite. Additionally, we discovered that actin waves drive the anterograde transport of RanGTP. Pharmacological disruption of actin waves abolishes the enrichment of RanGTP and reduces growing ncMT plus-ends at the neurite tip. These observations identify a novel regulation mechanism for ncMTs and pinpoint an indirect connection between the actin and MT cytoskeletons in neurons.

投稿學會：台灣認知神經科學學會 | 編號:39

Differences between family- vs. individual-level processing of objects: a cross-site fMRI study

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Abstract

The Ventral Occipital Cortex (vOT) is involved in the perception of visually presented objects, but how object shapes affect the processing of, and how these objects are processed in, vOT, have remained unclear. In the current study, we partly address this question by having participants categorize 9 novel objects ("Ziggerins") with either the categorization (among 3 families) or the individuation (among 3 individuals within each family) task.

The prior study demonstrates that fMRI activity of a region in the right fusiform gyrus increased after individuation task and was correlated with the magnitude of configural processing of the Ziggerins observed behaviorally. In contrast, categorization task caused distributed changes, with increased activity in medial portion of the ventral occipital-temporal cortex relative to more lateral area.

With this finding, we conducted a Multi Voxel Pattern Analysis (MVPA) searchlight analysis of 3 different comparisons: within-class or between-category classification, and cat vs ind comparison, for each 10-time-points(TP) of a trial. The results showed consistent higher classification at 6-10TP in the mid-fusiform seed region. According to this result, then we tested PsychoPhysiological Interaction (PPI) for 10TP across 18 subjects for cat vs ind conditions. As a result, we found a different fusiform-extrastriata connectivity pattern that shows higher for ind task in the early phase (3-6 TR/time resolution), and higher for cat task in the later phase (7-10 TR). The differences of the perception profile of object recognition were shown, while in the cat task recruited more medial region area in the early phase (1TR) and it slightly had low activation in the following TR. There was a significantly change in 3TR that the ind task gradually recruited more medial-posterior area which spread even more in 6TR. In contrast, the cat task recruited almost all the temporal lobe area in the rest TR (7-10TR), suggesting that the ind task requires more in the encoding phase, whereas the cat task requires more support in the decision phase.

The prior study did not have any significant result in medial area with four task-driven, cat_between-class-classification (bcc), ind_(bcc), cat_within-class-classification (wcc), and ind_wcc.

投稿學會：無	編號:40

Using ERP to Measure Learners' Extraneous Cognitive Load During the Simple Mathematics Addition Task

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Abstract

Cognitive load theory is an important reference for the application of learning science and educational psychology. However, it is unclear that the mechanisms of cognitive processing including intrinsic, extraneous and germane cognitive load. The present study examined extraneous cognitive load in the simple addition task based on even-related potentials. According to cognitive load theory, we manipulated three conditions which are normal numbers, colorful number and Chinese character numbers respectively. It is difficult to process the simple addition task in Chinese character numbers condition because the participants had to take more efforts to translate the characters into normal numbers before calculation. Given that the Chinese character numbers are unnecessary cognitive load, we adopted the condition as the extraneous load. The results revealed that there are the largest P2 and LPS components significantly in the extraneous condition for the stage of the augend number. Based on the cognitive load framework, the present study measures the components for encoding of extraneous cognitive load from ERP.

投稿學會：台灣基礎神經科學學會 | 編號:41

Chronic Elevation of Indoxyl Sulfate Causes Glutamate Uptake Impairment via Aryl Hydrocarbon Receptor in Chronic Kidney Disease Mouse Brain

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Abstract

Chronic kidney disease (CKD) is a progressive loss of renal function that gives rise to accumulation of uremic toxins in the blood that induces multiple organ pathology. Indoxyl-3-sulfate (I3S) is a physiological ligand of aryl hydrocarbon receptor (AhR), its excessive accumulation due to renal failure results in tissue toxicity and cannot be cleared by hemodialysis due to its protein-bound property. Recent studies indicated that CKD patients suffer from mental disorder and cognitive impairment even for those receiving hemodialysis, but the involvement of brain AhR was still unclear. In this study, we used 8 weeks after 5/6 nephrectomy in mice as the CKD animal model and nestin-Cre::Ahr-flx/flx (nAhRCKO) mice to study the role of brain AhR in CKD-associated brain disorders. Our data showed that I3S is significantly elevated in CKD mouse brain, accompanied with increased anxiety and impairment of recognition memory, and these effects were attenuated in nAhRCKO mice. Biochemical and histological analysis revealed that glutamate transporter 1 (GLT1) is selectively downregulated among other neuronal and glial proteins in the anterior cortex, suggesting that the functional disturbance of astrocytes in CKD mouse cortex. By using mouse cortical astrocytes and glia-neuron mix cultures subjected to 15-day I3S treatment to mimic the accumulation of I3S in CKD brain, we found that AhR is activated and downregulated, along with surface GLT1 reduction and impairment of glutamate uptake activity, and these effects can be reversed by AhR antagonist CH-223191. Interestingly, chronic I3S reduced PSD95 while increasing VGLUT1 in glia-neuron mix culture, suggesting the disproportion of excitatory synapses. Thus, chronic I3S accumulation in the brain causes astrocytic GLT1 hypofunction to disturb glutamate homeostasis in an AhR-dependent manner, which may contribute to the mood disturbance and cognitive declination in CKD. (Grant support: MOST 106-2320-B-010 -008 -MY3 from Ministry of Science and Technology, Taiwan)

投稿學會：台灣基礎神經科學學會 | 編號:42

The functional role of post-translational modification in ASIC4

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Abstract

ASIC4 is a member of acid-sensing ion channels and widely expressed the CNS. However, the physiological function of ASIC4 is still unclear. To probe the role of ASIC4, we generated an ASIC4-knockout/Cre-ERT2-knockin mouse line. Compared with wild-type littermates, ASIC4 knockouts showed higher levels of fear response (freezing) to the presence of 2,4,5-trimethylthiazoline (TMT), a component of fox urine; ASIC4 knockouts also demonstrated higher levels of anxiety-like behaviors in the open field (OF) and elevated plus maze (EPM) task. These phenotypes were opposite to the ASIC1a knockout mice, which showed lower levels of TMT-induced fear and less anxiety-like behaviors in these two mouse anxiety tasks. Based on previous results, we hypothesized that ASIC4 might modulate innate fear and anxiety state by counteracting the ASIC1a membrane protein expression. To prove this working hypothesis, we generated $ASIC4^{CreERT2/+}$::$ASIC1a^{f/f}$ conditional knockouts and screened the phenotypes in the TMT, OF and EPM tasks. Results indicated that conditional knockout of ASIC1a in ASIC4 positive neurons showed ASIC1a-like phenotypes in the innate fear and anxiety tests. We further examined whether ASIC4 knockout could affect ASIC1a protein expression in specific brain areas. Interestingly, we found ASIC1a membrane protein expression is increased in ASIC4 KO in PAG, BNST, pituitary gland, VPM/VPL, amygdala and cerebellum as compared with wildtype control. Because ASIC4 is composed of several glycosylation sites in the extracellular loop, we further generated different point mutations to see how glycosylation of ASIC4 can affect the interaction with ASIC1a.

投稿學會：台灣基礎神經科學學會 | 編號:43

A remote-control mechanism for sensing pH_o in TALK1 channels

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Abstract

Two-pore domain K^+ channels (K2P) are dimeric potassium channels that set the resting membrane potentials, and malfunctioning of K2P channels often causes diseases in humans, including cancer, migraine, and diabetes. K2P channels are gated by various external and internal signals, such as temperature, pressure, oxygen and pH. These stimuli are first sensed by various parts of the K2P channels, and later converge onto the selectivity filter via a mechanism that is similar to typical C-type inactivation in other potassium channels. TALK1, a pancreatic specific K2P channels are linked to type 2 diabetes in humans, is gated by pH_o, but how TALK1 senses pH_o is still unknown. Here, we identified an arginine (R233) on TALK1 serves as a remote-control for sensing pH_o via C-type inactivation that modulate the potassium occupancy on S_0. Mutating R233 to the neutral alanine (A) or negatively-charged glutamic acid (E) on TALK1 eliminated or inverted its sensitivity to pH_o, respectively. We further identified that high concentration of tetraethylammonium (TEA) could block TALK1 channel from external site. Alkalic pH_o prolonged the dissociation time constant (τ_{off}) of TEA inhibition in wild-type but not R233A TALK1, and this results further supported that the S_0 is involved in sensing pH_o. In conclusion, we have identified that R233 is the sensor for sensing pH_o. This residue remotely controls the S_0 binding site and consequently, regulate the TALK1 gating.

投稿學會：台灣基礎神經科學學會 | 編號:44

The Genetic Mapping of Kir6.2 in Whole-Body Expression Pattern

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Abstract

The central nervous system (CNS) is a master player in regulating glucose homeostasis, and a subset of neurons serve as the primary glucose sensors in this CNS-mediated glucoregulatory system. Although the glucose-sensing neurons have been found throughout the brain, the majority of these neurons are located in the hypothalamus and brainstem. Cumulative studies have demonstrated that the ATP-sensitive potassium channel (KATP channel) is a key molecular component for glucose sensing in the hypothalamic neurons; however, the KATP channel expression pattern in the brain and other peripheral organs remains elusive, mostly due to a low expression level and lack of a sensitive detection method. In this study, we plan to map the anatomical locations of the neurons expressing Kir6.2, the pore forming subunit of the KATP channel, and whether the expression levels of Kir6.2 can be altered under metabolic stress. We utilized a mouse genetic approach with a reporter gene, LacZ, which encodes for beta-galactosidase, knocked into KCNJ11, the gene encodes Kir6.2. We validated mouse whole-body Kir6.2 (beta-galactosidase) expression via X-gal staining and confirmed that there were no background signals in the wild-type mice. In the future, the mice will be fed with high-fat diet and further examined for Kir6.2 expression in the metabolic active organs.

投稿學會：台灣基礎神經科學學會 | 編號:45

Investigation of Novel Long Noncoding RNA Litchi Regulating Spinal Activities during Development

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Abstract

In recent years, long non-coding RNAs (lncRNA) have been found relatively high tissue specificity and this type of RNAs can also serve as regulators in cellular functions, especially in nervous system. Rather than brain regions, lncRNA-mediating function is poorly understood in the spinal cord. Here we found a novel lncRNA, long intergenic ncRNA near cholinergic locus (Litchi), is highly expressed in spinal motor neurons (MNs) but not in other spinal neuron types. However, the detail mechanisms of Litchi still remain unclear. To understand Litchi expression profiles, we performed real-time qPCR as well as *in situ* hybridization in both embryonic and neonatal spinal cords. We found Litchi is enriched in the embryonic spinal cord but barely presented in postnatal, implying that Litchi may play roles in motor circuit development. Moreover, we observed MN neurite growth using either mouse embryonic stem cell derived MNs or primary-cultured MNs from Litchi knockout mice in order to find Litchi effects on neural function. We found that both types of MNs have less neurite complexity under Litchi-knockout condition. Those results reveal that Litchi might regulate neurite growth during embryonic MN developing period. In addition, during neural development, spontaneous activities in the spinal cord occur during E12-18, and these activities hit the spot of Litchi expression profile. Therefore, we hypothesize that Litchi regulates neurite growth via neuronal activities. In the purpose of exploring this hypothesis, we will perform calcium imaging in embryonic stem cell derived MNs to observe physiological changes after Litchi knocking out. Furthermore, we will investigate calcium oscillation in the mouse spinal cord to understand *in-vivo* regulation of Litchi. Together, our data suggest that Litchi plays a role for MN development. We expect that these data will provide mechanisms how an lncRNA regulates motor circuit formation.

投稿學會：無	編號:46

miR-34/449 mediates precise interneuron assembly to exert proper core sensory-to-motor spinal network

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Abstract

Although the function of microRNAs (miRNAs) during embryonic development has been intensively studied in recent years, their postnatal physiological functions remain obscure largely due to the sophisticated genetic knonckout (KO) attributed to the redundant paralogs with the same seed miRNA families. This is particularly challenging to uncover miRNA functions at neural circuit level as ones might need to investigate animal behaviors upon the complete loss of miRNA family functions. Here we focused on *miR-34/449* family that manifest dynamical expression pattern in the developing spinal cord yet have uncharacterized role from postnatal to adult stages. The complete loss of the *miR-34/449* in the triple KO mouse model leads to several unexpected neural related phenotypes, including peculiar motor-sensory behaviors. Sensory-to-motor spinal behavioral assays reveals that the loss of *miR-34/449* perturbs the threshold of thermal-induced pain response. Mechanistically, *miR-34/449* targets to Satb2 directly to fine-tune the precise number of a sub-population of lamina V/VI inhibitory sensory relay neurons. Thus, *miR-34/449* is suggested to govern the optimal development of Satb2^{on} interneurons in the spinal cord to fine-tune the output of sensory-to-motor circuit.

投稿學會：台灣計算神經科學學會 | 編號:47

Analysis of brain images of Drosophila melanogaster acquired by x-ray synchrotron

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Abstract

The construction of connectomic data offers the basis for the understanding of the neuronal computing principles. Recently the electronic microscopy has resolved the finest hemibrain of a fly, to date. However, we still need a method to acquire neural network images in a high-throughput fashion at cellular resolution so that we can explore brains from different individuals efficiently. Or we will be limited to further unravel the knowledge such as the stereotypy of the wiring diagram. On the other hand, the diffusive tensor imaging is capable of generating data from many individuals, but the resolution cannot suffice our purpose. To overcome such a dilemma, the researchers develop a novel imaging technique, Accelerated X-ray Observation of Neurons (AXON) based on the synchrotron and the conventional Golgi stain. This imaging tool can image a fly brain within hours and the resolution is up to 0.5 μm. In this study, we develop a series of imaging analysis tools for the AXON-derived *Drosophila* brain images. Our results suggest that the morphological similarity and the spatial orientation of a neuron in a brain can help us identify neurons. Next, we identify the neuronal bundles to align brains from different images. Our future work is to identify a neuron in the AXON images by the neuron data in the fluorescent-image-based FlyCircuit database.

Keywords:
A *Drosophila* connectome, AXON, the Golgi stain, neuronal classification, neuronal bundles

投稿學會：無 | 編號:48

A Novel Genetic X-Ray CT Mapping of Animal Brain

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Abstract

It has been a main challenge to map the neural wiring network of any animal brain that consists of both cellular projections and synaptic connections. Although X-ray computed tomography (CT) is a powerful tool for brain mapping, currently neural labeling methods lack the capability for integration of X-ray CT reconstructed neural morphologic data with existing neuroanatomy knowledges, which is essential for data validation and further application. We developed a cell labeling technique that is genetic controllable, X-ray adsorbing, and localized under subcellular level that is available for syncrhrotron X-ray CT. By combining genetic expressing peroxidase, enzyme metallography, metal counterstaining, and Accelerated X-ray Observation of Neurons (AXON) system, this new technique acquire intact three-dimensional neuropil shape, single neuron morphology and subcellular protein localization in Drosophila brain with sub-micron level isotropic resolutions under X-ray CT. This method utilize X-ray CT to generate reproducible and analyzable neuron morphologic data with better resolution and faster labeling as well as data acquisition time from intact tissue without sectioning, releasing all potentials of X-ray CT in neuroscience research field.

投稿學會：台灣基礎神經科學學會 | 編號:49

Constructing Neuron-neuron Interaction Graph from Calcium Imaging Data

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Abstract

Calcium imaging with recently developing miniature fluorescent microscope systems has been being a powerful tool that can directly visualize neural activities in the brain in a freely behaving animal. With this technology, researchers have conducted many studies on including sensory neural, olfactory, auditory, and so on systems in various brain regions in different species. Therefore, this technology has been shifting the brain science study from region-to-region to neurons-to-neurons and from in vitro to in vivo. Since the captured images are usually blurry and unstably, a computational tool is always needed to enhance the quality and then detect the activated neurons. A few of popular tools, such as CaImAn, are available but all focus on how to identify individual activated neurons from captured noisy images. Moreover, most of their outputs can be obtained with only a set of individual neuron activities in an image format. Thus, how to process and analyze the neuron-neuron interaction in a network level is still a challenge for neuroscientists.

Here, we propose a computational pipeline to process the dynamic calcium imaging data and construct a graph isomorphism model to analyze activated neurons at a sub-network level. In the first step, an open source Python-based toolbox, called CaImAn, is applied to process the input calcium dynamic images and detect all activated neurons and their fluorescence intensity changes. Then, a homemade program is used to extract the raw data hidden in CaImAn internal files and transfer the data formats of positions and intensity values of each activated neuron. After that, the intensity correlations of each neuron with all other neurons are calculated. If two neurons their intensity profiles are correlated (>0.75), then an edge is created between the two neurons. After repeating the correlation calculation and edge creation process until all activated neurons have been done, a graph, called Neuron-Neuron Interaction Graph, is constructed where a node and an edge represent the neuron geometric position in the image and two connected nodes with similar correlated in intensity, respectively. In the result, a set of calcium dynamic images that captured by Doric Lenses system on mouse PVA region with various nerve stimulations have been processed by our proposed method. We found that neurons with simultaneous activity usually form several various sub-networks by different stimulations. In the future, we will develop different graph matching approaches to compare the patterned information or structural similarities between calcium imaging graphs by different conditions. Finally, we expect our developed method that can assist neuroscientists to explore and enhance the understanding of neuron activity at a network level.

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Identify the role of hippocampus plays in fear memory retrieval during sleep through calcium imaging

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Abstract

Hippocampus has long been regarded as a memory-consolidating center, and there are numerous researches further indicate the important role that it plays in fear memory retrieval and contextual fear learning. In general, experiments of these researches are mostly conducted on the sober stage of mice; however, whether hippocampus involved in retrieval of fear memory during sleep is still unknown. We hypothesized that during sleep, the fear-responded cells in the hippocampus are activated when the conditioned stimuli are presented.

To test the hypothesis, jGcamp7s, a calcium indicator, were expressed on hippocampal neurons by viral vector injection in hippocampus of mice. Then, we placed a GRIN lens in CA1 region of hippocampus and mounted a miniscope to receive neural fluorescence.

After operation, these mice would undergo several pairings of footshock and tone for few days in training period. Then, in testing session, we played the tone and record neural activity through the miniscope.

By visualizing collected data, we found that hippocampal neural activity seemed to correlate to the timing of the tones, which implies that hippocampus may also involve in fear memory retrieval during sleep.