	順序編號	英文姓名	中文姓名	Title
01-1	1	Kuo-Hua Huang	黃國華	A virtual reality system to study social interaction in adult zebrafish
01-2	1	Ting-Hsuan Liang		Investigating the circatidal rhythm in vertebrates using Periophthalmus modestus (Shuttles Hoppfish)
01-3	1	Li Jou Joey Lai	賴立柔	Optogenetic inhibition of vCA1 suppresses LH-related sleep disturbance induced by footshock in mice
01-4	1	Wen-Hsin Lu	呂文心	CPEB2-Activated Axonal Translation of VGLUT2 mRNA Promotes Glutamatergic Transmission and Presynaptic Plasticity
01-5	1	Ya-Chih Chien	簡雅致	Involvement of ASIC1a in ASIC4-positive BNST/amygdala neurons in modulating anxiety and fear.
01-6	1	Gary Chieh-Wei Lee	李杰威	Exploring Sng Behavior in Mouse Gait
01-7	1	Cheng-Han Lee	李政翰	An unexpected role of proprioceptors in the development of chronic muscle pain
01-8	1	Kuan-Yun Ting	丁冠云	Basal forebrain glutamatergic neurons inhibit reward-seeking behavior and the activity of bursting neurons
01-9	1	You-Jhe Jhong	鐘佑哲	Functional coupling between midbrain dopamine neurons and basal forebrain bursting neurons in the encoding of reward prediction error
01-10	1	Shih-Che Lin	林士哲	Distinct subsets of hypothalamic SF-1 expressing neurons encode an exploratory internal state that drives animals' investigative behaviors.
01-11	1	Ming Chia Chu	初銘家	Generational synaptic functions of GABAA receptor β3 subunit deteriorations in an animal model of social defcit
01-12	1	Jing-Ting Fu		Dopamine signaling in the regulation of neuroinflammation and glymphatic dysfunction during chronic feeding with high fat diet
01-13	1	Yi-Chun Yen	顏怡君	Effects of sleep restriction on brain function and cognition-enhancing protein klotho

O1-14	1	Sofia Ormazabal	鄒智文	Impact of information transfer in Periplaneta group dynamics and aggregation behaviour
O1-15	1	Imelda Margaretha Aritonang	艾曼達	Investigating the role of Cap-methyltransferase 2 in cerebellar development and neurodegeneration
P1-16	1	Pei-Rung Wu	吳佩容	Wdr4 Promotes Cerebellar Development and Locomotion through Arhgap17-Mediated Rac1 Activation
P1-17	1	Hui Yu Wang	王慧鈺	The function of Wuho in cortical development
P1-18	1	Wen-Hsin Hsu		To investigate the mechanisms regulating border formation of entorhinal cortex
P1-19	1	Dhivya Appan	賴文雅	Linking hippocampal patterning with hippocampus-related behaviors
P1-20	1	De-Fong Huang	黃得峰	Neuronal splicing regulator RBFOX3 mediates seizures via regulating Vamp1 expression preferentially in NPY-expressing GABAergic neurons
P1-21	1	Sayma Azeem		Characterization of the molecular and cellular defects underlying memory consolidation defect in CMTR1-cKO mice
P1-22	1	Pei Ling Ho	何霈翎	Effects of chronic antibiotics exposure on anxiety-related behavior
P1-23	1	Tung Hsiao Jou	董筱柔	The beneficial effects of dietary restriction on social defeat-induced cognitive decline
P1-24	1	Chun-Chieh Huang		Investigate the underlying mechanism of gut microbiota in dietary restriction-associated benefits
P1-25	1	EN CHI WEI	魏恩祈	Light dark cycle influences Gut microbe daily oscillation independent of peripheral clock
P1-26	1	Cheng-Yu Chen		Nesting behavior induced alterations in the gut microbiota of two passerine bird species
P1-27	1	Yu-Fan Huang		Intrinsically photosensitive retinal ganglion cells inhibit socio-sexual recognition memory through supraoptic oxytocin neurons

P1-28	1	Yu Kai Ma	麻彧愷	Positive correlations between social hierarchy and memory in weaning mice and young children
P1-29	1	Wen-Jie Chen	陳文傑	A research proposal for investigating the role of microtubule dynamics during long-term memory formation in Drosophila
P1-30	1	敬哲 強		The hybrid wiring pattern in a fly brain diversifies olfactory information processing
P1-31	1	Chien-Tzu Lin	林千慈	Exploring the osmosensing mechanism in a pair of thirst-broadcasting neurons
P1-32	1	Ping Yen Hsiao	蕭秉諺	Endolysosomal 2Cl-/H+ exchanger coordinates neuron-glia crosstalk to control synaptic bouton development
P1-33	1	TING-YI LU	呂亭儀	Modeling frequent human variants of Flower reveals its regulation on synaptic vesicle recycling and synaptic bouton architecture through distinct mechanisms
P1-34	1	PengTzu Chen		Studying the atypical localization of the centrosomal protein Cep170 during neuronal morphogenesis
P1-35	1	Shu-Fei Lin		Degree of dispersion in the cerebral cortex and the correlation with mental illness
P1-36	1	Chih-Yun Chung	鍾芷昀	Genetics association between schizophrenia and cerebral functional areas
P1-37	1	Chia-Yuan Chang	張家源	To investigate the role of Akt1, a schizophrenia candidate gene, in reward-based decision making- neural mechanism of dorsomedial striatum from multidisciplinary approaches
P1-38	1	Fang Zhi Chua	蔡枋芷	The morphological characteristics of vasopressin neurons in zebrafish brain
P1-39	1	Hsi Chen	陳熙	Tracking the movement of zebrafish larrve by multispectral imaging system
P1-40	1	Poulomi Adhikari		Neural circuit remodeling in the primary motor cortex during continual motor learning
P1-41	1	Shuo-Yen Chueh		The functional role of striatal projection neurons in the striosome in encoding motor and reward information during motor learning

P1-42	1	Ming-Ching Chiang	江明憬	Causal interactions between neuronal activities in midbrain dopaminergic neurons and basal forebrain neurons
P1-43	1	長亨 謝		14-3-3 proteins regulate Eag1 K+ channel degradation mediated by Cullin 7
P1-44	1	Yi-Hsuan Lin	林宜萱	Abnormal ankle proprioceptive acuity in young adults with a probable developmental coordination disorder
P1-45	1	Yi-Wen Chen	陳憶玟	The relationship between motor proficiency and lower limb proprioception in healthy young adults
P1-46	1	Wei-Fen Hu	胡瑋芬	Immunoproteasome inhibition modulates microglial polarization in erythrophagocytosis
P1-47	1	YE SHA-HUA	葉沙樺	Ischemia-induced M2-to-M1 transition in microglia was blocked by leptin that was independent from autophagy inhibition
P1-48	1	Yang-Teng Fan	范揚騰	The Excitatory–Inhibitory metabolite activity in the prefrontal cortex mediates the relationship between sensory responsivity and autistic traits
P1-49	1	Hui-Ching Hsu	許惠晴	Context effects on visual perception and probability estimation
P1-50	1	Bing Shan Wu	吳秉珊	Divergent Impacts of Complexity on Spatial and Temporal Sequence Learning
P1-51	1	Andhika Renaldi		The optimization of a novel statistical learning test with hidden temporal and spatial positional regularity
P1-52	1	Sarina Hui-Lin Chien	簡惠玲	Revisiting the effect of skin color on categorizing mixed-race faces spanning the Asian-White and the Asian-Black continuum: 2AFC and 3AFC tasks
P1-53	1	Muchen Wang		Over- and underreactions to regime shifts and their neurocomputational substrates
P1-54	1	Siao-Jhen Wu		Monetary gains and losses are represented by high-frequency oscillatory activity in the human brain
P1-55	1	MIN-MIN LIN	林敏敏	The Role of Self Emotional Processing and Empathic Responses in Gluckschmerz and Schadenfreude

投稿學會：台灣基礎神經科學學會	編號: P1-16

Wdr4 Promotes Cerebellar Development and Locomotion through Arhgap17-Mediated Rac1 Activation

Pei-Rung Wu1,2,*, Shang-Yin Chiang1, Robert Midence3, Wen-Chao Kao1, Chun-Lun Lai1, I-Cheng Cheng4, Shen-Ju Chou4, Chih-Cheng Chen3, and Ruey-Hwa Chen1,*

1 Institute of Biological Chemistry, Academia Sinica, Taipei 115, Taiwan 2 Cardiovascular and Mitochondrial Related Disease Research Center, Hualien Tzu Chi Hospital, Hualien 970, Taiwan 3 Institute of Biomedical Sciences, Academia Sinica, Taipei 115, Taiwan 4 Institute of Cellular and Organismic Biology, Academia Sinica, Taipei 115, Taiwan

Abstract

Patients with mutations of WDR4, a substrate adaptor of the CUL4 E3 ligase complex, develop cerebellar atrophy and gait phenotypes. However, the underlying mechanisms remain unexplored. Here, we identify a crucial role of Wdr4 in cerebellar development. Wdr4 deficiency in granule neuron progenitors (GNPs) not only reduces foliation and the sizes of external and internal granular layers but also compromises Purkinje neuron organization and the size of the molecular layer, leading to locomotion defects. Mechanistically, Wdr4 supports the proliferation of GNPs by preventing their cell cycle exit. This effect is mediated by Wdr4-induced ubiquitination and degradation of Arhgap17, thereby activating Rac1 to facilitate cell cycle progression. Disease-associated Wdr4 variants, however, cannot provide GNP cell cycle maintenance. Our study identifies Wdr4 as a previously unappreciated participant in cerebellar development and locomotion, providing potential insights into treatment strategies for diseases with WDR4 mutations, such as primordial dwarfism and Galloway-Mowat syndrome.

投稿學會：台灣基礎神經科學學會	編號: P1-17

The function of Wuho in cortical development

Hui-Yu Wang, Zihui Zhuang, and Shen-Ju Chou

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

Abstract

Wuho (also known as WDR4) is an essential gene for embryonic development. It regulates many critical physiological processes, including tRNA modification, genome stability, and germ cell homeostasis. As Wuho mutations in humans were linked to microcephaly primordial dwarfism, we study the function of the Wuho gene in cortical development. We first generated Wuho conditional knockout (cKO), in which Wuho is deleted by Emx1-Cre in the cortical progenitors and their progeny, the cortical excitatory neurons. We found the cKO mice weigh less and have a shorter lifespan than their wild-type littermates. Further, agreeing with the microcephalic phenotype found in the patients, cKO mice also have a smaller and thinner cortex. To investigate the causes of microcephaly in the cKO mice, we compared progenitor proliferation/differentiation and cell death in WT and cKO mice. We found a significantly increased number of apoptotic cells in the cKO cortices. Interestingly, the distribution of these apoptotic cells is not uniform in the cKO cortices: more of them are found in the rostro-medial cortex. We are currently investigating the molecular mechanisms for Wuho to regulate cell survival during cortical development.

投稿學會：台灣基礎神經科學學會	編號: P1-18

To investigate the mechanisms regulating border formation of entorhinal cortex

Wen-Hsin Hsu, Hsiang-Wei Hsing, Yu-Kuan Pan, Shen-Ju Chou

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

Abstract

The cerebral cortex consists of multiple functional domains, and each has specific functions, location, and neuronal properties. We study how, during development, a specific cortical region arises at a specific location, acquires its specific properties, forms appropriate neuronal connections, and generates boundaries with adjacent cortical regions. We previously identified COUP-TFI plays a critical role in the development of the medial entorhinal cortex (MEC), and also controls the location and integrity of neocortex (NC)/MEC border through differential cell affinities. We also demonstrated that protocadherin 19 (Pcdh19) is required for COUP-TFI-induced cell clustering. To further investigate the role of Pcdh19 in NC/MEC border formation, we analyzed the NC/MEC border in the Pcdh19 null mutants. Surprisingly, we detected a sharp border between NC and MEC in the Pcdh19 null mutants at P7, similar to wild type mice. This suggests that, in addition to Pcdh19, other adhesion factors might be involved in NC/MEC border formation. Moreover, we are interested in the mechanisms, which segregate EC into two functionally distinct domains, MEC and lateral entorhinal cortex (LEC). Thus, we analyzed published scRNA-seq data from mouse brains to identify genes that are differentially expressed among these regions. We will focus on the genes encoding cell adhesion molecules as they are likely to regulate border formation. This study will contribute to the establishment of cortical functional domains during cortical development.

投稿學會：台灣基礎神經科學學會	編號: P1-19

Linking hippocampal patterning with hippocampus-related behaviors

Dhivya Appan, Ching-San Tseng, Zi-Hui Zhuang and Shen-Ju Chou

Institute of Cellular and Organismic Biology, Academia Sinica

Abstract

COUP-TFI (chick ovalbumin upstream transcription factor I, or NR2F1), an orphan member of the nuclear receptor superfamily, is an important regulator of neurogenesis, cellular differentiation, and cell migration. It is also involved in the development of hippocampus, as the loss of COUP-TFI leads to hippocampal hypoplasia. We recently identified a novel function of COUP-TFI as the hippocampal patterning factor: First, we discovered a dose-dependent relationship between COUP-TFI expression level and the hippocampal volume, as the hippocampal volume is reduced in COUP-TFI conditional knockout (cKO) mice and expanded in conditional transgenic (cTG) mice. Second, we found hippocampus was dorsalized in the cKO and ventralized in the cTG. Thus, we investigate the effect of the patterning changes of hippocampus on mouse behaviors. The hippocampus has dual functions, with the dorsal part involved in spatial learning and memory and the ventral part involved in emotions and anxiety. We have performed a battery of behavior experiments exploring the possible disruption in hippocampus-related behaviors. We observe that the loss of COUP-TFI (in cKO) results in alleviated anxiety-like behaviors and excess COUP-TFI (in cTG) leads to impaired spatial memory. Our findings suggest that COUP-TFI regulates hippocampal patterning which in turn affects mouse behaviors. We also find out how these behavioral tasks activate neurons in different subfields of the hippocampus. Due to the observed difference in anxiety-like behaviors in the mutant mice, we are currently investigating the effect of external stress on the mutant mice.

投稿學會：台灣基礎神經科學學會	編號: P1-20

Neuronal splicing regulator RBFOX3 mediates seizures via regulating Vamp1 expression preferentially in NPY-expressing GABAergic neurons

De-Fong Huang, Chih-Yu Lee, Ming-Yi Chou, Tzu-Yin Yang, Xuhui Cao, Yu-Hsuan Hsiao, Rui-Ni Wu, Cheng-Chang Lien, Yi-Shuian Huang, Hsiang-Po Huang, Susan Shur-Fen Gau, and Hsien-Sung Huang

Graduate Institute of Brain and Mind Sciences, College of Medicine, National Taiwan University

Abstract

Epilepsy, which is also called a seizure disorder, is one of the most common neurological diseases that affects around 50 million people worldwide. Approximately 50 % of people with epilepsy have a genetic basis. With the genome-wide association studies (GWAS) and family studies, a number of genetic changes have been associated with epilepsy, including a neuronal splicing regulator gene, RNA binding fox-1 homolog 3 (RBFOX3). RBFOX3, also known as NeuN, is a well-recognized marker of postmitotic neurons that has widely been used in neuroscience research. It has been identified as an RNA alternative splicing regulator that mediates the hippocampal circuit balance and function, neurogenesis, synaptogenesis, and promotes neuronal differentiation during development. A causal relationship between genetic defect and epilepsy has been supported by observations that Rbfox3 knockout mice exhibit enhanced seizure susceptibility induced by kainic acid (KA) treatment, a well-established model of temporal lobe seizures. However, the mechanism of seizure mediation by RBFOX3 is still unclear. Here, we used different kinds of cell type-specific Rbfox3 conditional knockout mice (Gad2-Cre for GABAergic neurons; Camk2α-Cre glutamatergic neurons; Pomc-Cre for hippocampal dentate gyrus granule cells (DGGCs); and PV-Cre, SOM-Cre, VIP-Cre, Npy-Cre, CR-Cre, Cck-Cre, and Nos1-Cre for seven subtypes of GABAergic neurons) as models to enable genetic dissection of Rbfox3 and identify the pathophysiological mechanisms underpinning epilepsy. Mice with selective loss of Rbfox3 in GABAergic neurons but not glutamatergic neurons exhibit spontaneous seizures and high premature mortality due to increased presynaptic release, postsynaptic potential, neuronal excitability, and synaptic transmission in hippocampal DGGCs. Attenuating early excitatory GABA action by administering bumetanide rescued seizure phenotypes. Rbfox3 deletion reduced the expression level of VAMP1, a GABAergic neuron-specific presynaptic protein, in the hippocampus. Postnatal restoration of VAMP1 rescued premature mortality and neuronal excitability in DGGCs. Furthermore, loss of Rbfox3 in GABAergic neurons showed fewer neuropeptide Y (NPY)– expressing GABAergic neurons. In addition, deletion of Rbfox3 in NPY-expressing GABAergic neurons decreased intrinsic excitability and increased seizure susceptibility. Our results provide evidence for the crucial role of RBFOX3 in the pathogenesis of epilepsy and suggest the importance of an alternative splicing regulator in mediating brain function.

投稿學會：台灣基礎神經科學學會 | 編號: P1-21

Characterization of the molecular and cellular defects underlying memory consolidation defect in CMTR1-cKO mice

Sayma Azeem, and Yi Shuian Huang

1. Taiwan International Graduate Program in Interdisciplinary Neuroscience, National Yang-Ming University and Academia Sinica, Taipei, Taiwan 2. Institute of Biomedical Science, Academia Sinica, Taiwan

Abstract

Eukaryotic mRNA is 5 ‧-end-capped with m7 guanosine, known as cap0 (m7GpppNpNp, N: any nucleotide). Cap methyltransferase (CMTR1) further catalyzes 2 ‧-O-ribose methylation of the first transcribed nucleotide (N1 2 ‧-O-Me) to produce the cap1 (m7GpppNmNp) structure in all eukaryotes except yeasts. Although the cap0 structure is essential for mRNA stability and cap-dependent translation, it is not known whether cap1 modification also plays a role in regulating posttranscriptional gene expression. We found that CMTR1 deficiency affects dendritic arborization and cortical development. Because CMTR1 is highly expressed in the hippocampus, we investigate whether CMTR1 modulates synaptic plasticity and spatial memory by using conditional knockout (cKOCamk2, Cmtr1f/f, Camk2-Cre/+) mice whose Cmtr1 gene is ablated in the hippocampal CA1 region after postnatal 2-3 weeks. We found that CMTR1-cKO mice have impaired memory consolidation in the Morris water maze. Dendritic spine number was reduced in CMTR1-knockdown cortical neurons. Although one train of high-frequency stimulation (HFS) and Theta-burst stimulation (TBS) – evoked long-term potentiation (LTP) was comparable between cWT and cKO mice, long-lasting LTP elicited by 4 trains of HFS and TBS was found to be diminished in the cKO group. My recent transcriptome study projects the possibility of the involvement of NMDAR-related pathways. Further study is required to understand the molecular mechanism underlying impaired memory consolidation in CMTR1-cKO mice.

投稿學會：台灣基礎神經科學學會	編號: P1-22

Effects of chronic antibiotics exposure on anxiety-related behavior

Pei-Ling Ho and Yi-Chun Yen

Department of Life Science, Tunghai University, Taichung, Taiwan. Behavioral Neuroscience Laboratory, Department of Life Science, Tunghai University, Taichung, Taiwan.

Abstract

Microbiota in the gut is essential to various aspects of physiology. Imbalance of gut microbiota influences gut-brain communication that induces alterations in brain function, including emotional processing. Studies have revealed significant changes in gut microbiota in patients with affective disorder and stress-related rodent models. Therefore, we hypothesize that gut microbiota imbalance leads to emotional changes through stress response systems. However, the effects of gut microbiome on anxiety-related behavior are inconsistent in recent studies. Thus, this study aims to elucidate the effects of gut microbiota imbalance on anxiety-related behavior and its underlying mechanisms. We used antibiotics cocktail (ABX) treatment to induce microbiota imbalance in mice. After four-week of antibiotics exposure, anxiety-related behavior and stress response were assessed. Our results demonstrated that ABX treatment induced different levels of microbiota depletion. ABX-treated mice with complete microbiota depletion displayed hypolocomotion and increased anxiety-related behavior, but no changes in stress hormone (i.e., corticosterone) levels. In conclusion, antibiotics exposure leads to elevated levels of anxiety-related behavior, but the effect may not be related to stress response. We are working on delineating the molecular and cellular hallmarks of how the gut microbiota imbalance influences anxiety-related behavior. Further results will provide potential means to rescue the aberrant anxiety phenotype induced by antibiotics exposure.

投稿學會：台灣基礎神經科學學會	編號: P1-23

The beneficial effects of dietary restriction on social defeat-induced cognitive decline

Hsiao-Jou Tung, Chun-Chieh Huang, You-Cheng Liu, Wan-Kun Lin, Pei-Yu Wang

Graduate Institute of Brain and Mind Sciences, College of Medicine, National Taiwan University

Abstract

Chronic social stress is a complex psychiatric condition that can lead to major depressive disorder (MDD). More than 300 million people worldwide suffer from MDD, which has core symptoms such as negative emotion and cognitive deficits. Current treatment for MDD, including selective serotonin reuptake inhibitors and ketamine-based antidepressants can only improve the emotional response but are less effective on the other associated cognitive impairment. Thus, there is an urgent need to develop a promising intervention that can treat various disease-related symptoms. Our previous research demonstrated that dietary restriction (DR, defined as 60% of daily food intake of ad libitum, AL) can considerably improve behavioral outcomes and enhance the memory performance in both young and aged mice through the microbiota-gut-brain axis. Nevertheless, the effect of DR on depression and associated cognitive impairments is largely unknown. Therefore, we used the repeated social defeat stress model (RSDS) to induce depressive-like behavior and memory dysfunction in mice, and found that one month of DR can significantly reverse these behavioral deficiencies. More importantly, DR can also affect structural alterations of hippocampal neurons, and up-regulate the hippocampal neurogenesis. However, these beneficial effects of DR were largely abolished when antibiotics were added to the drinking water of RSDS mice. In addition, DR-derived fecal microbiota to RSDS mice was adequate to imitate DR-induced advantages and highlight the significance of gut microbiota. Overall, our findings demonstrated that the DR-induced beneficial effects under RSDS require gut microbiota. Microbiome analysis, metabolomics analysis, and metagenomic sequencing will be performed to understand the mechanism underlying DR-induced pro-cognitive effects. We believe that this study will make a significant impact on the treatment of emotional response and cognitive impairment associated with MDD in the future.

投稿學會：台灣基礎神經科學學會	編號: P1-24

Investigate the underlying mechanism of gut microbiota in dietary restriction-associated benefits

Chun-Chieh Huang, You-Cheng Liu, Wan-Kun Lin, Hsiao-Jou Tung, Pei-Yu Wang

Graduate Institute of Brain and Mind Sciences, College of Medicine, National Taiwan University

Abstract

Dietary restriction (DR), carried out as consuming 20% to 40% less of daily intake with sufficient micronutrient, is one of the most renowned diet regimens to improve physiological status, counter disease-related dysfunction, and mitigate age-related dysfunction. Mechanistically, DR improves proteostasis, regulates inflammation, modulate intracellular signaling, and maintain glycemic profile to achieve beneficial effects. Given the extreme challenge to sustain such rigid regimen in our daily life, it is crucial to pinpoint suitable downstream effectors and the physiological phenomenon to recapitulate the effect of DR. Recently, the gut microbiota, a group of commensal bacteria resided in our gut, has raised as an important factor that shape a wide range of biological functions. Various beneficial effects of DR have been proven to be correlated with the alteration of gut microbiota. Our current research aims to establish the causal relationship between DR-associated benefits and DR-derived gut microbiota, and to systematically decipher the mechanism underlying DR-associated benefits relevant to the gut microbiota, focusing on the effect of DR on pro-cognition, metabolic health, and longevity. Antibiotics-treated mice and germ-free mice would be used as loss-of-function models to examine the role of the gut microbiota. According to our hypothesis, DR-associated benefits mediated by the gut microbiota would be diminished in the absence of the gut microbiota. Furthermore, mice with normal food intake (ad libitum, AL) but receive fecal microbiota transplantation from DR would recapitulate DR-associated benefits. Next, to uncover underlying bacteria-host interaction and identify potential probiotics and postbiotics, we would implement transcriptomic, metagenomics, and metabolomics approach. These identified probiotics and postbiotics would be subjected to further in vivo validation in normal mice as well as diseased models. We believed this study would provide a comprehensive understanding of how DR-derived gut microbiota elicit beneficial effects, providing a solid backbone to identify a druggable target to recapitulate DR-associated benefits and ameliorate diseases.

投稿學會：台灣基礎神經科學學會 | 編號: P1-25

Light dark cycle influences Gut microbe daily oscillation independent of peripheral clock

En Chi, Wei Yun Pu, Li Yuan Yuan, Hsieh Shih Kuo, Chen

Department of Life Science, National Taiwan University

Abstract

Gut microbiota could interact with the body and further modulate physiological function. Recent research has discovered that gut microbiota displays diurnal oscillatory rhythm, which could be dampened by constant darkness or an abnormal light environment. Thus, it brings our interest in how gut microbiota is affected by the environment without directly receiving light signals. Recent evidence suggests that the crosstalk between the intestinal immune system and gut microbiota may contribute to the circadian rhythm of gut microbiota. Here, we focus on NOD2, a subtype of NOD-like receptors (NLRs), and its downstream pathway. We discovered that NOD2 and its downstream protein possess circadian rhythm under a normal light-dark (LD) cycle. Moreover, the oscillation has deteriorated under the dim-light-at-night (dLAN) cycle. To investigate how light signals regulate the NOD2 oscillation, we performed two set of experiments. First we conditionally knock out the peripheral clock gene, Bmal, in intestine epithelial cells and. Suprisingly, without the peripheral clock, diurnal rhythms of NOD2 and its downstream are sustained in the intestine epithelium. Due to the peripheral clock could not regulate the oscillation of the NOD2 pathway, we next examine the central clock, the suprachiasmatic nucleus (SCN). By lesioning SCN, we discovered that the diurnal oscillation of the is dampened. Together, our results suggest that the daily oscillation of gut microbe is control by central clock and external light dark cycle and NOD2 pathway, but not intestine clock.

投稿學會：台灣基礎神經科學學會 | 編號: P1-26

Nesting behavior induced alterations in the gut microbiota of two passerine bird species

Cheng-Yu Chen 1,2, Chih-Ming Hung 1, and Shih-Kuo Chen 2

1 Biodiversity Research Center, Academia Sinica 2 Department of Life Science, National Taiwan University

Abstract

The bidirectional neurohumoral communication of gut-brain axis is believed to integrate the gut and central nervous activities mediated by gut microbiota, and thus the concept of the microbiota-gut-brain axis is emerging. Recent studies have shed light on how gut microbiota affect animal behavior via the alterations of several neurological processes such as neurotransmitter turnover, neurogenesis, and neuronal morphology. Specifically, the oxytocin system and parenting behavior has been well-studied in rodent. However, little is known about how microbiota-gut-brain axis influence the parenting behavior prior to, and in the absence of, the stimuli associated with parturition, egg laying or offspring. Nesting behavior in birds is one of the robust early parenting behaviors to study the modulation effect of gut microbiota before the birth of offspring. In this study, to investigate the association among the gut microbiota, neuron activity and nesting behaviors, a series of nesting behavior experiment was conducted using the avian model, zebra finch (Taeniopygia guttata) and society finch (Lonchura striata domestica). Using 16S rRNA amplicon sequencing, we found that the composition of finch gut microbiota variated between a nesting and two non-nesting control groups in both finch species. In addition, certain nesting behaviors, such as duration of nest material fetching in males and time spent in the nesting box in females, appeared to correlate with the variation of gut microbes. Among the gut microbial communities, a bacterial family Campylobacterota, previously known as Epsilonproteobacteria, became the predominant in nesting finches. While the usage of nest material increased after four-days Gram-negative-specific antibiotic treatment compared to the finches drinking water. Collectively, these results suggest that gut microbial signals are closed related for programming of nesting behaviors in the passerines.

投稿學會：台灣基礎神經科學學會 | 編號: P1-27

Intrinsically photosensitive retinal ganglion cells inhibit socio-sexual recognition memory through supraoptic oxytocin neurons

Po-Yu Liao, Yu-Fan Huang, Jo-Hsien Yu, Shih-Kuo Chen

National Taiwan University (Life Science Department)

Abstract

Social memory between the same gender or even different gender is a complex and heavily modulated process in the nervous system. The regulation of memory formation could be influenced by both the internal status of the animal and the external environmental condition. Among many external stimulations, light profoundly affects physiology, behavior, and cognitive functions in humans and rodents. It has been shown that various forms of recognition memory, such as odor and object memory, are downregulated by acute light exposure in rodents. However, the neuronal circuitry involved in light-dependent social recognition memory modulation remains unclear. Here, we show that acute light exposure could impair the socio-sexual recognition memory (SSRM) in male mice. Activating oxytocin neurons in the SON (SONOT neurons) using channelrhodopsin is sufficient to enhance the SSRM performance in male mice. On the contrary, light exposure could inhibit SONOT neurons through M1 SON-projecting ipRGCs and GABAergic neurons (pSONGABA) in the pSON. Together, these results show that sensory input such as light could modulate SSRM through a minimal ipRGCs-pSONGABA-SONOT neuronal circuitry. Our findings demonstrate the neural basis of how luminance affects cognitive functions through the oxytocin system, which is a powerful modulatory neurohormone in the central nervous system.

投稿學會：台灣基礎神經科學學會 | 編號: P1-28

Positive correlations between social hierarchy and memory in weaning mice and young children

Yu-Kai Ma, Yi-Han Lu, Yu-Ju Chou, Tsung-Han Kuo

1 Institute of Systems Neuroscience, National Tsing Hua University, Taiwan 2 Department of Early Childhood Education, National Tsing Hua University, Taiwan

Abstract

Social hierarchy is prevalent in various animal groups and plays an essential role in maintaining the social structure. Most of the current research on mice focuses on the social hierarchy of adult but not young mice. Since young mice do not perform aggressive behavior, we used the tube test to establish the first social hierarchy for weanling mice and examined a variety of behaviors across social ranks. Surprisingly, there is no correlation between the social ranks and mobility, depression, and anxiety phenotypes of young mice. In contrast, the Novel Object Recognition Test and Y maze showed that the weanling mice with a higher social hierarchy have better memory ability. From the qPCR results, we also found higher expression of memory-related genes in mice with higher rank. Next, we injected memory-improving drugs into mice and found that mice treated with drugs showed not only better memory but also social dominance, further support the relationship between social hierarchy and memory. Lastly, our findings in mice led us to examine this phenomenon in preschool children. Consistent with our findings in weanling mice, children with higher dominance level also showed better memory ability than subordinate children. Our study shows remarkable similarities between humans and mice in the positive correlation between memory and social hierarchy. These findings may provide new perspectives on social interaction research in the fields of basic biology, psychology, and education.

投稿學會：台灣基礎神經科學學會 | 編號: P1-29

A research proposal for investigating the role of microtubule dynamics during long-term memory formation in Drosophila

Wen-Jie Chen(1,2), Xuejiao Tian(3), Bi-Chang Chen(3), Suewei Lin(1)

(1). Institute of Molecular Biology, Academia Sinica, Taiwan (2). Taiwan International Graduate Program in Interdisciplinary Neuroscience, National Cheng Kung University (3). Research Center for Applied Sciences, Academia Sinica, Taiwan

Abstract

Microtubules maintain neuronal survival and functions by physically supporting neuronal morphology, constituting tracks for cargo transportation, and mediating local signaling events. Previous studies implicate that dynamic microtubules are important for learning and memory by participating in the molecular mechanisms of synaptic plasticity. However, it is unclear how microtubules in engram cells contributes to memory formation. Here, we propose to investigate the role of dynamic microtubules in the well-established olfactory memory circuit of fruit flies. We will apply expansion microscopy to detect and examine whether microtubules in engram cells are remodeled or distinctively modified during LTM formation. In addition, we endeavor to adapt optogenetic tools to acutely control microtubule remodeling in adult fly's brain, which may allow us to directly study their role in LTM formation.

投稿學會：台灣計算神經科學學會	編號: P1-30

The hybrid wiring pattern in a fly brain diversifies olfactory information processing

Ching-Che Charng, Lishan Cheng, Ting-Kuo Lee, Chung-Chuan Lo, Ann-Shyn Chiang

Institute of Systems Neuroscience, National Tsing Hua University, Hsinchu, Taiwan

Abstract

Olfactory information is processed in the lateral horn, LH, and the mushroom body, MB, for innate and learned behaviors, respectively. The wiring pattern in LH is conserved and can be characterized into different groups for specific odor groups. However, the debates over how olfactory projection neurons connect to the intrinsic mushroom body neurons, Kenyon cells, last for decades. Previous studies claim that the wiring pattern is random for better memory capacity by random sampling. However, from a development angle, these two neuron types have spatial innervation preferences. The anatomic structure offers the possibility that the connection pattern is not fully random. Thus, disputes do not come to an end. To answer the question, we analyze the most resolved neuronal connection data – the FlyEM dataset which offers hemibrain data of one individual brain with synaptic data. Our results suggest that the circuitry has preference instead of full randomness and it is supported by spatial innervation preference. Furthermore, there is a hybrid connection pattern that confers the sensitive detection and experience generalization to food odors. On the other hand, for other odors, the pattern maintains a higher memory capacity. To sum up, the mushroom body performs different strategies for different odors.

投稿學會：台灣基礎神經科學學會 | 編號: P1-31

Exploring the osmosensing mechanism in a pair of thirst-broadcasting neurons

Chien-Tzu Lin, Suewei Lin

Institute of Molecular Biology, Academia Sinica

Abstract

Thirst is an internal state driven by increased blood osmolality resulting from dehydration. It motivates the animal to seek and drink water to restore body fluid homeostasis. Loss of fluid balance affects essential functions of animals which could threaten survival. Therefore, a robust osmosensing system is required to detect the fluid imbalance and elicit thirst responses. Here, we use the fruit fly Drosophila to study the osmosensing mechanism. Like mammals, the fly also exhibits thirst-driven behaviors. Our previous works discovered a pair of Leucokinin-expressing neurons (LHLK neurons) in the fly brain that responds to increased extracellular osmolality and regulates thirst-driven behavior. However, the underlying molecular mechanism that confers osmosensitivity to these neurons remains unclear. To study this question, we have first established a real-time imaging system to monitor Ca2+ signals in LHLK neurons under hypertonic stimulations ex vivo. We found that hypertonic stimulations increased the Ca2+ level in LHLK neurons, and the dynamics of the Ca2+ signals were highly correlated with the shrinkage of the cell body. This observation agreed with the long-standing hypothesis that osmosensation is mediated by mechanical sensors that detect membrane tension. Through an RNAi screening, we identified Pickpocket 26 (PPK26), a fly homolog of mammalian Acid Sensing Ion Channel (ASIC), as an osmosensor in LHLK neurons. Knockdown of ppk26 in LHLK neurons lowered the maximum value of the hypertonicity-induced Ca2+ activity without affecting its initial response rate and the cell shrinkage. Therefore, we proposed a two-phase osmosensing model where the less sensitive PPK26 cooperates with a more sensitive sensor to confer the full range of osmosensitivity in LHLK neurons. To further verify our hypothesis, we are now identifying the additional osmosensor in LHLK neurons. Results from this study will advance our understanding of the thirst sensation, a desire we experience every day.

投稿學會：台灣基礎神經科學學會	編號: P1-32

Endolysosomal 2Cl-/H+ exchanger coordinates neuron-glia crosstalk to control synaptic bouton development

Ping-Yen Hsiao1, Ming-Han Fan1, Tsai-Fan Lee1, Jhan-Jie Peng1, Yu-Jung Chen1, Hung-Lun Chen1, Chi-Kuang Yao2

IInstitute of Biological Chemistry, Academia Sinica, Taiwan

Abstract

Neurons and glia are highly polarized to adapt the demand for complex cellular connectivity and communication in the nervous system. Therefore, proper endolysosomal system that dictates the membrane and protein trafficking and turnover is crucial for maintaining such polarity. In mammals, Clcn3 and Clcn4, a subset of the CLC family, are two major 2Cl-/H+ exchangers expressed in the neural endolysosomes. Most importantly, recent clinical studies have identified various types of the mutations in both human CLCN3 and CLCN4, and the probands show severe neurodevelopmental defects leading to developmental delay, intellectual disability, and epilepsy. Similarly, double knockouts of mouse Clcn3 and Clcn4 result in prenatal lethality. Nonetheless, what roles CLCN exchangers play in neurodevelopment and how disease-associated variants dampen such roles are largely unexplored. In this study, through a forward genetic screen, we identify a mutation in clc-c which encodes the sole fly homolog of human CLCN3 and CLCN4. clc-c mutants are deficient in larval and pupal development. Furthermore, the mutants show outgrowth of the larval neuromuscular junction synaptic bouton, characterized as numerous main and satellite boutons. Clc-c is universally expressed, and the bouton defect results from loss of Clc-c in either of neurons, glia, and muscles. Although Clc-c is localized in distinct compartments of the endolysosomal system, its loss predominantly causes the defects in early and recycling endosomes. Mechanistically, we find that the elevated Mad signaling underlies the synaptic bouton outgrowth. Most intriguingly, disease-associated human CLCN4 variants exhibit partial/full activity, loss of function, or dominant-negative effect in a cell-type specific manner. Lastly, when expressed in the fly visual system, we can provide much precise diagnosis for defining the severity of human CLCN4 variants. Overall, our work reveals that the endosomal 2Cl-/H+ exchanger regulates neuron-glia communication during synaptic bouton development, and its pathogenic human variants hold differential activities in different cells.

投稿學會：台灣基礎神經科學學會	編號: P1-33

Modeling frequent human variants of Flower reveals its regulation on synaptic vesicle recycling and synaptic bouton architecture through distinct mechanisms

Ting-Yi Lu1*, Shih-Han Lin1*, Nguyen Hong Ngoc1, Yu-Jung Chen1, Aisha Mohamed Saeed Mohamed Al Shamsi2, Jill Rosenfeld Mokry3, Hugo Bellen4, Chi-Kuang Yao5

Institute of Biological Chemistry, Academia Sinica, Taiwan

Abstract

The activity-dependent change in synaptic structure and activity is essential for the plasticity of neural network. Endocytosis of the synaptic vesicle (SV) is tightly coupled to exocytosis which releases neurotransmitters to keep neurotransmission continuity. Mild activity stimulations elicit clathrin-mediated endocytosis (CME), while intense stimuli triggers activity-dependent bulk endocytosis (ADBE). Therefore, such stimuli-dependent mode switch is able to boost synaptic strength upon demand, yet the underpinning mechanism remains elusive. Here we investigate structure-function relationship of the Flower Ca2+ channel which promotes CME and ADBE respectively through channel-dependent and -independent functions. By characterizing the functional equilibrium between alternative splicing isoforms of Flower and its human homolog, CACFD1, in the fly system, we found that CACFD1 isoform 4 can function as Flower. Moreover, both their transmembrane domain region and cytosolic region are necessary for their sorting and localization to the SV. Furthermore, through the sequencing databases for healthy human population and the patients suffering from various disorders, numbers of frequently carried CACFD1 variants which result from missense mutations in evolutionarily conserved residues mainly spanning transmembrane domain region, as well as copy number gain are identified and subsequently studied in their functional impact. Interestingly, we found that these variants display the differential loss of the abilities to control SV endocytosis but also synaptic bouton formation. Hence, our work provides new mechanistic insights into the coordination between synaptic activity and structure. In addition, our data reveal that these CACFD1 variants may have potential pathogenic impacts on human health.

投稿學會：台灣基礎神經科學學會	編號: P1-34

Studying the atypical localization of the centrosomal protein Cep170 during neuronal morphogenesis

Peng-Tzu Chen 1,2, Angela Goh 1, Han-Chiang Huang 1,2, Eric Hwang 1,2,3,4*

1 Department of Biological Science and Technology, National Yang Ming Chiao Tung University 2 Institute of Molecular Medicine and Bioengineering, National Yang Ming Chiao Tung University 3 Institute of Bioinformatics and Systems Biology, National Yang Ming Chiao Tung University 4 Center for Intelligent Drug Systems and Smart Bio-devices (IDS2B), National Yang Ming Chiao Tung University

Abstract

Microtubules are essential cellular structures in neurons and participate in essentially every step of neuronal development. Proteins that interact with microtubules are called microtubule-associated proteins (MAPs) and affect neuronal development. Using quantitative proteomics to compare MAPs from undifferentiated stem cells and stem cell-derived neurons, we found that Cep170 is more enriched on neuronal microtubules. Cep170 is a forkhead-associated (FHA) domain-containing centrosomal protein and localizes to the subdistal appendage of the centriole in mitotic cells. Its mutations have also been found to be associated with human brain abnormalities, such as microcephaly and lissencephaly. These indicate that Cep170 plays an important role in the development of the central nervous system. To understand the role of Cep170 in neurons, loss- and gain-of-function experiments were conducted. The result shows that Cep170 promotes neurite elongation in both stem cell-derived neurons and primary neurons, and both axons and dendrites are promoted. However, no morphological phenotypes can be observed when Cep170 is depleted. We observed that Cep170 exhibits distinct localizations in neurons: 1) a single spot in the soma, 2) as discrete puncta along the neurite, 3) enriched at the tip of the axon. To find out how these distinct localizations affect neuron development, different truncations of Cep170 which compromise specific aforementioned localizations were overexpressed in neurons. The result shows that the punctate distribution along the neurite requires both the microtubule-binding domain and the FHA domain, while the tip enrichment is depended on the FHA domain. Also, we discovered that microtubule stability affects the distribution of Cep170 puncta along the neurites. On the other hand, the Cep170 localization at the axon tips is independent from microtubules.

投稿學會：台灣計算神經科學學會 | 編號: P1-35

Degree of dispersion in the cerebral cortex and the correlation with mental illness

Shu-Fei Lin, Albert Chih-Chieh Yang, Pei-Shan Hou

Institute of Anatomy and Cell Biology, School of Medicine, National Yang Ming Chiao Tung University Institute of Brain Science, School of Medicine, National Yang Ming Chiao Tung University

Abstract

Schizophrenia is a mental disorder which have high prevalence rate in global, approximately 1%. Symptoms of schizophrenia are complex include hallucination, delusion, lack of ability to normal social expression and some cognitive issue. Despite the known symptoms to identify and characterize schizophrenia, the pathological causes remain largely unclear. Magnetic resonance imaging (MRI) analyses provided structural related measurements of brain, these measurements could be catalogued by the surface-based and volume-based analyses. While grey matter volume or subcortical volume can be examined by volume-based analysis, surface-based analysis allowed us to evaluate the cortical thickness or local gyrification index of the distinct coordinate on the surface of cerebral cortex. To date, studies and clinical cases have identified several structural abnormalities in the brain of schizophrenia patients. Grey matter volume and cortical thickness showed a regional-specific different in schizophrenic cases, especially in frontal and temporal lobe. Nonetheless, our complex cerebral structures, such as gyrification pattern, are highly associated with the map of functional areas regions and multiple functional areas compose a single lobe. Here, to decipher the relationship between structural changes and functions, we performed variation analysis of neighbouring voxels or vertices using T1-weighted MRI analysis of subjects included 331 normal controls and 194 cases with Brodmann area and Destrieux Atlas. Based on our results, we identified several functional areas affected in schizophrenia and further genetic association test illustrate the possible genetic cause of schizophrenia.

投稿學會：台灣基礎神經科學學會 | 編號: P1-36

Genetics association between schizophrenia and cerebral functional areas

Chih-Yun Chung, Albert Chih-Chieh Yang, Pei-Shan Hou

1 Institute of Anatomy and Cell Biology, School of Medicine, National Yang Ming Chiao Tung University 2 Institute of Brain Science, School of Medicine, National Yang Ming Chiao Tung University

Abstract

Schizophrenia is a mental disease that impacts people's ability to express symptoms like delusion, hallucination, and no interest in social activities; the cause of the disease still remains unclear. In the past, magnetic resonance imaging (MRI) studies of schizophrenia, have shown some abnormalities in cortical structures compared to normal control, especially such as decreased grey matter volume in the prefrontal cortex and temporal lobe and temporal lobe. However, each lobe contains multiple functional areas and the phenotype of schizophrenia may not be resulted from a single area, which lets us hypothesize that the different combination of brain functional areas might be involved in the causes the symptoms of schizophrenia. Here, we performed in silico analysis of T1-weight MRI of 331 healthy control and 194 schizophrenia patients from the TAMI database. Global and regional cerebral grey matter volume differences between healthy control and schizophrenia patients were analyzed based on automated anatomical Labeling (AAL atlas) and Brodmann areas (BAs). Following group comparison and a general linear model using voxel-wise t-test analyses showed total grey matter volume was significantly decreased in schizophrenia patientspeople with schizophrenia. Our results showed multiple functional BAs were significantly affected in the cerebrum of schizophrenia patients. In addition, further investigations using a genome-wide association study (GWAS) suggested certain SNPs may affect the grey matter volume of the distinct BAs. Together with the expression pattern, these data will may provide insights in the cause of schizophrenia.

投稿學會：台灣基礎神經科學學會 | 編號: P1-37

To investigate the role of Akt1, a schizophrenia candidate gene, in reward-based decision making- neural mechanism of dorsomedial striatum from multidisciplinary approaches

Chia-Yuan Chang1,3, Ching Chen1, Tsung-Ren Huang1, Wen-Sung Lai1,2,3

1Department of Psychology, National Taiwan University, Taipei, Taiwan 2Graduate Institute of Brain and Mind Sciences, National Taiwan University. 3Neurobiology and Cognitive Science Center, National Taiwan University, Taipei, Taiwan

Abstract

Schizophrenia (SZ) is a neuropsychiatric disorder with severe cognitive deficits. Accumulating evidence from human genetic studies suggests that multiple susceptibility genes contribute to the pathogenesis of SZ, including AKT1, a kinase intermediate downstream of dopamine D2 receptor. Alterations of dopaminergic transmission have been implicated in the pathogenesis of SZ, and SZ patients also show worse performance than healthy controls in many decision-making tasks. Recent findings reveal Akt1 participates in the modulation of reward-based decision making, especially in the striatum. However, the importance of Akt1 in dorsomedial striatum (DMS) during decision making remain elusive. To this end, we proposed 4 specific aims in this study. (1) the impact of Akt1 in a probabilistic 2-choice-foraging-task. (2) The role of Akt1 in the regulation of DMS neural activity during different stages of decision-making. (3) The causal relationship between DMS neural activity and decision-making behavior. (4) The cell-type-specific role of DMS neurons in decision-making. Our results indicated that (1) Akt1 HET mice required fewer trials to achieve the criteria and higher ratio of win-stay behavior compared to WT. Taking advantage of a Bayesian approach to estimate the parameters in modified reinforcement learning model, we found that HET mice have a higher learning rate in the no-reward outcomes and lower choice consistency. (2) The in vivo recording showed that no-reward-evoked DMS LFP power is highly correlated with their behavioral performance and model parameters. The decoding of DMS-LFP by machine-learning reveals the choice-related information is embedded in DMS neural activity and it also shows the genotypic difference. (3) The direct inhibition of DMS by the chemogentics in HET mice make WT-like behavior, including higher accumulated trials and lower ratio of win-stay behavior. (4) The behavioral results from cell-type-specific modulation in DMS through parvalbumin-cre mice showed that the lesion of PV interneurons made the higher ratio of win-stay behavior, higher learning rate and lower choice consistency. Collectively, our results suggest Akt1 deficiency cause the alternation of neural oscillation in the DMS potentially through parvalbumin-interneurons in DMS, which is contributed to the learning rate which resulted in the differential selection of choice strategy during decision making.

投稿學會：台灣基礎神經科學學會 | 編號: P1-38

The morphological characteristics of vasopressin neurons in zebrafish brain

Fang Zhi Chua, Ming-Yi Chou

Department of Life Science, National Taiwan University

Abstract

Arginine vasopressin (AVP) is a nonapeptide produced by the hypothalamus and secreted from the pituitary. Vasopressin is well known for its functions as a hormone to regulate water reabsorption and ion balance. Besides, vasopressin also acts as a neurotransmitter and modulate various social behavior, especially aggression. Previous study in the bluehead wrasse showed that injected vasopressin increases aggression in non-territorial males while decreasing aggression in territorial males. However, the morphology and function of vasopressin meurons on teleost fish remain unclear. In the present study, we use zebrafish (Danio rerio) as model animal to study the morphology of vasopressin neurons and their effects on aggressive behavior. We first investigate the distribution of vasopressin neurons and their cell type in the whole zebrafish brain. Vasopressin neurons in the zebrafish brain are located in the anterior part of parvocellular preoptic nucleus (PPa), magnocellular preoptic nucleus (PM), ventromedial thalamic nucleus (Vm) and posterior part of parvocellular preoptic nucleus (PPp). Our immunohistochemistry/in situ hybridization results found that, about 9.5% of vasopressin neurons are also oxytocinergic neurons but not glutamatergic neurons, GABAergic neurons, and dopaminergic neurons. To evaluate the neural activity of vasopressin neurons, we also perform various behavior tests under 2 different durations of environmental stress (1hr and 4hr 5mM NH4+ exposure) on the zebrafish and observe the behavior changes. We performed mirror biting test and found that the biting number of the zebrafish is significantly reduced after 1hr stress exposure but did not affect after 4hr stress exposure. These indicate that the aggressiveness of zebrafish is reduced after stress exposure. Both environmental stresses did not affect anxiety levels and social preference in zebrafish. Overall, our study provides insight into the cell type, projection, and function in central nervous system of vasopressin neurons on teleost fish.

投稿學會：台灣基礎神經科學學會 | 編號: P1-39

Tracking the movement of zebrafish larrve by multispectral imaging system

Hsi Chen Bo-Kai Liao Ming-Yi Chou

Department of Life Science, National Taiwan University, Taipei, Taiwan Department of Aquaculture, National Taiwan Ocean University, Keelung, Taiwan

Abstract

In social behavior, a visual signal is an important factor. Zebrafish larvae exhibit shoaling behavior at 14 days post fertilization. They show preference for specific shoaling partners according to the specific characteristics in the skin such as body color and the pattern of stripes. However, the development of choosing a preferred partner is unknown. Also, the size of zebrafish larvae is small so it is difficult to obtain detailed information of the tiny structure of larvae that allows us to distinguish the different individuals. Compared to adult fish, the threshold for image analysis is high, and under normal camera shooting, the errors of computer tracking occur frequently. Multispectral imaging in the invisible light bands is known for non-invasive detection characteristics and can be used for freshness qualifying products. However, commercial hyperspectral cameras are costly. We designed a low-cost multispectral imaging system in the present study with a 3D printed outer casing, 24-wavelength LEDs, a microcontroller unit, and a no IR-cut digital camera. We collected a multispectral data cube by imaging in 24 wavelengths and 1 backlight for the contours of samples in this multispectral imaging system. Taking the wildtype zebrafish as the standard, multispectral data cubes were imaged for deep learning. The data cubes were first subjected to background removal, ROI extraction, and Hyperstack pre-processing, and then feature compression was accomplished by an encoder-decoder convolutional neural network (E-D CNN) for establishing a spectral map and morphological feature database. This multispectral imaging system and deep learning model developed in the present study would be utilized in zebrafish larvae social behavior analysis.

投稿學會：台灣基礎神經科學學會	編號: P1-40

Neural circuit remodeling in the primary motor cortex during continual motor learning

Poulomi Adhikari1,2,3, Shuo-Yen Chueh2,3, Tzu-Hsien Liu2,4, Ping-Yen Wu2,3, Yu-Wei Wu1,2,3,4*

1 Taiwan International Graduate Program in Molecular Cell Biology, National Defence Medical Centre and Academia Sinica, Taipei 112, Taiwan 2Institute of Molecular Biology, Academia Sinica, Taipei 115, Taiwan 3Neuroscience Program in Academia Sinica (NPAS), Academia Sinica, Taipei, Taiwan 4Department of Life Science, College of Life Science, National Taiwan University, Taipei, Taiwan

Abstract

Motor learning incorporates rapid formation and selective stabilization of synapses in the primary motor cortex (M1). Therefore, learning multiple tasks requires continual synaptic re-organization. However, it is unknown how re-organization of a part of the network does not lead to a severe loss of previously learned information, a phenomenon called catastrophic forgetting, which is a challenging issue in training artificial neural networks. Here we have established a mouse model of continual learning paradigm that is quantifiable, comparable, and has a different spectrum of difficulty. We adopted a programmable joystick that enables pre-determined movement trajectories of various lengths, turning angles, and artificial friction. The mice were trained to operate the joystick with their forearm to complete designed movement trajectories to obtain a sucrose reward. We established a relationship between task difficulty and behavioral performances by monitoring the trial attempts, success rates, and movement accuracy. We trained continually with three different tasks and simultaneously monitored the dendritic morphology of the pyramidal neurons in the M1 of the contralateral side of the movement arm with in vivo two-photon microscopy through a chronically implanted cranial window. We found that mice that learned the tasks have higher synaptic dynamics than non-learner control. Moreover, synapses have a high turnover during the first task, but learning multiple tasks reduces the turnover. Overall, we demonstrate that the synapses become more persistent with each subsequent task, even though the later tasks are more complicated than the first task. The new insights from this study will enable us to understand the underlying mechanism of circuit remodeling and retention of pre-existing information in the neural network.

投稿學會：台灣基礎神經科學學會 | 編號: P1-41

The functional role of striatal projection neurons in the striosome in encoding motor and reward information during motor learning

Shuo-Yen Chueh, Yu-Wei Wu

1. Institute of Molecular Sciences, Academia Sinica, Taipei 115, Taiwan 2. Neuroscience Program in Academia Sinica (NPAS), Academia Sinica, Taipei 115, Taiwan 3. Department of Life Science, College of Life Science, National Taiwan University, Taipei 106, Taiwan

Abstract

Optimizely acquiring motor skills by reward-based feedback is a fundamental instinct to enable the animal to achieve its goal effectively. As the input station of the basal ganglia, the striatum incorporates and pre-processes the information related to both reward and motor skills and transmit to the downstream circuit to enforce the learning performance. The early studies show that the striatal projection neurons (SPNs) in the different striatal compartments, the striosome and the matrix, play a crucial role associated with reward. Different cell-types of SPNs in the dorsolateral striatum (DLS) could develop a unique firing pattern in responsible to different motor skills. However, how does the SPNs in the striosome represent the information of reward driven motor skills is not clear. Here we have established a uniform paradigm and different degrees of complexity lever-pushing task machine to train the mice with fluorescent calcium indicator and using in vivo two-photon imaging via a gradient refractive index (GRIN) lens to observe the calcium signals in the SPNs of striosome in DLS on well-trained mice. We show that, in the striosome, different cell populations of SPNs are represented to different factors of the motor tasks i.e. pushing velocity, licking rate, received reward, and amount of reward. These representatives also demonstrate that, in the different cell populations with the diverse task complexity, the striosome circuit is involved in processing the information of motor skills and reward. The discovery of motor skills and reward information encoded in the SPNs of striosome is a robust niche to further explore the shaping mechanism of reward-feedback to enhance the motor skills development.

投稿學會：台灣基礎神經科學學會 | 編號: P1-42

Causal interactions between neuronal activities in midbrain dopaminergic neurons and basal forebrain neurons

Ming-Ching Chiang, You-Zhe Jhong, Mu-En Kuo, Shih-Chieh Lin

Institute of Neuroscience, National Yang Ming Chiao Tung University

Abstract

Reward prediction error (RPE), the difference between received and predicted reward, has been traditionally associated with neuronal activity in midbrain dopaminergic (DA) neurons in the ventral tegmental area (VTA). However, recent studies from our group have demonstrated that a special subset of noncholinergic neurons in the basal forebrain (BF), which we refer to as BF bursting neurons, are similarly modulated by RPE. In the accompanying poster, we show that the activities of BF and VTA neurons are functionally coupled in terms of both response amplitude and spike timing, with DA neuron spiking temporally leading BF spiking by roughly 10 msec. These observations raised the question of whether DA activity directly can cause the spiking of BF neurons. To test this idea, we selectively excited VTA DA neurons using optogenetic tools in DAT-cre mice while recording neuronal responses in the BF. Here we show that optogenetic activation of VTA DA neurons induced robust and transient activation of many BF bursting neurons within 5-15 msec. Increasing the frequency of DA neuron activation led to slower and less reliable BF responses, including decreased spiking fidelity, increased spiking latencies and variabilities. These effects were further exacerbated by isoflurane anesthesia. Together, these results revealed a novel and strong causal coupling between VTA DA neurons and BF bursting neurons, acting perhaps through a poly-synaptic pathway. This surprising finding suggests that the brain uses two major neuromodulatory systems to jointly encode reward prediction error.

投稿學會：台灣基礎神經科學學會 | 編號: P1-43

14-3-3 proteins regulate Eag1 K+ channel degradation mediated by Cullin 7

Chang-Heng Hsieh1, Chia-Cheng Chou2, Chih-Yung Tang3, and Chung-Jiuan Jeng1

1Institute of Anatomy and Cell Biology, College of Medicine, National Yang Ming Chiao Tung University, Taipei, Taiwan; 2National Laboratory Animal Center, National Applied Research Laboratories, Taipei, Taiwan; 3Department of Physiology, College of Medicine, National Taiwan University, Taipei, Taiwan

Abstract

The ether-à-go-go (Eag) potassium (K+) channel belongs to the superfamily of voltage-gated K+ channel. The neuron-specific voltage-dependent Eag1 potassium channel are linked to congenital neurodevelopmental diseases. Disease-causing mutant Eag1 channels manifest aberrant gating function and defective protein homeostasis. Both the E3 ubiquitin ligase cullin 7 and the small acid protein 14-3-3 serve as Eag1 binding partners. Cullin 7 mediates proteasomal and lysosomal degradation of Eag1 proteins. 14-3-3 proteins modulate the functional expression of the K+ channel in neurons. Co-expression with difopein, a peptide inhibitor of 14-3-3 proteins, increased mature and immature Eag1 by repressing protein degradation. Endogenous 14-3-3 proteins also contribute to excitotoxicity-induced proteasomal degradation of Eag1 protein in neurons. Inhibition of endogenous 14-3-3 function effectively perturbs the interaction of Eag1 with cullin 7. Further studies suggest that 14-3-3 proteins play a critical role in facilitating the binding of cullin 7 to two cytoplamsic domains of Eag1, both of which are essential for Eag1 degradation by cullin 7. These findings demonstrate a regulatory role of 14-3-3 in cullin 7–mediated Eag1 degradation in neurons.

投稿學會：	編號: P1-44

Abnormal ankle proprioceptive acuity in young adults with a probable developmental coordination disorder

Yi-Hsuan Lin1, Yi-Wen Chen1, Yu-Ting Tseng1,2*

1Department of Kinesiology, National Tsing Hua University, Hsinchu City, Taiwan 2Research Center for Education and Mind Sciences, National Tsing Hua University, Hsinchu City, Taiwan

Abstract

Background: Published literature has suggested that adults with a probable developmental coordination disorder (pDCD) showed abnormal upper limb position sense of the wrist. However, it is still unclear if the lower limb (i.e., ankle) proprioceptive function is impaired in adults with pDCD. Objective: This study aims to determine ankle position sense and its relation to different domains of motor functions in young adults with or without pDCD. Methods: Participants were young adults (N=28) aged from 19-22 years old, including 10 pDCD participants and 18 heathy controls. Young adults with pDCD were screened by the Bruininks − Oseretsky Test of Motor Profciency 2nd Edition (BOT-2) that total standard score of < 40 (<17th) which equals one standard deviation below the mean. Ankle position sense acuity was assessed using joint position sense matching paradigm under contralateral and ipsilateral conditions. Position sense error (PE) and position sense error variability (SDPE) were obtained to measure ankle position sense acuity. Result: The results showed that young adults with pDCD exhibited a significantly increased SDPE on both contralateral and ipsilateral conditions ($p < .05$) when compared to controls. In addition, the ankle position sense SDPE was significantly correlated with levels of fine manual control ($r = - .46$, $p = .014$), manual coordination($r = - .54$, $p = .003$), agility & strength ($r = - .52$, $p = .005$) and total score ($r = - .55$, $p = .002$) measured by BOT-2. That is, young adults with a more decreased position sense precision tended to show lower levels of motor proficiency. In summary, these results confirmed that impaired ankle position sense acuity in young adults with pDCD, which could contribute to motor abnormalities in young adults with pDCD.

投稿學會：	編號: P1-45

The relationship between motor proficiency and lower limb proprioception in healthy young adults

Yi-Wen Chen a, Yi-Hsuan Lin a, Yu-Ting Tseng a,b*

a Department of Kinesiology, National Tsing Hua University, Hsinchu, Taiwan b Research Center for Education and Mind Sciences, National Tsing Hua University, Hsinchu, Taiwan

Abstract

Proprioception allows humans to perform precise and coordinated movements by repeatedly updating information about limb and body movements. Empirical evidence has shown that proprioceptive sensitivity is closely related to motor proficiency. Still, it remains unclear how neural correlates underlie proprioceptive-motor processes and to what extent these measures correlate with a person's motor abilities. The aim of this study was to examine the behavioral and neurophysiological performances during the ankle proprioceptive-motor tasks, and how they are related to individuals' levels of motor proficiency. A total of 29 healthy young adults aged 20.45±1.06 years were recruited. All participants were asked to place their dominant foot on the paddle of the ankle apparatus, which passively moved the ankle joint at constant velocities of 22°-1. Participants were required to press the button held by the dominant hand as quickly as possible once they sensed the motion produced by the apparatus. We evaluated behavioral measures (i.e., motion detection time, MDT; its standard deviation, MDT-SD) and P3 components related to the proprioceptive-motor task. The motor function was examined by the Bruininks-Oseretsky Test of Motor Proficiency 2nd edition long-form (BOT-2, LF). Spearman's rank correlation coefficient analysis revealed that standard scores of BOT-2 total motor composites significantly correlated with the MDT ($r = -.615$, $p < .001$) and MDT-SD ($r = -.518$, $p = .004$). Furthermore, ERP findings revealed a significant negative correlation between P3 amplitude and MDT during the ankle motion detection task ($r = -.610$, $p < .001$) and MDT-SD ($r = -.665$, $p < .001$). The total scores of BOT-2 also significantly correlated with P3b amplitude ($r = .457$, $p = .025$) and P3b latency ($r = -.569$, $p = .004$). The results suggested that levels of motor proficiency are correlated with motion detection time and P3b component related to ankle proprioceptive tasks in young adults. Individuals with higher levels of motor coordination tended to show higher levels of ankle proprioceptive sensitivity that is associated with a greater ability to allocate proprioceptive signals.

投稿學會：台灣基礎神經科學學會	編號: P1-46

Immunoproteasome inhibition modulates microglial polarization in erythrophagocytosis

Wei-Fen Hu1, Mu-Ting Shih2, Pei-Chen Han2, Hsin-Ru Liu3, Shaik Ismail Mohammed Thangameeran4, Chia-Ho Lin1, Cheng-Yoong Pang3,4,5, Hock-Kean Liew1,3,5*

1 PhD Program in Pharmacology and Toxicology, Tzu Chi University, Hualien 970, Taiwan 2Department of Laboratory Medicine and Biotechnology, Tzu Chi University, Hualien 970, Taiwan 3 Department of Medical Research, Hualien Tzu Chi Hospital, Buddhist Tzu Chi Medical Foundation, Hualien 970, Taiwan 4 Institute of Medical Sciences, Tzu Chi University, Hualien 970, Taiwan 5 Neuro-Medical Scientific Center, Hualien Tzu Chi Hospital, Buddhist Tzu Chi Medical Foundation, Hualien 970, Taiwan

Abstract

Objective: Intracerebral hemorrhage-induced proteostasis disturbance/proteasome over-activation exacerbated ER stress and neuroinflammation, leading to neurological deficits. Immunoproteasome plays an important role in various neurological diseases under inflammatory and pathological conditions. The microglia M1/M2 functional phenotypes polarization may either exacerbate damage or induce repair and regeneration depending on different signals received by microglial receptors during pathological conditions. We intend to study the effect of immunoproteasome inhibition in facilitating erythrophagocytosis and microglia polarization in vitro ICH model. Materials & Methods: Rat primary microglia and BV2 cells and fluorescently labeled erythrocytes were used to study erythrophagocytosis in vitro with microglia phenotypic changes quantified by M1 (iNOS, CD86, CD16) and M2 (Arginase-1, CD163, CD206) gene expression, immunofluorescence staining, flow cytometry, and Western blot analysis. Results: Both flow cytometry and western blot showed that immunoproteasome inhibition by ONX-0914 (100 nM) increased microglia phagocytosis activation and promoted microglia activation toward M2 polarization accompanied by ROS and inflammatory cytokines reduction. Conclusion: Immunoproteasome inhibition may achieve neuroprotective effects through erythrophagocytosis by M2 microglia polarization and may lead to a novel treatment strategy for intracerebral hemorrhage recovery.

投稿學會：台灣基礎神經科學學會	編號: P1-47

Ischemia-induced M2-to-M1 transition in microglia was blocked by leptin that was independent from autophagy inhibition

Sha-Hua Ye, Chi-Mei Hsueh

Dept. of Life Sciences, National Chung Hsing University, Taichung City, Taiwan The iEGG and Animal Biotechnology Center, NCHU, Taichung City, Taiwan

Abstract

We previously demonstrated that leptin can protect brain from ischemia-induced inflammation. To further understand how leptin protected brain from ischemic insult, leptin's impact upon the ischemia-induced autophagy and M2-to-M1 phenotype transition were investigated in microglia. It is known that brain microglia can sense environmental changes to have either a pro-inflammatory (M1) or an anti-inflammatory (M2) phenotype. Activation of M1 microglia often further exacerbate brain injury whereas of M2 microglia may protect the injured brain. Ischemia-stimulated autophagy has been reported to promote M1 polarization of microglia. We, therefore, were interested to know if leptin blocked the M2-to-M1 phenotype transition in ischemic microglia, by inhibiting of the autophagy. In the study, an in vitro ischemic model (GOSD: glucose-oxygen-serum deprivation) was used to evaluate the impacts of leptin upon ischemic microglia. M1 and M2 microglia were distinguished from each other based on their secretory markers, surface markers and function. The current results showed that M1 markers (CD16, TNF-α, IFN-・) were significantly stimulated by GOSD (2h) whereas M2 markers (CD206, IL-10, TGF-β1) were not, except IL-4. Phagocytic activity of microglia (M2 function) was inhibited by GOSD. Leptin, however, blocked the GOSD-increased expression of CD16, TNF-α, IFN-・, and IL-4 but further elevated the protein expression of CD206 and TGF-β1 as well as the phagocytic activity of M2 microglia under GOSD condition. The above results suggested that ischemic stress promoted the M2-to-M1 phenotype transition in microglia whereas leptin blocked that and provided a less inflammatory environment in ischemic brain. GOSD-stimulated autophagy of microglia was not blocked instead increased by leptin co-treatment, indicating leptin-inhibited M2-to-M1 transition was likely not due to autophagy inhibition. The role of leptin-increased autophagy in GOSD-treated microglia is worth further investigation. In overall, the study has further expanded our knowledge about the mechanism(s) underlying the leptin-mediated protection of ischemic brain. The therapeutic value of leptin in the control of cerebral ischemia is also revealed.

投稿學會：台灣認知神經科學學會 | 編號: P1-48

The Excitatory–Inhibitory metabolite activity in the prefrontal cortex mediates the relationship between sensory responsivity and autistic traits

Yang-Teng Fan1*, Shang-Yueh Tsai2, Chung-Hsin Chiang3, Shih-Han Chou4, Chenyi Chen5, Yu-Chun Chen6, Ching-Ching Wong7, Ovid J. L. Tzeng8,9,10,11

1 Graduate Institute of Medicine, Yuan Ze University, Taoyuan 320, Taiwan 2 Graduate Institute of Applied Physics, National Chengchi University, Taipei 116, Taiwan 3 Department of Psychology and Research Center for Mind, Brain and Learning, National Chengchi University, Taipei 116, Taiwan 4 Department of Physical Medicine and Rehabilitation, Taipei Medical University Hospital, Taipei Medical University, Taipei 110, Taiwan 5 Graduate Institute of Injury Prevention and Control, Taipei Medical University, Taipei 110, Taiwan 6 Department of Physical Education, National Taiwan University of Sport, Taichung 404, Taiwan. 7 Child Developmental Assessment & Intervention Center, Department of Child & Adolescent Psychiatry, Taipei City Hospital, Taipei 103, Taiwan 8 Cognitive Neuroscience Laboratory, Institute of Linguistics, Academia Sinica, Taipei 115, Taiwan 9 Department of Biological Science and Technology, National Chiao Tung University, Hsinchu 300, Taiwan 10 College of Humanities and Social Sciences, Taipei Medical University, Taipei 110, Taiwan 11 Department of Educational Psychology and Counseling, National Taiwan Normal University, Taipei 106, Taiwan

Abstract

An imbalance between excitation and inhibition (E–I) within specific brain areas has been hypothesized to be a major cause of autism spectrum disorders (ASD). Although this theoretical model is promising, the existing empirical evidence is now only partial and investigating the causal roles of balance between brain E–I levels in major characteristics of ASD remains unresolved. In this study, therefore, we recruited 120 adolescents and young adults (28 individuals with ASD and 92 neurotypical controls) through evaluations of sensory features and autistic traits as well as brain magnetic resonance spectroscopy (MRS) measurements. We then stratified all participants based on their levels of sensory responsivity into two subgroups (those with and without sensory over-responsivity, or SOR and NSOR, respectively). Behaviorally, compared to those with the NSOR group, participants with the SOR group were more likely to be diagnosed with ASD. In addition, the SOR group had greater levels of sensory responsivity and more severe autistic traits than the NSOR group. Neuropsychologically, elevated Glx/GABA ratios within the auditory cortex, the somatosensory cortex, and the prefrontal cortex (PFC) were observed in the SOR group, and an increased cortical excitatory in the PFC correlated with both degrees of sensory responsivity and autistic traits. Results of mediation analysis further indicated that levels of brain E–I balance served as a mediator for the severity of sensory features–autistic symptoms relation in the SOR participants. Specifically, an elevated Glx/GABA ratio in the PFC was associated with higher levels of sensory responsivity and more severe of autistic traits. These findings not only provide supportive evidence that alterations in the prefrontal E–I levels have causal influences on ASD-associated symptoms, but also have implications for prognostic predictions of ASD, and, ultimately, to targeted ASD interventions.

投稿學會：台灣認知神經科學學會 | 編號: P1-49

Context effects on visual perception and probability estimation

Hui-Ching Hsu, Wan-Yu Shih, Shih-Wei Wu

Institute of Neuroscience, National Yang Ming Chiao Tung University, Taipei, Taiwan Brain Research Center, National Yang Ming Chiao Tung University, Taipei, Taiwan

Abstract

Many cognitive functions—from perception, learning and memory, to judgment and decision making—are strongly affected by the context of our experience. But do context effects seen in one domain of cognition (e.g. perception) relate to those seen in another domain (e.g. judgment)? While context effects have been extensively studied, few studies compared context effects between different domains of cognition. Characterizing the similarities and differences in how context impacts different cognitive functions can provide insights into the nature of cognition, for example, whether there are common principles that guide context-dependent computations in perception and judgment. To address this issue, in this study human subjects (N=62) performed two tasks, a color perception task and a reward probability estimation task. In the perceptual task, the subjects had to judge the degree of blueness of a colored disc in each trial. The color ranged from completely blue, ambiguous (in between blue and purple), to completely purple. In the probability estimation task, the subjects in each trial had to estimate the reward probability associated with a visual stimulus. Different visual stimuli represented different reward probabilities that ranged from small, medium, to large. Context was similarly manipulated between the two tasks. In both tasks, the subjects faced three different contexts in different blocks of trials. Each context consisted of stimuli that differed in the degree of blueness (perceptual task) and reward probability (probability estimation task). Context was systematically manipulated such that the experience of a single stimulus differed between contexts in the other stimuli that were present in the same blocks of trials. We found similar effects of context across the two tasks. The subjects were more susceptible to the impact of other stimuli present in the same context when they faced a stimulus that carried larger uncertainty—in the perceptual task, it was the ambiguous stimulus whose color was in between purple and blue; in the probability estimation task, it was the stimulus that had 50% chance of reward. Computational modeling of behavioral data suggested that these effects were driven by two computational principles, namely reference dependency and uncertainty dependency, and their interactions. Our results identified the common principles for context-dependent computations in perception and judgment under uncertainty.

投稿學會：	編號: P1-50

Divergent Impacts of Complexity on Spatial and Temporal Sequence Learning

Bing Shan Wu Erik Chih-Hung Chang

Action & Cognition Laboratory, Institute of Cognitive Neuroscience, National Central University

Abstract

Sequence learning refers to improvements in performance through repeating the same series of movements. The current study aims to quantify the impacts of spatial complexity of a sequence on sequence learning in spatial and temporal domains. In the spatial domain, we compared the learning effect in the Serial Reaction Time Task (SRTT) under factorial combinations of different complexity and sequence length. While the learning effect indexed by the RT difference between the Random and Regular sequence is smaller in the high than low complexity, it did not differ between short and long sequences. On the other hand, the trends of learning rates remain the same even under the two different complexities. These results suggest that complexity does modulate the overall amount, but not the speed, of sequence learning. In the temporal domain, the learning was examined by measuring RT to flashes separated by a repeated sequence different time intervals. The comparison between Random and Regular sequences found that, unlike the spatial domain, learning only occurred under the low complexity condition of temporal SRTT. Hence, we speculate that sequence learning might not rely on a general mechanism underlying both spatial and temporal domains. Finally, we applied transcranial direct current stimulation (tDCS) over the contralateral primary motor cortex to inspect the learning of sequences varying both in spatial and temporal complexities, and attempted to tease apart the impacts of tDCS on the learning of each domain. The preliminary outcomes indicate that the primary sensorimotor regions contribute to the learning of the integration of spatiotemporal variation in the motor sequence, and the spatial variation may be the predominant information that is learned.

投稿學會：台灣認知神經科學學會 | 編號: P1-51

The optimization of a novel statistical learning test with hidden temporal and spatial positional regularity

Andhika Renaldi, Denise Wu

National Central University

Abstract

Our previous findings on statistical learning demonstrated that positional regularity in the temporal and spatial format can both be learned when the visual stimuli only presented one types of such regularity. However, information in the real world usually presents different kinds of regularity simultaneously. To determine whether people would have preferential performance in learning temporal and spatial regularity when both types of information is available, we developed novel positional statistical learning tests, which can be employed to examine the underlying neural mechanisms in the future. Specifically, each trial in the PSL test contained two consecutive frames, each of which contained two spatial positions on the left and right side of the fixation. One of 16 geometric shape that were not easily namable was assigned to occupy the left or right position in either the first or the second frame. For shapes that presented temporal regularity, it always appeared on the first or the second frame, with half of the times on the left position and the other half of the times on the right position. For shapes that presented spatial regularity, it always appeared on the left or right position, with half of the times in the first frame and the other half of the times in the second frame. After encountering independent and random presentation of temporally and spatially regular shapes across mixed trials, participants' learning performance was measured with multiple-choice questions. The results from a series of four experiments showed that 1) spatial positional regularity embedded in shapes was easier to detect than temporal positional regularity, and 2) adding non-informative monotones or unique environmental sounds to individual shape presentation did not improve the learning performance of temporal regularity, but 3) adding informative environmental sounds to indicate temporal and spatial trials (but not individual shapes) improved the learning performance of temporal regularity significantly. Overall, our findings revealed the learning preference for hidden spatial over temporal positional regularity, and the latter can become salient by combining with informative but not over-loading sounds. Future explorations with neuroimaging techniques would be pursued based on this optimized positional statistical learning paradigm..

投稿學會：台灣認知神經科學學會 | 編號: P1-52

Revisiting the effect of skin color on categorizing mixed-race faces spanning the Asian-White and the Asian-Black continuum: 2AFC and 3AFC tasks

Chien-Kai Chang1, Evelyn Hsin-Yi Tsai 4, Yi-Chiao Wu1, Yi-Chia Chen1, Yu-Tzu Hsu1, Pei-Tzu Liu3, Sarina Hui-Lin Chien1,2,*

1. Graduate Institute of Biomedical Sciences, China Medical University, Taiwan 2. Center for Neuroscience and Brain Diseases, China Medical University, Taiwan 3. Department of Biomedical Imaging and Radiological Science, China Medical University, Taichung, Taiwan 4. Graduate Program of Cognitive Sciences in Education, Columbia University, NYC

Abstract

Categorizing racially ambiguous faces has become an emerging research topic; however, most of this work has focused on biracial Black/White stimuli with Caucasian participants, and the influence of skin color is not fully understood. Here we investigated how skin color may affect the accuracy of categorizing faces spanning the Asian-White and the Asian-Black categorical continuum in Taiwanese adults. We conducted two behavioral experiments measuring participants' responses in a traditional 2AFC (2-alternative-forced-choice: own-race, other-race) and a novel 3AFC (3-alternative-forced-choice: own-race, other-race, biracial) paradigm to build the psychophysical profile of race categorization with grayscale and color face images. Forty Taiwanese adults (between 19 and 42, M = 23.27 years, 20 females) joined the study; each received a 2AFC and a 3AFC racial categorization task in two separate sessions. Each session contained four blocks (Asian-White grayscale, Asian-White color, Asian-Black grayscale, and Asian-Black color), allowing for comparison between the contribution of physiognomy and skin color within each participant. For the Asian-White morphing faces, our results show that the Taiwanese adults tend to classify the 50% morphed faces (i.e., the proportions of Asian faces and Caucasian faces are equal to half-and-half) as white, and not until 60% in the 2AFC session, and not until 70% in the 3AFC session, are morphed faces being classified as Asian, consistent with our previous findings. For the Asian-Black morphing faces, the general trend of categorization is similar to that of the Asian-White continuum; however, participants responded more accurately that the 50% morphed faces were consistently judged as biracial in the 3AFC task. Overall, the results suggest that people rely on both physiognomy and skin color to distinguish subtle differences in biracial faces. Skin color enhances perceptual richness that helps people correctly attribute morphed faces to the corresponding side of the racial continuum and correctly classify faces as biracial when faces are evenly morphed. In conclusion, this study provides cross-cultural evidence of the role of skin color may play in categorizing biracial faces in the under-represented Asian context.

投稿學會：台灣認知神經科學學會 | 編號: P1-53

Over- and underreactions to regime shifts and their neurocomputational substrates

Mu-Chen Wang1, George Wu2, Shih-Wei Wu1

1 National Yang-Ming University, 2 University of Chicago

Abstract

In dynamic environments where technology, markets, competitors and even narratives change regularly, many decisions are tightly associated with our ability to detect changes in the environment in a timely fashion. Previous studies on judgment and decision making found that people show systematic biases in response to regime shift—possible changes in the environments. In particular, humans tend to overreact to imprecise signals in stable environments, but underreact to precise signals in unstable environments. Methods. The goal of this study was to investigate the neurocomputational substrates for under- and overreactions to regime shifts. In a probability estimation task, subjects had to estimate the probability of change from one regime (the red regime) to the other (the blue regime) based on the signals they received. The signals were generated from one of the regimes, which always started from the red regime but can shift to the blue regime based on some transition probability. We investigated the impact of transition probability and signal diagnosticity—the relative ratio of red to blue balls in a regime—on the ability to detect regime shifts. Results. We replicated the systematic biases shown in previous studies: compared with the ideal Bayesian model, the subjects (n=30) tended to overreact to imprecise signals (low signal diagnosticity) in stable environments (small transition probability) by giving larger probability estimates of regime-shift. By contrast, subjects tended to underreact to precise signals (large signal diagnosticity) in unstable environments (larger transition probability) compared with the ideal Bayesian. Further, we fit a quasi-Bayesian model that incorporate free parameters to separately estimate sensitivity to transition probability and signal diagnosticity under different environmental conditions. We found that sensitivity to both transition probability and signal diagnosticity are a decreasing function of their respective dimensions, consistent with a system-neglect model in which people respond primarily to the signal and secondarily to the system that generates the signal. The fMRI results showed that the ventromedial prefrontal cortex (vmPFC) represented the probability estimates of regime shift. In addition, activity in the frontoparietal control network that included the dorsomedial prefrontal cortex (dmPFC), the dorsolateral prefrontal cortex, and the posterior parietal cortex represented the diagnosticity-dependent evaluation of sensory signals critical to estimating regime shift. Finally, neural model comparison revealed that the system-neglect model better described patterns of activity in the dmPFC in response to signal diagnosticity than the Bayesian model. Together, these results indicated that under- and overreactions to regime shifts arise from the fronto-parietal control networks involved in the evaluation of sensory signals in light of system parameters, before they are being used to compute probability estimates in the vmPFC.

投稿學會：台灣認知神經科學學會 | 編號: P1-54

Monetary gains and losses are represented by high-frequency oscillatory activity in the human brain

Siao-Jhen Wu Shih-Wei Wu

Institute of Neuroscience, National Yang-Ming Chiao Tung University Brain Research Center, National Yang-Ming Chiao Tung University

Abstract

Many decisions we face involve choosing between options that carry potential gains and losses. Decades of research from psychology show that people are loss averse — that “losses loom larger than gains”. Human fMRI studies showed that many brain regions, including the ventromedial prefrontal cortex (vmPFC), orbitofrontal cortex (OFC) and ventral striatum, represent information about monetary gains and losses during decision making. It remains controversial, however, whether these regions simultaneously represent gains (positive) and losses (negative). In this study, we attempted to address this issue using human stereo electroencephalography (sEEG). In a mixed-gamble task, human subjects (n=21) on each trial faced a 50/50 lottery of a potential monetary gain or loss and had to decide whether to play the lottery. As part of treatment plan attempting to identify epileptogenic zone, multi-contact depth electrodes were implanted in different brain regions including the OFC, dorsal-to-mid cingulate cortex, amygdala and insula. These four brain regions, with a total of 330 electrode contacts across subjects, were the focus of this study. Behaviorally, we replicated loss aversion in the patient population. Lambda, the ratio of sensitivity to changes in losses to gains inferred from choice behavior was around 2.3, suggesting that subjects were loss averse. Neurally, we found evidence for gain and loss representations in high-gamma activity (80-150 Hz). However, most contacts represented either gains or losses; very few contacts represented both gain and loss information. Together, these results suggest that gains and losses are more likely to be represented by different populations of neurons in these regions rather than by the same population of neurons.

投稿學會：台灣基礎神經科學學會	編號: P1-55

The Role of Self Emotional Processing and Empathic Responses in Gluckschmerz and Schadenfreude

Min-Min Lin, Ming-Tsung Tseng

Graduate Institute of Brain and Mind Sciences, National Taiwan University College of Medicine, Taipei, Taiwan

Abstract

Empathy, the capacity to share sensory or emotional feelings of others with self-other distinction, plays a crucial role in human emotional experience, social interaction and survival. However, in certain situations, people fail to empathize with others properly. They may experience displeasure from others' good fortunes (i.e., gluckschmerz) or pleasure from others' misfortunes (i.e., schadenfreude). Gluckschmerz and schadenfreude are socially undesirable emotions and have been termed counter-empathy, antipathy, or apathy. Although many previous studies have reported the neural substrates of empathy, little attention has been paid to the neural basis underlying gluckschmerz and schadenfreude. The relationships among self emotional processing, empathic responses and gluckschmerz, and schadenfreude also remain largely unclear. In a behavioral pilot study with competitive contexts, we devised a pain-related social emotional paradigm to assess gluckschmerz and schadenfreude resulting from others' relatively rewarding and punishing pain experience, respectively. Our preliminary results show that gluckschmerz and schadenfreude respectively exhibited an inverse relationship with the change in positive and negative empathic responses, which appears to suggest that both gluckschmerz and schadenfreude are counter-empathy. In addition, compared to self emotional processing, participants' empathic responses had a larger influence on their gluckschmerz and schadenfreude. In the near future, we plan to use functional magnetic resonance imaging (fMRI) with this paradigm to examine the neural mechanisms underlying the associations among self emotional processing, empathic responses and gluckschmerz, and schadenfreude. Results obtained from this study will enhance our knowledge about the nature of socially undesirable emotions and cerebral mechanisms underlying emotional processing in social contexts.