Nanosymposium session III

Neurofilament light chain: emerging evidence in neuropsychiatric disorders

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Abstract

Neurofilament light chain (NFL) is a protein located mainly in the axon region of myelinated brain neuron. This small protein has been studied intensively for its characteristic of sending brain neuronal damage messages toward the peripheral blood. It makes diagnoses of brain injuries possible through taking peripheral blood rather than cerebral spinal fluid. Using single molecule array method for NFL concentration measurement as an in vitro diagnostic device for neurodegenerative diseases, such as dementia, amyotrophic lateral sclerosis, multiple sclerosis, and Parkinson’s disease, has already been approved in Europe. Recent studies have also shown that peripheral blood NFL level may indicate the severity of psychiatric diseases, including schizophrenia, major depressive disorder, and substance use. In this report session, the researchers will report the discovery of NFL in psychiatric diseases and its role in the pathological symptom. I will summarize recent literature reports on NFL that reveal its potential applications for both basic and clinical research.

Selected recent publications:


Exploring the role of neurofilament light chain in addiction

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M.D., Ph.D.,

Abstract

Substance use disorders have been associated with persistent neurocognitive impairment and structural brain abnormalities. Because brain imaging facilities are not universally available for healthcare systems, it may be feasible to look for a reliable and adequate substitute indicator for clinicians to evaluate and monitor the neurotoxicity severity. We examined the level of NFL in patients with ketamine dependence (KD) and alcohol dependence (AD) the explored its relationship with clinical characteristics. We found significantly increased NFL levels following chronic and heavy ketamine or alcohol use. In patients with KD, we found pronounced increase of NFL levels in the those comorbid with MDD. The observation warrants further investigation of a potential neuroaxonal vulnerability of depressed patients to prolonged ketamine exposure. In addition, NFL level might be a novel indicator for AD and reflect clinical severity in craving and psychological symptoms (anxiety and depression). Also, the aldehyde dehydrogenase gene polymorphism rs671 seemed to influence the level of NFL. These observations collectively indicate NFL levels may serve as a potential indicator for neurotoxicity related to addictive disorders.

Selected recent publications:


Nanosymposium session III

Neurofilament Light Chain Is a Novel Biomarker for Major Depression and Related Executive Dysfunction

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M.D., Ph.D., National Yang Ming Chiao Tung University.

Abstract

Background: Evidence suggests that major depressive disorder is related to neuroaxonal injury and that neurofilament light chain (NfL) is a biomarker of neuroaxonal injury. In addition, proinflammatory cytokines have been reported to be associated with major depression and neuroaxonal injury. Methods: Forty patients with major depression and 40 age- and sex-matched healthy control subjects were enrolled for the measurement of NfL and proinflammatory cytokines and assessment of executive function. General linear models were used to examine the association between NfL levels, proinflammatory cytokine levels, and executive function. Results: Patients with major depressive disorder exhibited significantly higher NfL levels (p = 0.007) than the control subjects. NfL levels were positively related to log-transformed levels of tumor necrosis factor (TNF)-α (p = 0.004). Higher levels of NfL (p = 0.002) and TNF-α (p = 0.013) were associated with greater deficits in executive function. Discussion: NfL was a novel biomarker for major depressive disorder and related executive dysfunction. Further studies are necessary to elucidate the role of NfL in the pathophysiology of major depression and related cognitive impairment.

Selected recent publications:


The Interplay of Stress, Inflammation, and Nutrition in Depression

Kuan-Pin Su

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M.D., Kaohsiung Medical College, Kaohsiung, Taiwan

Ph.D. Institute of Psychiatry, King’s College London, UK

Abstract

The increasing global burden calls for the development of novel approaches to tackle unmet needs in prevention and treatment of depression underlying biological, psychological and social dysregulations. Depressed patients with chronic low-grade inflammation might be classified as a subgroup of major depressive disorder (MDD); therefore, looking for antidepressant therapies from anti-inflammatory pathways could improve treatment effectiveness for this subgroup of patients. Omega-3 (or n-3) polyunsaturated fatty acids (PUFAs) are anti-inflammatory both in peripheral organs and central nervous systems and have clinically applied in the treatment and prevention of depression, cardiovascular diseases, dyslipidaemia, diabetes and arthritis. Anthropological studies suggest that human beings evolved to a modern diet with less than one-tenth of omega-3 to omega-6 PUFAs intake ratio, which leads to a constitutional bias toward chronic systemic inflammatory status to explain dramatically increasing of depression and chronic medical illnesses in modern world. The presentation is to provide our recent clinical and pre-clinical studies and an overview about the role of inflammation in “mind-body” comorbidity and present anti-inflammatory mechanisms by which n-3 PUFAs may orchestrate the molecular and cellular functions and facilitate the therapeutic pathways in chronic medical illnesses and depression.

Selected recent publications:


Loss of Function of Shaker type Potassium Channels caused Epileptic Encephalopathy

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Abstract

Potassium (K+) channels are essential for the regulation of neuronal membrane potential and electrophysiological excitability. The Shaker-type potassium channels (Kv1) family contains 8 members including Kv1.1-Kv1.8. Six were prominently expressed in the CNS (Kv1.1-6). Hitherto, two of the Kv1 genes (KCNA1 and KCNA2) have been reported to cause human epilepsies. In this study, we identified three patients with de novo missense pathogenic variants in three additional Shaker-type channel genes (KCNA3, KCNA4, and KCNA6). All variants were located in important functional domains such as the selectivity filter or the S6 hinge responsible for gating. Clinically, they presented as early-onset epileptic encephalopathy which subsided after the infantile period. Electrophysiological studies demonstrated loss of function effects in two of the variants, which could impair repolarization of the action potentials causing hyperexcitable neuronal activities. The disease course coincided with the age-related expression of these genes. Our findings expand the list of potassium channels genes, especially the Shaker-type, that cause human epilepsies.

Selected recent publications:


Epileptogenesis In Cerebral Cavernous Malformations: Genomic Landscape, Biomarkers And Neuroplasticity

Yo-Tsen Liu(劉祐岑)MD, PhD
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Division of Epilepsy, Neurological Institute, Taipei Veterans General Hospital, Taipei, Taiwan.
國立陽明交通大學醫學系兼任副教授
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Abstract

Cerebral cavernous malformation (CCM), characterized by enlarged, blood-filled endothelial compartments within the venous-capillary vascular bed, is the most frequent epileptic substrate of cerebral vascular malformations. Epilepsy is the leading clinical manifestation of CCM, observed in 50% of patients. However, the clinical behavior of a CCM lesion could be highly variable and it is quite challenging to predict the epilepsy risk and outcome of different lesions. The uncertainty thus leads to the consensus of ideal treatment for CCM remains inconclusive. Familial CCM and some sporadic cases caused by loss-of-function mutations in one of the three genes, CCM1, CCM2 and CCM3, with the hallmark of multiple lesions, have significant higher risk of CCM-related epilepsy (CRE). Recently, the substantial contribution of somatic mutations to the epileptogenic mechanism of CCM has been recognized. Understanding the genomic landscape is essential to decipher the mechanisms of the development and progression of CRE. It is also important to search biomarkers from the transcriptome, proteome and connectome of CCM which can correlate with the patient’s seizure outcome and other clinical relevant phenotypes. Further, the key molecules shaping the CCM microenvironments and the whole picture of the crosstalk between abnormal vasculogenesis and altered neuronal plasticity could be unraveled. The goal is to improve precision treatment of CCM.

Selected recent publications:


Deciphering age-dependent neuronal hyperexcitability caused by CDKL5 deficiency

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Professor, Institute of Neuroscience, National Cheng-Chi University
Ph.D. National Yang-Ming University

Abstract

Cyclin-dependent kinase-like 5 (CDKL5) is a serine-threonine kinase, localized primarily in the nucleus to control gene expression and neuronal development by regulating protein phosphorylation. Mutations in CDKL5 gene cause CDKL5 deficiency disorder (CDD), a developmental and epileptic encephalopathy characterized by severe early-onset seizures, developmental delay and intellectual disability. Despite well-defined genetic cause, children with CDD are resistant to most of the anti-epileptic drugs and the pathogenic mechanisms for early-onset seizures in CDD remain unclear.

Through longitudinal EEG recordings coupled with videotaping in preweaning mouse pups, we found that Cdkl5 null pups displayed increased EEG discharges at postnatal day 12 (P12) compared to their wild-type (WT) littermate controls. The increased burst discharges in mutants returned to the comparable pattern of WT mice after P14, suggesting that CDKL5 deficiency may increase neuronal excitability in an age-dependent manner. Through electrophysiological study and whole-genome RNA sequencing, we further revealed that CDKL5 ablation increases firing rate of neurons and down-regulates genes for tangential migration in dorsal hippocampus at P7. By contrast, the transcript levels of marker genes for GABAergic interneurons, such as parvalbumin and somatostatin, were up-regulated in cortical tissue of mutants at the age of P17 and 3-month, consistent to the increased number of parvalbumin-positive neurons found in the primary motor cortex of adult Cdkl5 null mice. Our results demonstrate that CDKL5 is required to preserve gene expression essential for interneuronal migration during early postnatal age. Loss of CDKL5 may affect tangential migration of interneurons that leads to hyperexcitability soon after birth. Our discoveries may provide a basis to develop therapeutics for early-onset seizures in developmental disorders, including CDD.

Selected recent publications:

Cellular and molecular mechanisms for malformations in cortical development

Jin-Wu Tsai

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Dean of Research and Development, NYCU, Taiwan

Adjunct Professor, Department of Biological Science & Technology, NYCU, Taiwan

Ph.D., Columbia University

Abstract

Neural developmental disorders are divesting neurological diseases resulting from defects in neural progenitor proliferation, neuronal migration and connections during development. These defects may lead brain malformations, including microcephaly, lissencephaly, double cortex, and focal cortical dysplasia. The patients often suffer from epilepsy, developmental delay, and cognitive impairments. To date, the genetic causes of a number of brain malformations have been identified, such as LIS1, DCX, ARX, TUBA1A, NDE1, KATNB1, and CDK5. Using in utero electroporation to knock down these genes and monitoring cellular and subcellular events with live cell imaging in brain slices, we found that LIS1 together with dynein facilitates centrosomal and nuclear movements during neuronal migration. Using whole exome sequencing (WES) in a cohort of patients with cortical malformation, we further identified variants in novel genes, CEP85L and BICD2, that cause lissencephaly. However, many genetic mutations involved in cortical malformations still remain unidentified. Recently, we developed an in vivo genetic screen paradigm that utilizes in utero electroporation of transposons into mouse embryos to induce insertional mutations in neural stem cells (i.e., radial glial cells; RGCs). We identified 33 potential genes, many of which have been previously implicated in neuronal development and related disorders, including holoprosencephaly, microcephaly and mental retardation. Bioinformatics analysis demonstrated that these candidate genes are highly associated with neuronal development and various neuronal disorders. In this presentation, molecular mechanisms of these genes in cortical malformation will be discussed.

Selected recent publications:


Abstract

In the past decades, exosomes derived from mesenchymal stem cells have been used as therapeutics in various fields. Recently, we first demonstrated the antinociceptive effects of human umbilical cord mesenchymal stem cell (hUCMSC) exosomes, given intrathecally, in the L5/6 spinal nerve ligation neuropathic pain model. The therapeutic effects were associated with anti-inflammatory and neurotrophic effects of exosomes. We then further tested locally applied exosomes, embedded in alginate scaffold, in the pain model and found similar analgesic and neurotrophic effects. To further extent the application of exosomes, we examined the use of exosomes, embedded in gelfoam, in T9 spinal cord hemisection injury model. Our recent work demonstrated the nerve regenerative and motor function-improving potential of hUCMSC exosomes in the spinal cord injury model.

Selected recent publications:


Nutrition intervention for neuropathic, muscular and acute pain

Jen-Yin, Chen (陳貞吟)
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Ph.D. National Chung Hsing University

Abstract

Vitamin D and vitamin C are essential micronutrients for human health. In cell and animal models, vitamin D has concentration-dependent anti-inflammatory effects through inhibiting the production of nitric oxide. Nitric oxide increases phosphorylated N-methyl-D-aspartate receptors in spinal dorsal horn neurons leading to central sensitization and mechanical allodynia. Vitamin D deficiency induces excessive reactive oxygen species resulting in cold pain, produces a marked dysbiosis and alters nociception via molecular mechanisms involving the endocannabinoid and related mediator signaling system. Clinically, we discovered that patients with neuropathic pain had a high prevalence of hypovitaminosis D which was associated with increased spontaneous cold pain and brush-evoked pain based on the DN4 questionnaire. Vitamin C exerts antinociception and neuromodulation. Spontaneous pain symptoms include tingling, prickling, pins and needles sensation, as well as electric shock, bursting, jumping, shooting, stabbing and burning pain. We discovered that the patients' plasma vitamin C concentrations were negatively correlated with spontaneous pain and with tingling, prickling or pins and needles sensation by the LANSS Questionnaire. Intravenous high-dose vitamin C relieved spontaneous pain effectively but not brush-evoked pain in patients with neuropathic pain. We will describe the findings in patients with muscular pain as well. A pneumatic tourniquet involving a previous exsanguination is often used in total knee arthroplasty. The procedures induce muscle ischemia leading to tissue acidosis and aching sensation (soreness, tingception). Tingception depends on the proton-sensing neurons and is a specific somatosensory function that transmits the aching sensation from the peripheral to the central nervous system when the tissue acidosis. High-dose vitamin C pretreatment reduces exercise-induced muscle soreness, oxidative stress and with little loss on muscle function. The benefits of intravenous high-dose vitamin C in patients with total knee arthroplasty will be reported.

Selected recent publications:


New advances in myofascial pain syndrome

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Director, Department of Physical Medicine and Rehabilitation, Asia University Hospital, Asia University, Taiwan.

M.D., Ph.D. China Medical University

Abstract
Myofascial pain syndrome, characterized by the presence of myofascial trigger points (TrPs), is recognized as a common source of musculoskeletal pain. Myofascial TrP is a hyperirritable spot within a taut band of skeletal muscle that is painful on compression, stretch, overload, or contraction of the tissue which usually responds with a referred pain that is perceived distant from the spot. Several needling therapies are proposed for treatment of myofascial pain syndrome. In fact, two different needling applications can be used to inactivate TrP: wet needling (injections) or dry needling. Fu’s subcutaneous needling (FSN) is one of the newly invented dry needling methods. The treated target is tightened muscle (the muscle including TrPs). The soft tissue pain diseases might be treated via FSN combined the special techniques (swaying movement and reperfusion approach).

The application of TrP needling therapy is able to reduce this excitability by reducing peripheral nociception from the TrP, by reducing dorsal horn neuron activity, and by modulating brainstem areas. However, effects are only seen at short-term and effect sizes are small, pointing to a particular role that TrPs play within the complex chronic pain experience. When providing needling therapy to chronic pain patients, therapists are advised to integrate contemporary pain neurosciences when providing the treatment. This implies applying evidence-based recommendations; therefore, TrP needling therapy can be part of, but should never be, the only treatment plan for individuals with chronic pain. A comprehensive management program should include neuroscience pain education, exercise programs, self-management (including graded activity), stress management (psychological), sleep management, and other individually tailored aspects of self-management.

Selected recent publications:
Mechanism of prolotherapy and ultrasound in chronic muscle pain

Der-Sheng Han

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Director, Taiwan Academy of Physical Medicine and Rehabilitation
Director, Taiwan Osteoporosis Association
Director, Taiwan Society of Neurorehabilitation
Education Committee, ISPRM
PhD.Graduate Institute of Clinical Medicine, National Taiwan University, Taiwan.

Abstract

Prolotherapy is widely used in pain control and tissue repair in pain medicine. The classical mode is injection with hypertonic dextrose in muscle or perimysium. However, the analgesic mechanism is still not known. Here we successfully established dextrose-mediated analgesia in a mouse model of fibromyalgia. The analgesic effects of dextrose injections were evaluated in a mouse model of fibromyalgia, in which bilateral chronic mechanical hyperalgesia was induced by unilateral intramuscular acid injection. The injectant (dextrose), dose (≥ 5%) and volume (>10 uL) but not osmolarity were essential for the prolotherapy. Further studies showed that activation of acid-sensing ion channel 1a (ASIC1a), neural activation, and the release of substance P from muscle afferents were required in the dextrose-induced analgesia. Both pharmacological blockade and genetic deletion of ASIC1a or substance P as well as lidocaine abolished the dextrose-induced analgesia in mice with chronic hyperalgesia. Moreover, intramuscular dextrose injection induced phosphorylated extracellular signal-regulated kinase (pERK) expression in dorsal root ganglia neurons expressing substance P; the pERK expression was inhibited by the ASIC1a antagonist PcTx1. The optimal settings for prolotherapy in fibromyalgia-like pain are dextrose- and volume-dependent, and the peripheral analgesia involves ASIC1a and substance P signaling in muscle afferents. We suggest a possible mechanism of action of dextrose prolotherapy in noninflammatory muscle pain such as fibromyalgia and provides insights for treating other types of chronic pain.

Selected recent publications:


Inflammation and Nutrition in Child Mental Health: Focus on ADHD

Jane Pei-Chen Chang
Director, Child Psychiatry Division, Department of Psychiatry, China Medical University Hospital, Taichung, Taiwan
Assistant Professor, College Medicine, China Medical University, Taichung, Taiwan

M.D., China Medical University, Taichung, Taiwan
M.Sc., China Medical University, Taichung, Taiwan
Ph.D., Department of Psychological Medicine, Institute of Psychiatry, Psychology and Neuroscience (IoPPN), King’s College London, UK

Abstract

Inflammation and deficiency of omega-3 polyunsaturated fatty acids (n-3 PUFAs), such as docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA), have been suggested to play a role in attention deficit hyperactivity disorder (ADHD). Studies have suggested that children with ADHD tend to have higher inflammatory biomarkers and less intake and lower blood levels of n-3 PUFAs. This talk will provide a brief overview focused on personalized medicine of n-3 PUFAs in ADHD with the most updated research findings supported by cross-sectional studies, meta-analyses and randomised controlled trials.

Selected recent publications:


Nanosymposium session III

Effects of Anthocyanin-rich Mulberry Milk on Working Memory and Mental Wellbeing in Healthy Working Population

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Research Institute for High Human Performance and Health Promotion, Khon Kaen University, Khon Kaen, Thailand

MD. Khon Kaen University

Abstract

Anthocyanins from various types of berries have been investigated for positive effects on cognitive, mood, anxiety, and stress symptoms. Our group conducted an open-labelled study in 300 healthy volunteers to consume either 1 or 2 servings of the anthocyanin-rich mulberry milk daily for 6 weeks. We monitored performances on 7 computerized working memory tests, General Health Questionnaire-28 (GHQ-28), Hospital Anxiety and Depression Scale (HADS), saliva activity levels of acetylcholinesterase (AchE), monoamine oxidase (MAO), and cortisol at the baseline and after 6 weeks. We found significant improvement of performances on all working memory tasks, significantly decreased GHQ-28 and HADS total scores and all their subscales, and significantly decreased activity levels of AchE, MAO-A, MAO-B, and cortisol in both groups (all p < 0.05). We speculated that a daily consumption of anthocyanin-rich mulberry milk improved psychological and somatic symptoms via suppressions of AchE, MAO and cortisol activity.

Selected recent publications:


Brain Ageing: Potential Avenue for Antioxidant Compounds?

Hanafi Ahmad Damanhuri
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Ph.D., Macquarie University, Australia

Abstract

Brain aging is a continuous and complex multifactorial process manifested by physiological and cognitive deterioration, ultimately leading to death. Brain aging influences mental health and affects individuals' ability to carry out their daily routines. For the past decades, research has been conducted to understand how the ageing process affects the brain, from the molecule to the functional perspective. Despite the aggressive effort, many more questions are being raised and fail to clearly explain how the actual process occurs during brain ageing. It was further supported by the fact that the numbers of drugs or natural products extract results in inconclusive findings in delaying brain ageing progression in various contexts. Several questions remain unsolved and require extensive investigation. The controversies and relevant issues will be discussed further in the presentation.

Selected recent publications:


Inflammatory Cytokines in and Cognitive Function of Adolescents with First-Episode Schizophrenia, Bipolar Disorder, or Major Depressive Disorder

Mu-Hong Chen
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Department of Psychiatry, College of Medicine, National Yang Ming Chiao Tung University, Taipei, Taiwan
M.D., Ph.D., National Yang Ming Chiao Tung University.

Abstract

Background: Few studies have explored the complex relationship of pro- and anti-inflammatory cytokines with cognitive function in adolescents with first-episode schizophrenia, bipolar disorder, or major depressive disorder. Methods: In total, 26, 35, and 29 adolescents with first-episode schizophrenia, bipolar disorder, and major depressive disorder, respectively, and 22 age- and sex-matched controls were included in the current study. Cytokines, namely interleukin (IL)-2, IL-6, tumor necrosis factor (TNF)-α, and C-reactive protein (CRP), were assessed. The Wisconsin Card Sorting Test (WCST) and the working memory task were administered to assess cognitive function. Results: Using generalized linear models with adjustment for demographic data and clinical symptoms, patients with bipolar disorder were found to exhibit the highest levels of CRP (p = 0.023), IL-6 (p = 0.022), and TNF-α (p = 0.011) and had the lowest IL-2 levels (p = 0.034) among the four groups. According to the results of the WCST and working memory task, adolescents with schizophrenia exhibited the lowest performance in cognitive function. In addition, among the assessed cytokines, only CRP levels (p = 0.027) were negatively associated with WCST scores. Discussion: Dysregulated pro- and anti-inflammatory cytokines and impaired cognitive functioning were observed in first-episode adolescent-onset schizophrenia, bipolar disorder, and major depressive disorder. The altered cytokine profiles may play important roles in the pathophysiology of schizophrenia, bipolar disorder, and major depressive disorder.

Selected recent publications:


HF–Age–Gender (HAG) Index as a Biomarker for Sleep Disorder in mTBI

John Chung-Che Wu

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Division of Neurosurgery, Department of Surgery, School of Medicine, College of Medicine, Taipei Medical University

M.D., Ph.D., Taipei Medical University

Abstract

Biomarkers are emerging as a useful tool for prediction of outcomes in TBI. To identify a screening tool for poor self-reported sleep quality in the first week after mild traumatic brain injury (mTBI) for sleep disorder at 12 weeks, data from 473 mTBI participants were collected and follow-ups were performed at 12 weeks. Patients were then divided into two groups according to the Pittsburgh Sleep Quality Index based on whether or not they experienced poor sleep quality at 12 weeks post-mTBI. The analysis was performed on personal profiles and heart rate variability (HRV) in the 1st week. An analysis of the non-invasive patient data for mTBI patients who did and did not complain of poor sleep quality revealed several factors relevant to the delayed onset of poor sleep quality, including age, gender, and HRV measurements. The HRV–age–gender (HAG) index has a 100% sensitivity (cut-off, 7; specificity, 0.537) to predicting whether the patient will experience poor sleep quality after mTBI at the 12-week follow-up. The HAG index allows identifying the patients with mTBI who have no initial sleep quality complaints but are prone to developing poor self-reported sleep quality at 12 weeks.

Selected recent publications:


Abstract

Visual working memory (VWM) is a cognitive faculty that allows us to remember visual information from one fixation to another. However, VWM capacity is far from perfect, and research has demonstrated a high degree of individual difference in VWM performance. Perhaps due to its imperfection and fragility, VWM capacity has been shown to be highly sensitive to one’s neurological development and frontoparietal functioning, and can be a sensitive marker to many factors such as aging, and neurodegenerative diseases, etc. In this talk I will explore the possibility of applying VWM to research in traumatic brain injury, as well as some of the brain stimulation techniques such as transcranial electric stimulation and sensory stimulation that have been known to facilitate VWM performance, that may be of interest to clinicians working with TBI patients.

Selected recent publications:

Task-related fMRI reveals age-related neuro-functional differences in younger and older adult decision processes

Joshua Goh (吳恩賜)
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Ph.D. University of Illinois, Urbana-Champaign

Abstract
The ability of the human brain to make appropriate decisions given the context undergoes notable changes with age. This is a puzzling phenomenon since both younger and older adults are faced with the same contextual scenarios, yet differences in decision behaviors reflect age-related influences in the underlying neural processes that culminate in the chosen actions or responses. In a series of studies, our lab has applied the use of psychological value-based decision-making in lottery choices and complex rule contingency decision-making in functional magnetic resonance imaging experiments applied on younger and older adults. Our findings highlight drastic differences in neural network systems engaged by younger and older adults as they process value-based decisions as well as non-linear rule-mappings. Crucially, in these fMRI tasks, we found a shift from rapid, simplifying heuristic-like processing in younger adult striatum and prefrontal operations to slower, more biased processing in older adult medial and lateral frontal systems. These findings delineate how the human brain reorganizes its decision-making neural circuits in the face of neurobiological changes and lifespan experiences with age.

Selected recent publications:
Nanosymposium session III

Sleep disturbance and its impacts on cognitive deficits following traumatic brain injury

Hsiao-Yean(Shannon) Chiu(邱曉彥)
Associate Professor and Deputy director of School of Nursing,
College of Nursing, Taipei Medical University
Supervisor, Department of Nursing, Taipei Medical University Hospital
RN, Ph.D.Taipei Medical University

Abstract
Traumatic brain injury (TBI) is one of the most common neurological disorder, which can be classified as mild, moderate or severe types, and may have wide-ranged physical and psychological sign and symptoms. On the other hand, with increasing life expectancy in modern societies, the epidemiological pattern of TBI has changed in recent decades, with an increasing proportion of TBI survivors falling into the older age group (one-third of TBI survivors). In general, TBI has become a major public health concern for young and older adults. Note that some symptoms could appear immediately after brain trauma, while others days or weeks later. Among these symptoms, sleep disturbance is one of the most common complaint in the population. In the lecture, I will review our past study findings regarding the sleep patterns of TBI survivors at acute and chronic stages. During acute stage, sleep duration seems play a critical role in recovering cognitive impairment caused by head trauma; in the chronic stages, psychological factors such as depression may contribute to the development of sleep disturbance following TBI. Furthermore, I will describe the association between post-TBI sleep disturbance and cognitive impairment and illustrate possible nonpharmacological interventions targeting sleep disturbance following TBI.

Selected recent publications: