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SCHOOL OF BIOMEDICAL SCIENCES
THE UNIVERSITY OF HONG KONG
香港大學生物醫學學院

he 4th International Anatomical ciences and Cell Biology Conference

h – 6th December, 2016 (Sun – Tue) | Hong Kong

CONFERENCE VENUE

Cheung Kung Hai Conference Centre
i Ka Shing Faculty of Medicine
The University of Hong Kong

This conference is part of the 130th Anniversary Frontiers Conference Series in celebration of 130 Years of Medicine in Hong Kong.

130 YEARS OF MEDICINE IN HONG KONG
WISDOM · COMPASSION · COMMITMENT
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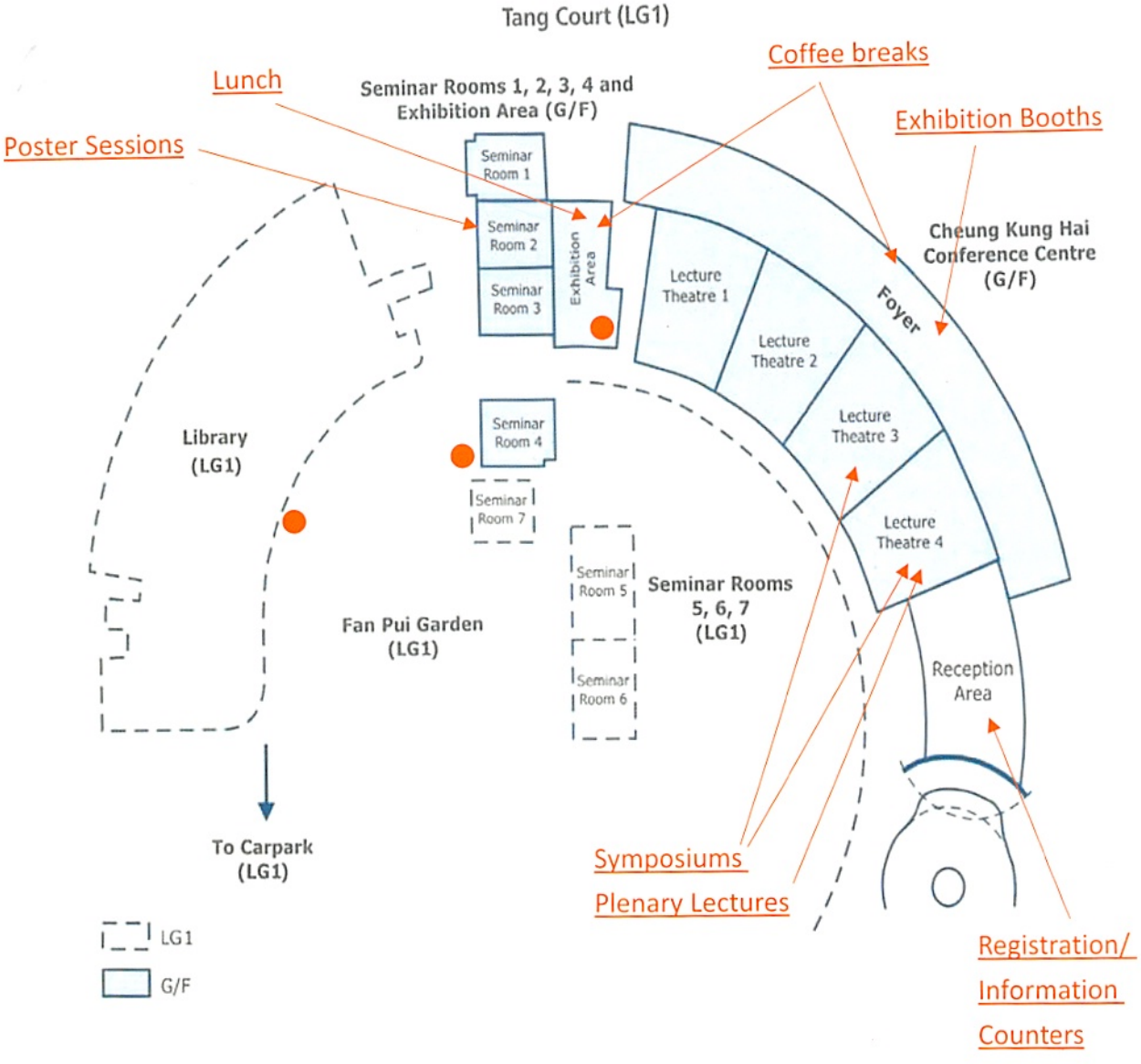
Conference Venue

Cheung Kung Hai Conference Centre

Li Ka Shing Faculty of Medicine

The University of Hong Kong

William MW Mong Block, 21 Sassoon Road, Pokfulam, Hong Kong



To be environmental friendly, please bring your reusable own bottle to the Conference. Water dispensers are available inside the Exhibition Area and the following locations in our campus:

- Near the Library, LG1
- Near Seminar Room 4 in the Lift lobby, G/F

Schedule-at-a-Glance

Super-resolution & Intra-vital Imaging Workshop
December 4, 2016 (Sunday)

08:30 – 17:00	Registration	
Time	**Programme**	**Venue**
09:30 – 11:00	L1 – L3	Lecture Theatre 3
11:00 – 11:30	Coffee Break	Exhibition Area
11:30 – 12:30	L4 – L5	Lecture Theatre 3
13:00 – 14:00	Lunch	Exhibition Area
14:00 – 18:00	Demonstration Session 1 - 4	FCF

Conference – Day 1
December 5, 2016 (Monday)

Time	Programme	
08:30 – 17:30	**Registration**	
	Lecture Theatre 4	
09:00 – 09:20	**Opening Ceremony** Guest of Honor: **Professor LEUNG, Suet-yi** Associate Dean (Research), LKS Faculty of Medicine The University of Hong Kong, Hong Kong	
09: 20 – 10: 50	Lecture Theatre 4 Plenary Lecture 1	
10:50 – 11:15	Coffee Break / Poster Session	
	Lecture Theatre 3	Lecture Theatre 4
11:15 – 12:45	SYM 1.1 Advances in Neuroscience	SYM 1.2 Innovations in Teaching Histology
12:45 – 14:00	Lunch Break / Poster Session	
	Lecture Theatre 3	Lecture Theatre 4
14:00 – 15:30	SYM 2.1 Advances in Developmental Studies	SYM 2.2 Good Practices in Teaching Gross Anatomy
15:30 – 16:00	Coffee Break / Poster Session	
16:00 – 17:30	Lecture Theatre 4 Plenary Lecture 2	
18:30 – 21:00	**Banquet** (Full registration participants and invited guests) Golden Lilies Banquet, Cyberport, Pokfulam	

Conference – Day 2
December 6, 2016 (Tuesday)

Time	Programme	
08:30 – 14:00	**Registration**	
09:00 – 10:30	Lecture Theatre 4 Plenary Lecture 3	
10:30 – 11:00	Coffee Break / Poster Session	
	Lecture Theatre 3	Lecture Theatre 4
11:00 – 12:30	SYM 3.1 Advances in Cancer Research	SYM 3.2 Body Donation
12:30 – 13:45	Lunch Break / Poster Session	
	Lecture Theatre 3	Lecture Theatre 4
13:45 – 15:15	SYM 4.1 Advances in Aging Studies	SYM 4.2 Innovations in Teaching Gross Anatomy
15:15 – 15:45	Coffee Break / Poster Session	
15:45 – 17:15	Lecture Theatre 4 Plenary Lecture 4	
17:15 – 17:30	**Closing Ceremony / Poster Awards**	

Contents

Welcome Message from Chairman of Organising Committee

Dear Friends and Colleagues,

On behalf of the Organising Committee, I am honored and delighted to welcome all of you to the 4th International Anatomical Sciences and Cell Biology Conference (IASCBC). It is also our pleasure to be the Organiser for the conference this year. We aim to make this conference a successful one in providing a forum to share and discuss recent advances in Anatomy education and cell biology research. For this year's meeting, we have invited over 40 distinguished speakers, from all over the world, in various disciplines to share their experiences and achievements with us.

The advances in microscopy, cell and molecular biology have placed the anatomical science and cell biology at new horizons. Anatomical science is no longer a morphological discipline on structures. Recent development in live cell imaging enables us to examine the dynamic physiological and disease processes in live cell and tissues in real time.

We have included one-day workshop for advanced microscopy in this meeting which will cover super-resolution microscopy and intravital imaging. For the core meeting, we will have parallel sessions to cover both Anatomy education and basic scientific research in biomedical sciences. The Anatomy education sessions will cover innovative teaching methodology of anatomical sciences aiming to facilitate our students to explore and learn in an interactive environment. There will be a special session on body donation programme which is crucial for anatomical education. For the parallel scientific sessions, we will cover recent advances in neurosciences, developmental biology, ageing and cancer. We aim to provide an interactive forum for discussion in anatomical sciences and cell biology.

I would like to express my appreciation and gratitude to the International Advisory Committee for their tremendous input and brilliant suggestions to the programme; the Adjudication panel for their thorough and timely review of the posters; our co-organisers and sponsors for their efforts and support to the conference; and the Local Organising Committee members for their dedication and hard work to ensure the success of this meeting.

Finally, I hope you all find the conference informative and enjoyable. And for participants from overseas, I wish you a very pleasant stay in Hong Kong.

George Tsao Sai Wah
Chairman of Organising Committee
IASCBC 2016

International Advisory Committee

BAY, Boon Huat
National University of Singapore, Singapore

CHOMPOOPONG, Supin
Mahidol University, Thailand

CHONGTHAMMAKUN, Sukumal
Mahidol University, Thailand

DHEEN, S Thameem
National University of Singapore, Singapore

DRAKE, Richard
Cleveland Clinic Lerner College of Medicine, USA

LEE, Wang Jae
Seoul National University College of Medicine, Korea

HING, Hiang Lian
Universiti Kebangsaan, Malaysia

MAHAKKANUKRAUH, Pasuk
Chiang Mai University, Thailand

PAWLINA, Wojciech
Mayo Clinic College of Medicine, USA

TOPP, Kimberly
University of California, USA

TSENG, Guo-Fang
Tzu Chi University, Taiwan

ZHANG, Shao-Xiang
Third Military Medical University, China

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School of Biomedical Sciences

CHAN, Ying Shing
School of Biomedical Sciences

CHANG, Raymond Chuen Chung
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CHUNG, Sookja Kim
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YANG, Jian
School of Biomedical Sciences

YU, Cheng-Han
School of Biomedical Sciences

ZHOU, Zhongjun
School of Biomedical Sciences

Programme in details

Super-resolution & Intra-vital Imaging Workshop
International Anatomical Sciences & Cell Biology Conference 2016
December 4, 2016 (Sunday)

08:30 – 17:00	Registration	
Time	**Programme**	**Venue**
09:30 – 10:00	**L1: High resolution imaging of synaptic structure and molecules** **OKABE, Shigeo** *Chair Professor* *The University of Tokyo, Japan*	**Lecture Theatre 3**
10:00 – 10:30	**L2: 4D Light-Sheet Fluorescence Microscopy for Live Cell Imaging: LBS 1.0** **DU, Shengwang** *Associate Professor* *The Hong Kong University of Science and Technology, Hong Kong* *Chief Technical Advisor, Nanobioimaging Ltd*	**Lecture Theatre 3**
10:30 – 11:00	**L3: Mechano-transduction of integrin signaling and podosome formation** **YU, Chenghan** *Assistant Professor* *The University of Hong Kong, Hong Kong*	**Lecture Theatre 3**
11:00 – 11:30	**Coffee Break**	**Exhibition Area**
11:30 – 12:00	**L4: Advances in Super-Resolution and Lightsheet Microscopy for Live Cell and Deep Imaging** **KOCH, Daniel** *Senior Reginal Application Specialist* *Carl Zeiss Microscopy GmbH, Germany*	**Lecture Theatre 3**
12:00 – 12:30	**L5: Intravital imaging in the mouse brain** **LAI, Cora Sau Wan** *Assistant Professor* *The University of Hong Kong, Hong Kong*	**Lecture Theatre 3**
13:00 – 14:00	**Lunch**	**Exhibition Area**
14:00 – 15:00	**Demonstration session 1**	**FCF**
15:00 – 16:00	**Demonstration session 2**	**FCF**
16:00 – 17:00	**Demonstration session 3**	**FCF**
17:00 – 18:00	**Demonstration session 4**	**FCF**

Four equipment demonstrations will be run in parallel. 4 sessions will be scheuled for each equipment demostration. 5 registrants are expected in each session.

Equipment for demostration:

1. Zeiss Elyra S1 SIM super-resolution system
2. NBI STORM super-resolution system
3. Olympus FVMPE-RS hybrid multi-photon intra-vital imaging system
4. LBS Light-Sheet Microscope and Live Cell Imaging System

OKABE, Shigeo MD, PhD
Professor, Department of Cellular Neurobiology,
The University of Tokyo, Japan

I started my academic career as an assistant professor in Prof. Nobutaka Hirokawa's laboratory in the University of Tokyo, where I studied microtubule dynamics in neurons. As a postdoc in the laboratory of Prof. Ron McKay in NINDS/NIH, I switched my subject to neuronal stem cells. In 1996, I came back to Japan and became a Principal Investigator of National Institute of Bioscience and Human-Technology in Tsukuba and started my new research project on synapse dynamics. In 1999, I moved to Tokyo Medical and Dental University and then back to the University of Tokyo in 2007.

My current research interest is on the mechanisms of synapse development and remodeling in the mammalian cortex. Our research strategy is based on real-time imaging of synaptic molecules using modern fluorescence imaging techniques, such as in vivo two-photon microscopy.

Recent publications:
1) Nature Communications 3, 722, 2012., 2) Neuron 76, 549-564, 2012., 3) Nature Communications 4, 1440, 2013., 4) Nature Communications 5, 4742, 2014.

High resolution imaging of synaptic structure and molecules

Neuronal synapses are specialized cellular junctions formed between pre- and post-synaptic structures. Neurotransmitters released from presynaptic vesicles activate postsynaptic receptors, which are concentrated within the postsynaptic densities (PSDs). To understand the mechanisms of postsynaptic signal transduction, structural properties of postsynaptic spines and molecular organization of PSDs should be clarified. However, their small size prevents analyses of molecular localization by light microscopy. Recent progress in microscopic techniques (such as SIM, STED, and PALM/STORM) enabled us to reconstruct cellular structures with effective resolution better than the diffraction limit. We applied these high resolution techniques to the analyses of spine structure and PSD organization in cultured hippocampal neurons. Quantitative analyses revealed basic structural properties of spines and their alterations in disease-related conditions. High resolution imaging of synapses provides new information in physiology and pathology of synapses.

DU, Shengwang PhD
Associate Professor,
Department of Physics & Division of Biomedical Engineering,
The Hong Kong University of Science and Technology,
Hong Kong

Prof. Shengwang Du obtained his BS degree in Electrical Engineering from Nanjing University in 1996, MS degree in Physics from Peking University in 1999, and PhD in Physics from University of Colorado at Boulder in 2005. He had worked as postdoctoral scholar at Stanford University from 2005 to 2008 before joining the Hong Kong University of Science and Technology (HKUST). Now he is an Associate Professor at the Department of Physics and Division of Biomedical Engineering, and also the Co-director of the Super-Resolution Imaging Center, at HKUST. Prof. Du's research interests are atomic physics, quantum optics, and optical microscopy for bioimaging. Prof. Du is a co-founder of two Hong Kong companies: NanoBioImaging Ltd (NBI, for super-resolution microscopy) and Light Innovation Technology Ltd (LiT, for light-sheet microscopy and live-cell imaging).

4D Light-Sheet Fluorescence Microscopy for Live Cell Imaging: LBS 1.0

We demonstrate a simple and efficient method for producing ultrathin Bessel non-diffracting light sheets using a line-shaped beam and an annulus filter. With this robust and cost-effective technology, we built a light-sheet fluorescence microscope and obtained multicolor 4D images of biological samples with lateral/axial spatial resolution of 250 nm / 400 nm, and 2 Hz temporal resolution for a volume of $20\times20\times50$ um^3. As compared to a traditional confocal microscope, our line Bessel sheet (LBS) microscope remains near diffraction limited lateral resolution but have a higher axial resolution and much faster temporal speed. Meanwhile, the LBS microscope has a much lower level of photo-bleaching/ photo-toxicity that is only about 1/1000 of a typical confocal microscope. The LBS microscope is ideal for 4D (3D space + 1D time) live cell imaging.

YU, Cheng-han PhD
Assistant Professor, School of Biomedical Sciences,
The University of Hong Kong, Hong Kong

Cheng-han was born in Yilan, Taiwan. He graduated from National Taiwan University with a B.S. in Physics (2000). He then joined the research group of Jay T. Groves at University of California, Berkeley to complete his PhD (2002-2007), focused on lipid membrane biophysics, T cell immunological synapse, and nanofabrication technology. Continuing his research interest in dynamical regulation of cellular membrane proteins, Dr. Yu started his postdoctoral training with Michael P. Sheetz at Mechanobiology Institute (MBI) in National University of Singapore. In August 2014, Cheng-han joins the School of Biomedical Sciences in the University of Hong Kong.

Mechano-transduction of integrin signaling and podosome formation

Extracellular signaling molecules can activate designated membrane receptors upon binding and trigger receptor oligomerization. Integrin-mediated cell adhesion, a well-known example, involves micron-scale protein clustering and actin cytoskeleton remodeling at the interface of cell and extracellular matrices. Spatial-temporal clustering of integrin receptors serves as a mechano-transduction hub for downstream biochemical reactions at the cellular interface. Here, we developed nano-patterned supported RGD membranes as a novel platform to decipher how mechanical cues in matrices regulate cell adhesion transformation. Previously, we demonstrated that the formation of different adhesion structures, such as focal adhesion and podosome are modulated by mechanical characteristics of matrices. Lack of matrix-force at activated integrin clusters and decrease of RhoA-GTP levels result in podosome formation in PI3K and FAK/PYK2 dependent manner. In addition, we recently reported that the contractile force development at cell-matrix interface regulates integrin-beta3 signaling cascade. Absence of contractile force development results in down-regulation of integrin-beta3 via clathrin-mediated endocytosis. In particular, Dab2 directly binds to tyrosine residues at the cytoplasmic tail of integrin-beta3, recruits clathrin-mediated endocytic machinery, and causes the internalization of RGD-integrin clusters to Rab5-positive early endosomes. The switching of adhesion signaling by modulating extracellular microenvironment is remarkable. These findings suggest programmable adhesion signaling and adhesion transformation by matrix-mediated mechano-transduction.

KOCH, Daniel PhD
Senior Regional Application Specialist, ZEISS Group,
Carl-Zeiss-Promenade, Jena, Germany

PhD in Physics at University of Leipzig in Germany studying actin and cell dynamics. Postdoctoral Research Fellow at Georgetown University, Washington DC studying force generation of neuronal growth cones and 3D cell migration of cancer cells.
Expert on Super-Resolution Microscopy, 3D Imaging, Light Sheet Fluorescence Microscopy, Traction Force Microscopy, Cell Mechanics, Neuroscience and Cancer Research.
Senior Regional Application Specialist for ZEISS for the past 5 years based out of Germany and Singapore.

Advances in Super-Resolution and Lightsheet Microscopy for Live Cell and Deep Imaging

Observation of biological processes and structures in living specimen or fixed samples is a main achievement for experimental studies in biology and life sciences. We present novel technologies with significant improvements in sensitivity, speed, and resolution at strongly reduced laser intensities.

Airyscan introduces a new concept in confocal microscopy to collect all light of an Airy pattern simultaneously. Knowing the spatial distribution of each Airy pattern provides additional information from your sample which is used to significantly improve performance. The ZEISS LSM 8 family with Airyscan offers 4-8x higher sensitivity, 1.7x increase in resolution in 3D, as well as faster speed, at extremely low laser intensities, for standard fluorescent samples as well as in multi-photon imaging. The Airyscan Fast module further increases speed and images with up to 27 fps at 480 × 480 pixels.

Light-sheet fluorescence microscopy provides an alternative approach. The ZEISS Lightsheet Z.1 achieves high spatial resolution and fast acquisition speeds at high penetration depths that allows very gentle 3D imaging of large living samples over extended periods of time. Optical clearing methods further extend these imaging modes into the extreme deep imaging regime.

The ELYRA system, our dedicated super-resolution system, provides further increases in 3D resolution (SR-SIM) as well as 3D single molecule imaging (PALM/dSTORM) and pushes the limits of currently available fluorescence technologies.

These novel approaches provide flexible solutions in 3D imaging with each having unique features and distinct advantages. Based on the experimental needs one can choose the optimal acquisition strategy.

LAI, Cora Sau Wan PhD
Assistant Professor, School of Biomedical Sciences,
The University of Hong Kong, Hong Kong

Dr. Cora Lai obtained her bachelor and doctoral degrees at HKU. She later joined the Skirball Institute of Biomolecular Medicine in the Langone NYU Medical Center (New York, USA) for postdoctoral training. At NYU, she has been focusing on intravital imaging of the mouse central nervous system in learning and memory, particularly in studying synaptic plasticity in fear associative learning. She is a faculty in the School of Biomedical Sciences, HKU since 2014.

Intravital imaging in the mouse brain

In the past two decades, the development of multi-photons microscopy and *in vivo* fluorescence labeling techniques has opened an expanding field of high-resolution imaging studies in intact tissues and living animals. The imaging of live animals at microscopic resolution is a powerful tool to reveal dynamic biological processes over time and space under environments that are close to physiological conditions. In this talk, I will first review some of the recent intravital imaging techniques and fluorescence probe development in neurobiology. Further on, I will discuss how I applied the intravital two-photons imaging technique in the study of synaptic plasticity in learning and disease.

Programme in details

Conference – Day 1
December 5, 2016 (Monday)

08:30 – 17:30	**Registration**

Time	Programme	
	Lecture Theatre 4	
09:00 – 09:20	**Opening Ceremony** **Guest of Honor:** Professor LEUNG, Suet-yi Associate Dean (Research), LKS Faculty of Medicine The University of Hong Kong, Hong Kong	
Plenary Lecture 1		
09:20 – 10:05	**Chairperson: CHAN, Ying Shing** (The University of Hong Kong, Hong Kong) **Embryonic patterning: Insights from developmental spatial transcriptomics of gastrulation** **TAM, Patrick** (University of Sydney, Australia)	
10:05 – 10:50	**Chairperson: LEE, Wang Jae** (Seoul National University College of Medicine, Korea) **Chinese Visible Human and Digital Medicine** **ZHANG, Shaoxiang** (Third Military Medical University, China)	
10:50 – 11:15	**Coffee Break / Poster Session**	
	Lecture Theatre 3	**Lecture Theatre 4**
	SYM 1.1 **Advances in Neuroscience** **Chairperson:** **CHAN, Sun On Hector** (The Chinese University of Hong Kong, Hong Kong) **CHANG, Raymond Chuen Chung** (The University of Hong Kong, Hong Kong)	**SYM 1.2** **Innovations in Teaching Histology** **Chairperson:** **HING, Hiang Lian** (Universiti Kebangsaan Malaysia, Malaysia) **CHUNG, Sookja Kim** (The University of Hong Kong, Hong Kong)
11:15 – 11:33	**Calcium-responsive transcription coactivator (CREST) promotes neuroblastoma cell differentiation** **LI, He** (Tongji Medical College of HUST, China)	**Innovations of Curriculum Construction for Histology & Embryology Course in the Era of Education Informatization** **CHEN, Hong** (Fudan University, China)
11:33 – 11:51	**Maternal diabetes alters expression of microRNAs that regulate genes critical for neural tube development** **DHEEN, S Thameem** (National University of Singapore, Singapore)	**The reform of histology education: then as student, and now as teacher** **CHUNG, Sookja Kim** (The University of Hong Kong, Hong Kong)
11:51 – 12:09	**Molecular determinants of synapse development: focus on dendritic mRNAs** **LAI, Kwok On** (The University of Hong Kong, Hong Kong)	**Team based learning and remediation in histology** **LEE, Jong Eun** (Yonsei University, Korea)
12:09 – 12:27	**Dendritic Spine Plasticity in Depression** **LAI, Cora Sau Wan** (The University of Hong Kong, Hong Kong)	**Implementation of An Online Digital Learning Platform for Studying Histology and Other Medical Morphological Disciplines** **LI, He** (Tongji Medical College of HUST, China)
12:27 – 12:45	**What happen in basketball players' brain?** **RHYU, Im Joo** (Korea University, Korea)	**Developing clinical reasoning in histology course by using team-based approach with audience response system questions** **PAWLINA, Wojciech** (Mayo Clinic College of Medicine, USA)
12:45 – 14:00	**Lunch Break / Poster Session**	

Conference – Day 1

December 5, 2016 (Monday)

Time	Programme	Time	Programme
Lecture Theatre 3		**Lecture Theatre 4**	
SYM 2.1 **Advances in Developmental Studies** **Chairperson:** **CHAN, Wood Yee** (The Chinese University of Hong Kong, Hong Kong) **SHAM, Mai Har** (The University of Hong Kong, Hong Kong)		**SYM 2.2** **Good practices in Teaching Gross Anatomy** **Chairperson:** **DHEEN, S Thameem** (National University of Singapore, Singapore) **CHEUNG, Annie Lai-Man** (The University of Hong Kong, Hong Kong)	
14:00 – 14:20	**Asymmetric localization of Dlc1 defines neural crest polarity for directional migration** **CHEUNG, Martin Chi Hang** (The University of Hong Kong, Hong Kong)	**14:00 – 14:18**	**Free regional anatomy textbook, full of schematics and mnemonics** **CHUNG, Min Suk** (Ajou University, Korea)
14:20 – 14:40	**Redox homeostasis in stem cell maintenance** **BAEG, Gyeong Hun** (National University of Singapore, Singapore)	**14:18 – 14:36**	**Today's Anatomy Educator** **DRAKE, Richard** (Cleveland Clinic Lerner College of Medicine, USA)
14:40 – 15:00	**Wnt Regulation of Planar Cell Polarity** **GAO, Bo** (The University of Hong Kong, Hong Kong)	**14:36 – 14:54**	**(TBC)** **LIU, Shuwei** (Shandong University, China)
15:00 – 15:30	**Fgf signaling pathways in mouse development** **SORIANO, Philippe** (Icahn School of Medicine at Mount Sinai, USA)	**14:54 – 15:12**	**Gross anatomy- the foundation for producing good medical doctors** **DAS, Srijit** (Universiti Kebangsaan Malaysia Medical Centre, Malaysia)
		15:12 – 15:30	**Good Practices in Teaching Gross Anatomy** **MAHAKKANUKRAUH, Pasuk** (Chiang Mai University, Thailand)
15:30 – 16:00	**Coffee Break / Poster Session**		
Lecture Theatre 4			
Plenary Lecture 2			
	Chairperson: SHAM, Mai Har (The University of Hong Kong, Hong Kong)		
16:00 – 16:45	**Transcriptional and non-transcriptional functions of Irx homeodomain proteins** **HUI, Chi Chung** (University of Toronto, Canada)		
	Chairperson: TSAO, George Sai Wah (The University of Hong Kong, Hong Kong)		
16:45 – 17:30	**Our Responsibility in Building a Future for Anatomists** **TOPP, Kimberly** (University of California, USA)		
18:30 – 21:00	**Banquet** **(Full registration participants and invited guests)** **Golden Lilies Banquet, Cyberport, Pokfulam**		

TAM, Patrick BSc(Hons), MPhil, PhD CBiol
Professor, Children's Medical Research Institute and School of Medical Sciences, Sydney Medical School, The University of Sydney, Australia

Patrick Tam is the Deputy Director and Head of the Embryology Research Unit at the Children's Medical Research Institute; Professor in the School of Medical Science, Sydney Medical School of University of Sydney and Mok Hing-Yiu Distinguished Visiting Professor of the University of Hong Kong. His research focuses on the systems-based investigation of the functional attributes of gene regulatory network in body patterning during mouse development and the biology of stem cells. The embryological research has revealed the cellular and molecular mechanisms that underpin the organization of the basic body plan of the early embryo. The knowledge of lineage differentiation laid the foundation for directing the first steps of differentiation of stem cells into clinically useful cell types. Patrick Tam is an Editor of the journal *Development* and member of the editorial board of *Developmental Cell, Developmental Biology, Differentiation* and *Genesis*. He chairs the scientific advisory board of Stem Cell Australia, and serves on the advisory council of RIKEN Centre for Developmental Biology. He was the President's Medalist of the Australian and New Zealand Society for Cell and Developmental Biology and elected Fellow of Australian Academy of Science, Australian Academy of Health and Medical Sciences, Royal Society of Biology and Royal Society of London.

Embryonic Patterning: Insights from Developmental Spatial Transcriptomics of Gastrulation

Gastrulation is a critical milestone of early mouse embryogenesis at which the primary germ layers are formed and the multipotent embryonic cells are allocated to the progenitors of major tissue lineages. Fate mapping of the mouse embryo has revealed a distinctive regionalization of cell fates in the embryo before gastrulation is completed, and lineage tracing studies showed that, concurrent with the formation of primary germ layers, the differentiation potency of embryonic cells is progressively restricted, culminating in the generation of lineage-restricted progenitors for specific tissue types. While snapshots of the expression of a selected set of genes may inform us of the signalling activity, tissue patterning and acquisition of specific cell fates, developmental transcriptome of the embryo has offered additional insights into the genetic activity that underpins the developmental transition through gastrulation. To investigate the genetic activity that mediates the regionalization of cells fate and the organization of the body plan during the transition from cellular pluripotency to the restriction of lineage potency, we performed a high-resolution RNA-seq analysis on single gastrulation stage mouse embryos to collate a developmental series of spatial transcriptome of cell populations at defined locations in the epiblast. Outcome of the analysis of the transcriptome along the development timeline and in correlation with the regionalization of cell fates in the embryo highlights the progressive delineation of region-specific transcriptional activity in the epiblast, and provides the molecular annotation of the process of germ layer formation and a first glimpse of the genome activity in lineage specification and differentiation.

ZHANG, Shaoxiang PhD, MD
President, International Society of Digital Medicine (ISDM)
Chairman, Chinese Society of Digital Medicine
President, Chinese Association of Anatomical Sciences
Editor-in-chief of Digital Medicine
Professor & Director, Institute of Digital Medicine
Third Military Medical University (TMMU), China

Chinese Visible Human and Digital Medicine

In 2002, our team at the Third Military Medical University accomplished the project "Creation of the Chinese Visible Human (CVH) Data Set". To date, 6 high-precision datasets of CVH have been obtained, which accelerates the basic research on Digital Medicine (DM) in China. In recent years, research on DM in China has been transited from "CVH" to "digital physical human" and "digital physiological human"; from theoretical to applied research in various fields. Such as, in diagnosis of ultrasonography, a digital human model and ultrasound section image matching system has been developed, with the purpose of simulating the corresponding section images provided by ultrasonography based on the digital human model, to assist clinicians in identifying the images of patients' body structures; as to radiotherapy simulation, we have built a simulation system for radiotherapy, which can accurately locate the foci, applicable to conformal and intensity-modulated radiotherapy; in simulation of orthopedic surgery, we have conducted a research on simulated screw fixation in vertebral arch surgery, in which the injures to nerves and vessels can be effectively prevented from in light of the accurate position of screw; in terms of interventional diagnosis and treatment, we simulated coronary arteriography surgery and the process of coronary stent implantation, and further research on actual training system for simulated operation is underway; in research on digitized plastic surgery, we simulated the predicted-appearance after surgery, to adjust the relative parameters of surgery; in terms of digitized teaching, we have established the Digital Anatomy Teaching System, which is applied to teaching and learning of human anatomy; our team also has made great effort in building research institutes and academic organizations of DM , such as, Chinese Society of Digital Medicine established on 5 May, 2011, and International Society of Digital Medicine(ISDM) established on 17 June, 2016. With in-depth research and advanced techniques, CVH will be applied more widely in digital medicine.

LI, He PhD
Professor, Division of Histology and Embryology,
Department of Anatomy, Tongji Medical College,
Huazhong University of Science and Technology, China

He Li, PhD, received his doctorate degree in Neurobiology from Tongji Medical University, and completed his postdoctoral training at both Kyoto University (1995 to 1997) and Emory University (1998 to 2001). Dr. Li is currently the Head Professor in Histology and Embryology at Tongji Medical College, Huazhong University of Science and Technology. His research interests focus on the molecular mechanisms underlying genetic neurodegenerative diseases, intracellular trafficking of endocrine cells, and neuropathway tracing. Dr. Li currently is Chairman of Histology and Embryology Branch of Chinese Society for Anatomical Sciences, the Editor-in-Chief of Chinese Journal of Histochemistry and Cytochemistry, a member of Editorial Board of Acta Histochemica et Cytochemica and of Histochemistry and Cell Biology, a member of the Scientific Advisory Board of the International Congress of Histochemistry and Cytochemistry (ICHC). He is the recipient of The National Science Fund for Distinguished Young Scholars, College Young Teachers Award and Renowned Teacher Award of Hubei Province.

Calcium-responsive transcription coactivator (CREST) promotes neuroblastoma cell differentiation

Calcium-responsive transcription coactivator (CREST), a neuronal nuclear protein, can mediates calcium influx-induced gene transcription and basic transactivation via recruiting histone acetylases, such as CBP and P300. It has been reported that CREST plays an important role in neuronal development, promoting growth and branching of neuronal processes. However, the relevance of CREST to neuronal differentiation is unknown. In the present study, the role and the underlying mechanism of CREST in neuronal differentiation was examined in neuroblastoma (N2a) cells. Our results indicate that CREST might increase the stability and activity of p53 via recruiting histone acetylases, such as CBP and P300, and then promoting the acetylation of p53, which increases the expression of p21 and decreases phosphorylation of Rb through inhibition of activity of cyclin-dependent kinases (CDKs), including CDK2 and CDK4, therefore down-regulating cyclinD1, E1 and A1, results in the arrest of cell cycle, promoting differentiation of N2a cells. This work was supported by NSFC (31171155).

DHEEN, S Thameem BSc, MSc, MPhil, PhD
Assoc Professor, Head of Department of Anatomy, Yong Loo Lin School of Medicine, National University of Singapore, Singapore

Maternal diabetes alters expression of microRNAs that regulate genes critical for neural tube development

Maternal diabetes has been shown to alter the expression of developmental control genes contributing to the neural tube patterning defects in embryos and neuropsychological deficits in adults. The regulation of gene expression during development involves activation of epigenetic mechanisms which include chromatin reorganization, histone modification, DNA methylation and altered expression of micro RNAs (miRNAs). Recently, miRNAs have been shown to regulate neural tube formation and critical for brain development. In view of this, we hypothesized that maternal diabetes alters the expression of miRNAs that regulate genes involved in neurogenesis and neural tube development. To address this, we performed miRNA expression profiling in neural stem cells (NSCs) isolated from the forebrain of embryos from normal or streptozotocin-induced diabetic pregnancy.

Distinct miRNA expression patterns were observed in NSCs from normal and diabetic pregnancy. Pathway analysis identified several molecules involved in various developmental events including axon guidance to be deregulated in NSCs from diabetic pregnancy. Among the differentially expressed miRNAs, the expression of miRNA-30 family was upregulated in NSCs from diabetic pregnancy when compared to the control. miRNA-30b has been predicted to target Sirt1, a silent information regulator 2 (Sir2) ortholog, which is involved in the self-renewal, multipotency, and fate determination of NSCs. Overexpression or knockdown of miR-30b in NSCs, resulted in altered expression of Sirt1, leading to altered lineage specification of NSCs suggesting that miR-30b regulates NSC fate *via* Sirt1. Overall, these results indicate that altered miRNA expression induced by maternal diabetes may form the basis for deregulation of several metabolic pathways resulting in defective brain development and function.

Dheen ST, Sukanya S, Ramya S and Bay BH
Department of Anatomy, Yong Loo Lin School of Medicine, National University of Singapore, Singapore

LAI, Kwok-On PhD
Assistant Professor, School of Biomedical Sciences,
The University of Hong Kong, Hong Kong

Dr. Kwok-On Lai received his Bachelor degree of Biochemistry from the University of Hong Kong. He then pursued postgraduate studies at the Hong Kong University of Science and Technology (HKUST). With the award of the Croucher Foundation Fellowship, he performed postdoctoral research at UCLA in the United States.

Using a combination of molecular, biochemical and cellular approaches, Dr. Lai aims to elucidate the signal transduction mechanisms that underlie the development and plasticity of neuronal synapses in the brain. This would lead us to a better understanding of the cellular and molecular mechanisms underlying long-term memory, and the pathophysiology of various neurodevelopmental disorders in the brain.

Molecular determinants of synapse development: focus on dendritic mRNAs

Most excitatory synapses of the postsynaptic neuron in the brain are located on dendritic spines, which are heterogeneous and exist with various shapes. The maturation of dendritic spines requires local dendritic synthesis of new proteins in response to spontaneous glutamate release; on the other hand dysregulated dendritic mRNA transport and protein synthesis can lead to altered spine morphology in neurodevelopmental disorders such as Fragile-X syndrome and autism. Nonetheless, the molecular mechanism underlying activity-dependent spine maturation is not fully understood. Dendritic RNA granules are believed to be transported along microtubules by the molecular motors kinesin and dynein. In this presentation I will describe the functional role of kinesin in dendritic spine development of hippocampal neurons. I will also discuss our recent findings on how dendritic spine morphology is controlled by the scaffold protein STRN4, the mRNA of which is dendritically localized and its expression is tightly regulated by synaptic activity.

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LAI, Cora Sau Wan PhD
Assistant Professor, School of Biomedical Sciences,
The University of Hong Kong, Hong Kong

Dr. Cora Lai obtained her bachelor and doctoral degrees at HKU. She later joined the Skirball Institute of Biomolecular Medicine in the Langone NYU Medical Center (New York, USA) for postdoctoral training. At NYU, she has been focusing on intravital imaging of the mouse central nervous system in learning and memory, particularly in studying synaptic plasticity in fear associative learning. She is a faculty in the School of Biomedical Sciences, HKU since 2014.

Dendritic Spine Plasticity in Depression

Depression is a mental disorder that is estimated by the World Health Organization to affect 350 million people worldwide. Depression is a leading cause of disability that severely affects the patient's work and life. Although the number of individuals with depression is constantly growing, the understanding of the pathogenesis of depression is still insufficient and effective treatments are lacking. Antidepressants are commonly used as treatments for depression, such as selective serotonin reuptake inhibitors, which increase the availability of synaptic monoamine neurotransmitters. The use of these treatments is based on the theory that neurotransmitter imbalances lead to depression. However, the therapeutic effects of these antidepressants emerge only after weeks of chronic administration, even though they increase the availability of synaptic monoamine levels within hours, which suggest that this hypothesis is insufficient. Furthermore, these antidepressants also show a high percentage of treatment resistance. Despite decades of research, the pathogenesis of depression is still not clear. Our limited understanding could be due to the lack of high-resolution longitudinal studies of the pathological changes in the progression of depression in patients. Therefore, we used an animal model of depression with in vivo imaging to study the short- and long-term dendritic spine plasticity in the progression of depression by high-resolution two-photon microscopy. We also explored the antidepressant effects of ketamine in regards to its short- and long-term effects on dendritic spine plasticity in the animal depression model.

RHYU, Im Joo MD, PhD
Professor, Department of Anatomy, College of Medicine,
Korea University, Korea

Rhyu IJ is a neuroanatomist with a background in medicine. He is interested in macroscopic and microscopic plastic response of the nervous system in response to various stimulations, especially motor activities. He demonstrated macroscopic brain plasticity of athletes with MRI and he investigates ultrastructure of the nervous tissue with various microscopic technique. With wide spectrum of morphological research tools, he is contributing to understand brain structure and function.

What happen in basketball players' brain?

My laboratory has investigated brain plasticity in response to motor activity in acrobat animal model, treadmill running monkey, and elite sportsmen with wide range of tools from electron microscope to MRI. I have analyzed the brains of basketball players with MRI based on the hypothesis, brain regions related with motor control would have morphological plasticity. I found cerebellar vermis lobules (VI, VII) of basketball players are larger than that of control, which looks like to be influenced by white matter contribution. In addition to cerebellum, striatum volume was also increased in basketball players. The manual volumetric analyses of basketball players' brain limit further investigation of other brain regions might be changed in the athletes. Therefore, I employed the latest MRI analysis tools including VBM, Brain Surfer and FSL to understand holistic morphological evaluation of the basketball players.

The VBM analysis result showed that increase volume of white matter of left precentral gyrus, and right superior semiluar lobule adjacent to vermian lobule VI, VII. Free Surfer analysis revealed increased cortical thickness of left precentral gyrus. FIRST analysis showed shape alteration was detected in left thalamic nuclei such as ventral anterior, ventrolateral, and pulvinar. In addition, left amygdala also showed morphological change in basketball players.

This study implies that plastic change of cortico-striato-thalamo-cortical loop and cortico-cerebello-thalamo-cortical loop are key modules in motor activities of the basketball players. In addition, this study suggests that non-motor function might contribute to these elite athletes.

Im Joo RHYU[1], Nam Joon LEE[2]
[1]*Department of Anatomy, Korea University College of Medicine*
[2]*Department of Radiology, Korea University College of Medicine*

CHEN, Hong BMed, MMed, PhD, Reprod.Biol.
Professor, Department of Anatomy, Histology & Embryology,
Fudan University, Shanghai, China

Dr. CHEN Hong, a Mrs. Ivy Wu Fellow of HKU in 1999, is now a Full Professor of Shanghai Medical College of Fudan University, and a Course Leader of Histology & Embryology (English) that is now awarded as a Shanghai Model Course for Overseas Students. She got her MD degree in Shanghai Medical University in 1990 and PhD degree in HKU in 2004, and finished her Postdoc training in University of Delaware, USA. Concurrently, Prof. Chen is a regular member of SSR of USA, and peer reviewers of international journals, such as *PLoS ONE, Antioxid Redox Signal., etc.* As a course leader, she got the 2nd Prize of Shanghai Teaching Achievements Awards in 2013, while as a mentor of graduate student, the Prize of Shanghai Excellent Graduate Thesis Awards in 2014. Now Prof. Chen is mainly engaged in spermatids differentiation and related epigenetic mechanisms. Since 2009, Prof. Chen has got 4 national grants supported by NSFC and 973 Program Subproject, respectively. And 14 papers are published in the international peer-reviewed journals, such as *Cell, FRBM, ARS, JCMM, BOR, etc.* Prof. Chen also got one of the USA patents, and as editor-in-chief, published an English textbook entitled *Practical Manual of Histology.*

Innovations of Curriculum Construction for Histology & Embryology Course in the Era of Education Informatization

Histology and Embryology course is one of the most important fundamental courses to provide medical students with comprehensive understand of the human body at both the gross anatomy and microanatomy level. This course also form a concrete basis for the future studies in Pathology and Pediatrics studies. Recently, under the impact of new concept of *public innovations and internet plus* proposed by Premier Li Keqiang, we are continuously and actively collaborating with the University of Hong Kong Department of Anatomy (now the School of Biomedical Sciences) to pursue the mission for core course construction in Medical School of Fudan University. The mission is to build up a curriculum for basic medical sciences with the traits of the newest textbook, the best faculty, the most effective approach, and the most reasonable evaluation system. To realize the mission in our Department, the innovations we made cover the following six aspects: 1) a new teaching concept upon the internet plus idea; 2) construction of an English/bilingual textbook; 3) multifaceted teaching format and approach; 4) formative and summative evaluation system; 5) online learning platform for self-study and self-assessment; 6) cultivation of internationalized teaching faculty. We strongly believe that these innovations covering above aspects will create a better learning experience for medical student education.

CHUNG, Sookja Kim BA, MA, PhD
Professor, School of Biomedical Sciences,
The University of Hong Kong, Hong Kong

Sookja K. Chung is currently a Professor at School of Biomedical Sciences, Honorary Professor at Dept. Ophthalmology at HKU and Visiting Professor at 4th Military Med Sch, Xian. She also held Research Fellow position (equivalent to Honorary Professor) at Shanghai Jiao Tong University. She is a member of State Key Laboratory for Pharmaceutical Biotechnology. She is an elected member of International Molecular Biology Network. She received BA. degree double majoring in Biology and Chemistry, MA at Department of Chemistry, University of Illinois at Chicago, USA and PhD degree at Department of Anatomy & Cell Biology, University of Illinois College of Medicine, Chicago. She was NIH Postdoctoral Fellow at Northwestern University Medical Center and Norman and Rosita Winston Foundation Fellow at The Rockefeller University in New York. Since 1991, she has been with The University of Hong Kong starting as Research Officer at Institute of Molecular Biology to Professor in the School of Biomedical Sciences (Anatomy Division). She is the coordinator of Histology. Her main research focus is to use genetically engineered mouse models with pharmacological approaches to understand the gene functions in osmotic/oxidative/ischemic stresses, and dysfunction under human disease conditions, such as diabetes and its complications involving macrovascular and microvascular system.

The reform of histology education: then as student, and now as teacher

The importance of medical science-based education has been emphasized as early as 1910 by Abraham Flexner, a research scholar at the Carnegie Foundation for Advancement of Teaching. Histology is often taught in first year of medical education as basic science courses emphasizing the function of cells, tissues and organs at the cellular and molecular level. The histology educators are usually engaged in biomedical science research and provided the students with the up-to-date scientific breakthrough in cell biology and histology. As a student at the University of Illinois, I witnessed the traditional way of teaching and learning histology, which involved viewing microscope slides with biological specimens prepared by histotechnologists in special laboratory. The student evaluation also took place in a large laboratory, where students are asked to go through the multiple microscopes to answer the specific question under each microscope. The lecture notes are distributed to students as co-Op notes approved by lecturers. However, as a teacher at The University of Hong Kong, I have been involved in some reform in Histology education, although we still provide ample lectures and practicals. In addition to the traditional way of teaching using microscope, we have introduced a computer-assisted learning of histology by making use of the Aperio System. In addition, we also provide HKU Anatomy Atlas as a study aid. As for my Alma Mater, I understand that the histology education is minimum. The changes in histology education, then, now and future will be discussed in detail.

LEE, Jong Eun PhD
Professor, Department of Anatomy,
Yonsei University College of Medicine, Seoul, South Korea

Lee, Jong Eun, Department of Anatomy, Yonsei University College of Medicine obtained PhD degree majoring in Biochemistry from Yonsei University at Feb 1993. She is a Vice Dean of Student Affairs, Yonsei University College of Medicine and is working for medical education since Mar 1985 in different teaching capacities. She had served as a Chair Professor in Department of Anatomy, Yonsei University College of Medicine from Mar 2008 to Feb 2012. She did her postdoctoral studies in Department of Anesthesia, Stanford University Medical School, USA during 1996-98. She served as a visiting associate professor (July-Sep 2000) and as a visiting scientist (Jan-Feb 2002) in Stanford University, USA. She obtained Womens international scientific cooperation travel grant funded by American Association for the Advancements of Science (2002) and been honored with HanGok award for superior achievement and contribution to Korean Society of Anatomists (2005). She is an editorial board member for the Korean Journal of Anatomy since 2003 and now the executive editor for the Anatomy and Cell Biology and Experimental Neurobiology. Currently her research interests lies in developing the new functional stem cell and their therapeutic potential in various neuronal disease models and in establishing the therapeutic strategy for Alzheimer's pathology.

Team based learning and remediation in histology

There have been various changes in the education of Anatomy in the last few years in medical colleges in South Korea. The Federation of Korean Basic Medical Scientists set the students of the virtue which they will be required in the future as medical doctor and has proposed that medical school should provide to students the adequate educational courses in order to help them acquire such characters. Medical college of Yonsei University pursuits linkages based on integrated SPICE modules through CDP2004 program. It provides autonomy to students through diversification of education such as problem-based learning, therefore pursuing education solely focused for students. The CDP2013 developed our mission further and brought student-oriented learning, outcome-based learning, research-oriented learning, and integrated learning (SORI). Such waves in education at medical colleges have led to the following changes at Yonsei University: Histology and Physiology are integrated into one subject such as 'Cell Structure and Function', and particular classes of Histology are dealt in modules of circulatory system, respiratory system, digestive system, reproductive system and endocrine system. Medical college of Yonsei University approves team-based learning not only for the integration of modules but also for student focused education. In addition, grades are marked on an absolute evaluation, instead of a curve evaluation, which means scoring over 80% is in a pass, and students who receive a non-pass will have to study again in order to receive a pass for the final grade. It will demonstrate how the outcome-based education is managed within the course of Histology.

LI, He PhD
Professor, Division of Histology and Embryology,
Department of Anatomy, Tongji Medical College,
Huazhong University of Science and Technology, China

He Li, PhD, received his doctorate degree in Neurobiology from Tongji Medical University, and completed his postdoctoral training at both Kyoto University (1995 to 1997) and Emory University (1998 to 2001). Dr. Li is currently the Head Professor in Histology and Embryology at Tongji Medical College, Huazhong University of Science and Technology. His research interests focus on the molecular mechanisms underlying genetic neurodegenerative diseases, intracellular trafficking of endocrine cells, and neuropathway tracing. Dr. Li currently is Chairman of Histology and Embryology Branch of Chinese Society for Anatomical Sciences, the Editor-in-Chief of Chinese Journal of Histochemistry and Cytochemistry, a member of Editorial Board of Acta Histochemica et Cytochemica and of Histochemistry and Cell Biology, a member of the Scientific Advisory Board of the International Congress of Histochemistry and Cytochemistry (ICHC). He is the recipient of The National Science Fund for Distinguished Young Scholars, College Young Teachers Award and Renowned Teacher Award of Hubei Province.

Implementation of An Online Digital Learning Platform for Studying Histology and Other Medical Morphological Disciplines

The effectiveness and efficacy of Histology learning is limited by the availability of equipment and materials, such as microscopes and teaching slides, and the presence of teachers/demonstrators, which are only provided to students during practical sessions. Therefore, there is an increasing need to provide students with resource to make use of the off-class time for self-learning of histology. Here we have implemented an online digital learning platform for students to view histology slides. This web based platform is user-friendly to every common operating system, and is able to show slides in a series of magnification, ranging from 0.31X to 40X, which is useful especially on devices with touch screen. With the structures labeled beforehand, text and video descriptions to each slide, the platform can transfer histology practical teaching into a flipped-classroom manner. The quiz bank module is also available for online examination paper formation and student assessment. Moreover, the platform integrates other morphological disciplines, such as pathology and parasitology, providing Organ and System Based Education (OSBE) to current medical students. This cost-effective online digital learning platform could serve as a promising and powerful teaching tool to medical faculties, and students could study histology in practice with this platform anytime, anywhere.

PAWLINA, Wojciech MD
Professor of Anatomy and Medical Education, Department of Anatomy,
Mayo Clinic College of Medicine, Rochester, MN, USA

Wojciech Pawlina, MD, is a Professor of Anatomy and Medical Education at Mayo Clinic College of Medicine in Rochester, Minnesota, USA. He earned his medical degree from the Jagiellonian University College of Medicine in Cracow, Poland, where he worked in the Department of Anatomy while completing his residency in Obstetrics and Gynecology. Since 1986 he worked in the Department of Anatomy and Cell Biology at the University of Florida College of Medicine. In 1999 he relocated to Mayo Clinic in Rochester, Minnesota and currently serves as Chair of the Department of Anatomy and Director of the Procedural Skills Laboratory. He teaches gross anatomy, histology, and embryology to medical and dental students, as well as residents and fellows. He received numerous teaching awards from both the University of Florida and Mayo Clinic including Distinguish Mayo Educator Award. His research interest is directed towards strategies of implementing innovative teaching methodologies in anatomical sciences education, teaching professionalism, leadership, and teamwork in early medical curriculum. He has co-authored several textbooks in the field of anatomy, histology and medical education, as well as numerous scientific papers. He serves on several editorial boards of scientific journals and is also Co-Editor-in-Chief of the Anatomical Sciences Education.

Developing clinical reasoning in histology course by using team-based approach with audience response system questions

Decreasing contact hours in medical school curricula require educators to develop new teaching strategies in order to maintaining effective learning process. Histology courses in the United States medical curricula are undergoing identity crisis. They often loose independent course status as they are incorporated into integrated medical curricula. Many institutions converted the majority of histology laboratory instructions to self-study online modules that utilize virtual microscopy. At Mayo Medical School, histology is a standalone course integrated with genetics course for a 6 week-long didactic block. The team-based learning approach utilizing virtual microscopy and audience response system (ARS) is directed toward learning histology as a tool in solving clinical problems. Students are provided resources for preparation for each session. Each histology session starts from ARS questions that show images and short clinical cases involving a disease process in specific organ systems that are relevant to the subject discussed. Students are allowed 2-3 minutes to discuss each case among them and select the correct answer. Faculty members explain each question with clinical reasoning steps that should lead students to the correct recognition of provided tissue or the correct clinical diagnosis. The ARS questions allow students to see differences between the normal as well as the pathological changes in tissues and organs. Visual display of class performance showing students answered choices for each ARS scenario provides them with immediate feedback on their performance. Student feedback has been positive and ARS is regarded as an essential tool that enhances the performance of students in medical curriculum.

CHEUNG, Martin Chi Hang PhD
Assistant Professor, School of Biomedical Sciences,
The University of Hong Kong, Hong Kong

Dr. Martin Cheung received his BSc. in Biochemistry from the Chinese University of Hong Kong and PhD in the Division of Genetics from the University of Nottingham. He then went on to carry out his Medical Research Council postdoctoral fellowship under the mentorship of Prof. James Briscoe at the National Institute for Medical Research, where he demonstrated the importance of SoxE family transcription factors in neural crest development. He then joined HKU as a Research Assistant Professor in the former Department of Biochemistry and became Assistant Professor in the School of Biomedical Sciences. His lab's research focuses on deciphering the molecular regulatory circuitry that orchestrates cell migration in both neural crest development and neural crest-derived melanoma.

Asymmetric localization of Dlc1 defines neural crest polarity for directional migration

Coordinated regulation of directional neural crest delamination is essential for the subsequent migration and differentiation into different derivatives. Among the molecules that regulate neural crest development, SoxE family transcription factors, in addition to acting as neural crest specifiers, together with Rho GTPases play key roles in directing neural crest migratory behavior but how these two classes of factors are functionally linked is unclear. Here, we show that RhoA is highly active and enriched at the rear part of the cell body while it is also dynamically localized at the protrusive front in the direction of migration. This polarized RhoA activity determines the head-tail (or front-rear) polarity of the cell for directed migration and is regulated by an asymmetric expression of Rho GAP isoform, Dlc1α. Both dominant-negative inhibition and overexpression of Dlc1α results in reduced neural crest delamination due to altered front-back polarized RhoA activity that affects directional migratory behavior. Furthermore, mass spectrometry analysis identify Nedd9 to partner with Dlc1α and is required for its polarized expression. Dlc1 and Nedd9 are subjected to the transcriptional regulation of SoxE family members. Together, these data reveal a novel SoxE-Dlc1/Nedd9-RhoA regulatory axis to govern directional delamination of neural crest cells.

BAEG, Gyeong Hun PhD
Assistant Professor, Department of Anatomy,
National University of Singapore, Singapore

Dr. Baeg Gyeong Hun has been working as an Assistant Professor in the Department of Anatomy, Yong Loo Lin School of Medicine, National University of Singapore, Singapore since 2012. He completed his PhD in Biomedical Science at the Osaka University School of Medicine, Osaka, Japan and his Postdoctoral training in Dr. Norbert Perrimon's laboratory in the Department of Genetics, Howard Hughes Medical Institute, Harvard Medical School, USA. He had also worked as an Assistant Professor in the Department of Pediatrics at New York Medical College, USA between 2006 and 2011. Dr. Baeg's research interest encompasses "Redox homeostasis in stem cell maintenance and "The JAK/STAT signalling pathway in cancer".

Redox homeostasis in stem cell maintenance

Reactive oxygen species (ROS) are byproducts produced by cellular metabolism. Dysregulated ROS levels are closely associated with various human diseases, including cancer. Cancer stem cells (CSCs), a subset of cancer cells, are responsible for pathophysiological mechanisms underlying the various types of cancers, and considered one of the primary causes for drug resistance and tumor recurrence. Interestingly, oxidizing agents selectively eliminate leukemia CSCs by increasing ROS production, suggesting that CSCs are sensitive to redox alterations. CSCs share many properties with normal stem cells; they self-renew, have potent differentiation capacity and maintain low ROS. Hence, characterizing the mechanisms utilized by ROS to influence the behavior of normal stem cells may yield valuable insights into how CSCs are regulated by redox changes. We are utilizing the *Drosophila* testis and human embryonic carcinoma Ntera2 cells as a model to investigate the effects of ROS on stem cell behavior. In testis, excessive amounts of ROS induced by the modulation of Keap1/Nrf2 activity led a decrease in germline stem cell (GSC) number by promoting premature GSC differentiation through the activation of EGFR signaling. By contrast, low ROS triggered an over-growth of GSCs. In Ntera2 cells, oxidative stress suppressed the expression of stemness genes, including Nanog, Oct4 and Tdgf1, while enhancing the spontaneous expression of neuronal marker genes such as NEUOD1 and TUJ1. The cells with high ROS showed a neuronal morphology, extending out long neurite-like processes. Our study suggests that the balance of redox homeostasis facilitates stem cell behavior across phyla. In an attempt to identify the molecular regulators of redox homeostasis within normal stem cells and ROS-associated effectors of stem cell behavior, genome-wide *in vivo* RNA interference screen and gene/microRNA expression profiling are currently being performed.

GAO, Bo PhD
Assistant Professor, School of Biomedical Sciences,
The University of Hong Kong, Hong Kong

Dr. Gao obtained his bachelor and doctoral degrees from the Shanghai Jiao Tong University in Shanghai, China. He received his research training in the fields of human and mouse genetics. After completing a postdoctoral fellowship in mammalian developmental genetics at U.S National Human Genome Research Institute, he worked as staff scientist at the National Institutes of Health (NIH). In 2015, Dr. Gao joined The University of Hong Kong as Assistant Professor. He is interested in studying major signaling pathways in both normal developmental processes and human diseases. His current research include Wnt/Planar Cell Polarity (PCP) signaling and human skeletal disorders.

Wnt Regulation of Planar Cell Polarity

Cell signaling is a major strategy that all living beings take to control basic cellular activities and coordinate their actions across tissues and organs. It plays fundamental roles in both development and physiology by spatial and temporal coordination of cell proliferation, differentiation, survival and polarity. We are interested in understanding the molecular mechanism of how Wnt signaling regulates Planar Cell Polarity (PCP), which is an evolutionarily conserved developmental process whereby a field of cells is able to point in the same direction. Perturbation of Wnt/PCP signaling underlies skeletal disorders, polycystic kidney disease and a broad spectrum of neural tube defects. We use molecular, cellular, developmental and genetic approaches to investigate how Wnt signals are integrated to establish PCP and their functions in development and disease.

SORIANO, Philippe PhD
Professor, Developmental and Regenerative Biology,
Icahn School of Medicine at Mount Sinai, New York, USA

Dr. Philippe Soriano is an accomplished mouse geneticist whose research has focused on cell signaling pathways in development. He received his graduate training in France, did postdoctoral work in Germany and at the Whitehead Institute with Rudolf Jaenisch, and has assumed faculty positions at Baylor College of Medicine, the Fred Hutchinson Cancer Research Center and Mt. Sinai. He was a pioneer in using targeted gene knock-outs to study the gene function. His more recent research focuses on the study of signaling specificity downstream of PDGFs and FGFs in the embryo, particularly as it relates to craniofacial development.

Fgf signaling pathways in mouse development

FGF signaling governs multiple processes important in development and disease. Many lines of evidence have implicated Erk1/2 as the predominant effector pathway downstream of Fgfrs, but these receptors can also signal through other mechanisms. To better understand the function of Erk1/2-independent signaling downstream of Fgfrs in the mouse, we have engineered allelic series of knock-in point mutations designed to disrupt Fgfr1 and Fgfr2 signaling functions individually and in combination. Multiple developmental contexts were affected including preimplantation, posterior outgrowth, limb patterning, skeletal development and craniofacial morphogenesis. Analysis of signaling mutants indicates that Frs2 binding to Fgfr1 and subsequent Erk1/2 engagement has the most pleiotropic functions in development, but that Crk proteins and Plcγ also contribute to Erk1/2 activation, providing a biochemical mechanism for additive signaling requirements. Frs2 engagement is surprisingly dispensable for Fgfr2 signaling. Genetic and biochemical evidence indicates that both receptors utilizes multiple pathways additively *in vivo*, and that the kinetics of signaling differ according to the cell type.

CHUNG, Min Suk PhD
Professor, Department of Anatomy, School of Medicine,
Ajou University, Korea

Min Suk CHUNG is a professor in Department of Anatomy, Ajou University School of Medicine, Suwon, South Korea. He received his BS, MS, and PhD degrees from Yonsei University. For his master's and doctoral theses, he studied clinical anatomy by dissecting cadavers. After PhD acquisition, he became interested in virtual dissection; so he has produced the sectioned images and stereoscopic models of cadavers (Visible Korean project). His new job was to draw the learning comics that deal with systemic anatomy and the comic strips that depict mnemonics and humors of anatomy. Including the comics and other schematics, he wrote an anatomy textbook titled "Memory booster of Regional Anatomy". All educational materials can be downloaded from the homepage (anatomy.co.kr) free of charge.

Free regional anatomy textbook, full of schematics and mnemonics

In order for the medical students to learn anatomy with comfort, a cheerful textbook dealing with concise contents is needed. So the authors have elaborated a regional anatomy book fitting for the purpose. Only anatomical facts essential for cadaver dissection were included. Not only the simplified figures but also the comics depicting mnemonics and humors of anatomy were contained. The electronic book (a PDF file) titled "Memory booster of Regional Anatomy" could be downloaded from the homepage (anatomy.co.kr) without payment or registration.

The creative work was utilized as the textbook in a medical school that the authors belong to; then its educational effect was evaluated. As a result, correlation between the reading times of book and the grades of written examinations (even the grades of lab examinations) was approved. The additional feedback from the students was relatively positive; but they also reported weaknesses of the book. The assessment of learning utility is being done in more medical schools in Korea.

Hopefully, the presented book would function as a pleasant resource to help the medical students learn anatomy efficiently. Simultaneously, the book would inspire other anatomists to produce their own books.

DRAKE, Richard BSc, PhD
Professor, Cleveland Clinic Lerner College of Medicine, USA

Richard L. Drake, PhD, FAAA, Director of Anatomy and Professor of Surgery, Cleveland Clinic Lerner College of Medicine of CWRU, is known as a teacher, educational researcher, author and leader. A master teacher, he has awards from students, institutions, and the American Association of Anatomists (AAA) receiving the 2011 Henry Gray/Elsevier Distinguished Educator Award. An accomplished researcher, he gives talks on educational topics at national and international meetings, and has published numerous peer-reviewed papers and book chapters. He was appointed a fellow of the AAA in 2009 (FAAA). He is one of the authors of *Gray's Anatomy for Students*, *Gray's Basic Anatomy*, *Gray's Atlas of Anatomy* and *Gray's Anatomy for Students Flash Cards*. His current leadership roles include Treasurer of the International Federation of Associations of Anatomists and Co-Editor-in-Chief of *Anatomical Sciences Education*, the education journal of the AAA. Previous leadership activities include past Secretary/Treasurer of the American Association of Anatomists (11 years) and past President of the Association of Anatomy, Cell Biology and Neurobiology Chairpersons.

Today's Anatomy Educator

Adaptability is an excellent personality trait for today's anatomy educator. Emphasis is shifting away from traditional approaches (lectures and dissection) to more current pedagogy such as active learning, increased integration, contextual learning, team-based and interprofessional approaches, and longitudinal formats. Best advice in the current environment – be nimble and forward thinking.

DAS, Srijit MBBS, MD
Professor, Faculty of Medicine,
Universiti Kebangsaan Malaysia Medical Centre, Kuala Lumpur, Malaysia

Prof. Dr. Srijit Das is working in the department of Anatomy, Faculty of Medicine, Universiti Kebangsaan Malaysia since 2007. Prof. Das is actively involved in teaching and research. To date, he has published more than 400 papers in Scopus indexed journals and his 'H index' is 15. His research areas include herbal medicine, antioxidants, atherosclerosis, clinical anatomy, surgical anatomy, psychiatric disorders and molecular biology. He has been declared as the most prolific writer in the field of medicine in Malaysia by Scopus, Elsevier publishers. He is the Editor-in-Chief of Medicine & Health (Malaysia); External Editor of McGill Journal of Medicine (Canada); Section Editor for Anatomy & Cell Biology (South Korea); Associate Editor for Head & Neck section for Surgical and Radiologic Anatomy (Springer); Section Editor for Gross and Imaging Anatomy for Journal of Anatomical Society of India (Elsevier) and Associate Editor for Revista Argentina de Anatomia Clinica. He is also the editorial board member for several international peer reviewed medical journals. He has a passion to write and train youngsters to excel the art of research article writing. Prof. Das has been an Associate for Faculty of Surgical trainers as well as an examiner for MRCS exam conducted by Royal College of Surgeons, Edinburgh.

Gross anatomy - the foundation for producing good medical doctors

Gross anatomy is essential for any medical student in the initial year of entry into any medical course. Gross anatomy is best learnt through cadaveric teaching. Although, several new information technology tools, innovations and methods have emerged, there is no other method which can replace gross anatomy teaching. Good practices include spending of more hours in the practical class with prosected or dissected specimens. Exposure to cadavers improves the knowledge and skill of any students which may prove to be beneficial in treating patients at a later stage. Unfortunately, a change in the curriculum at many places allows lesser number of hours to be spent for Anatomy practical teaching. Integrated curriculum in various institutions has made anatomy more interesting as the students relate the anatomical knowledge with clinical importance in the initial year of their course. Practical sessions supplemented with demonstrations, quizzes, seminars, small group discussions, tutorials, critical appraisal and short term research may also help in producing better skilled doctors. One of the best methods for teaching gross anatomy is having interactive sessions where both the students and teachers interact. There is also a need for continuous assessment of both teachers and students for better learning output. The incorporated research in regular teaching also makes the subject of gross anatomy more interesting. To sum up, gross anatomy is the backbone for effective anatomy teaching and learning.

MAHAKKANUKRAUH, Pasuk MD
Professor, Department of Anatomy, Faculty of Medicine,
Chiang Mai University, Thailand

Dr. Pasuk Mahakkanukrauh was a professor in Anatomy at Department of Anatomy, Faculty of Medicine, Chiang Mai University and the present position is a professor and director of the Excellence center of osteology research and training and the vice director of Cadaveric surgical and training center, Faculty of Medicine, Chiang Mai University. In 2013, she received the Outstanding Preclinical Teacher Award in Faculty of Medicine and National Excellent Teacher Award (Council of the University Faculty Senates of Thailand).

Good Practices in Teaching Gross Anatomy

As Hippocrates used to say 500 years before Christ that the natural form of the human body is the beginning of the medical science. So good practices in gross anatomy is cadaveric teaching related with clinical problem.

As for the clinical anatomy we use in our medical school in Chiang Mai: we have evolved from a whole year dissection of the whole body in the beginning to a block system in which the medical students are exposed to periodical dissection and alternating clinical anatomy lectures selectively and to a day of on hand practice.

For residency training: we provide fresh cadaver for surgical, orthopedic, emergency-room, and ear-nose-throat residences to practice appropriate skills to ensure the safety of their patients. They participated enthusiastically for their own sake. Furthermore along their practicing life they can return for more training when encountering unfamiliar new problem. As for the undergrads clinical problem will always be provided in lectures. Embalmed bodies will further heighten their interest in anatomy in the laboratory session. When they are in their fifth year level they will practice on performing cut-down, tracheostomy, intercostal drainage and subcutaneous nodule excision in fresh cadaver.

These are practices I have used these days in teaching gross anatomy.

HUI, Chi-chung PhD
Professor, Program in Developmental & Stem Cell Biology,
The Hospital for Sick Children, Toronto, Canada

Dr. Hui is Senior Scientist in the Program in Developmental & Stem Cell Biology at the Hospital for Sick Children and Professor of Molecular Genetics at the University of Toronto. He did his undergraduate and Masters studies at the University of Hong Kong. After doctoral and postdoctoral studies at the National Institute for Basic Biology, Okazaki in Japan and postdoctoral training at the Samuel Lunenfeld Research Institute in Toronto, he joined the Hospital for Sick Children Research Institute in 1994. Dr. Hui is an internationally recognized leader in mouse genetics and well known for his pioneering work on Hedgehog signalling and Iroquois homeobox genes. His group has developed various mouse models for studying cancer, congenital malformation syndrome, heart disease, and metabolic dysfunction. He has published more than 150 scientific papers, reviews, and book chapters including many in leading journals such as Cell, Developmental Cell, and Nature. He is currently a Visiting Research Professor in the School of Biomedical Sciences at the University of Hong Kong, a Mentor in the Shanghai Institute of Biochemistry and Cell Biology of the Chinese Academy of Sciences, and an Adjunct Professor in Zhejiang University School of Medicine.

Transcriptional and non-transcriptional functions of Irx homeodomain proteins

Iroquois homeobox (*Irx*) genes are known to act as regulators of pattern formation and cell differentiation in *Drosophila* and vertebrates. They encode proteins of the TALE subclass of atypical homeodomains that are characterized by an additional three amino acid residues (proline-tyrosine-proline) between helix 1 and helix 2, acting as both transcriptional activator and repressor. We have been studying the function of the mouse Irx3 and Irx5 genes in development and disease. In the developing limb, Irx3 and Irx5 control the formation of "anterior" skeletal elements and counterbalance the "posterior" signal Sonic hedgehog (Shh) by directly up-regulating the transcription of *Gli3*, a major repressor of Shh signaling. In this talk, I will present our recent work showing that, apart from their transcriptional role, Irx3 and Irx5 regulate morphogenetic mechanisms that shape the limb bud primordium in a non-transcriptional fashion. We discovered that Irx3 and Irx5 interact with the Cohesin subunit Smc1 and Cux1, which are known to regulate sister chromatid segregation, and that cells lacking Irx3 and Irx5 exhibit abnormal segregation of sister chromatids during cell division. Together, these results suggest that Irx3 and Irx5 program embryonic limb development through both transcriptional and non-transcriptional mechanisms.

TOPP, Kimberly PT, PhD, FAAA
President of American Association of Anatomists
Professor, Dept of Anatomy, University of California, San Francisco, USA

Dr. Kimberly Topp is Professor and Chair of the Department of Physical Therapy and Rehabilitation Science and Professor in the Department of Anatomy at the University of California San Francisco. She completed her PhD in Anatomy and Cell Biology at the University of California Davis and postdoctoral training in Neurobiology at UCSF. She oversees the anatomy curriculum for the doctor of medicine with 650 students, and the full curriculum for the doctor of physical therapy with 150 students. The Department of Physical Therapy includes the PhD in Rehabilitation Science, and outpatient physical therapy practices with 27 physical therapists. Dr. Topp's research interests are in neurobiology, and she has studied biomechanical properties of peripheral nerve and neuropathic pain. She is collaborating with scientists at UCSF to quantify the effects of chemotherapeutic drugs on the sensory and motor systems of patients with cancer. Dr. Topp has been the recipient of the Kaiser Award for Excellence in Teaching and the Sexton Sutherland Endowed Chair in Human Anatomy. She is a member of UCSF Academy of Medical Educators, a Fellow of American Association of Anatomists, and an honorary Fellow of The Anatomical Society. Dr. Topp is the current President of the American Association of Anatomists.

Our Responsibility in Building a Future for Anatomists

The rapid pace of scientific discovery, advances in health care and globalization of knowledge have resulted in consolidation of academic departments, development of entirely new fields of scientific inquiry and drastic change in health professions education. Where are anatomists in this dynamic environment? Are we evolving rapidly enough to keep pace with these changes, let alone *lead* change? Are we able to maintain our identity as anatomists while embedded in a multi-disciplinary research center or a preclinical education group? I will argue that we must step up our pace. We must hone our skills in team science, bring our knowledge of the morphological sciences into advanced clinical investigation and practice, develop the databases needed for precision health care, and collaborate with administration in rapid-pace curricular advances. Of paramount importance, we must ourselves embrace change, and instill in our trainees the skills and desire to negotiate for and lead change. We ourselves were empowered in our careers by pioneers in anatomy and dedicated scientist educators who laid a solid foundation for our investigations and curricular developments. It is our collective responsibility to prepare a bright future for anatomists now entering our treasured field.

Programme in details

Conference – Day 2
December 6, 2016 (Tuesday)

08:30 – 14:00	**Registration**

Time	Programme
	Lecture Theatre 4
Plenary Lecture 3	
09:00 – 09:45	**Chairperson: TSAO, George Sai Wah** (The University of Hong Kong, Hong Kong) **Transcriptional memory from cells-of-origin bypasses the requirement of β-catenin mediated self-renewal in cancer stem cells** **SO, Eric CW** (King's College London, United Kingdom)
09:45 – 10:30	**Chairperson: CHAN, Lap Ki** (The University of Hong Kong, Hong Kong) **The silent mentor program of Tzu Chi University: body donors are altruistic mentors of the students** **TSENG, Guo-Fang** (Tzu Chi University, Taiwan)
10:30 – 11:00	**Coffee Break / Poster Session**

Time	Programme	
	Lecture Theatre 3	**Lecture Theatre 4**
	SYM 3.1 **Advances in Cancer Research** **Chairperson:** **BAY, Boon Huat** (National University of Singapore, Singapore) **CHAN, Leung Franky** (The Chinese University of Hong Kong, Hong Kong)	**SYM 3.2** **Body Donation** **Chairperson:** **Chongthammakun, Sukumal** (Mahidol University, Thailand) **TSENG, Guo-Fang** (Tzu Chi University, Taiwan)
11:00 – 11:18	**SVCT-2, an indicator biomarker for treatment of breast cancer by L-ascorbate** **LEE, Wang Jae** (Seoul National University College of Medicine, Korea)	**Body Donation in Thailand** **CHOMPOOPONG, Supin** (Mahidol University, Thailand)
11:18 – 11:36	**Exploiting 'stemness' as a cancer cell vulnerability** **MA, Stephanie Kwai Yee** (The University of Hong Kong, Hong Kong)	**Body Donation in Southern Medical University** **OUYANG, Jun** (Southern Medical University, China)
11:36 – 11:54	**Understanding RNA Editing in Gastrointestinal Cancer: Causes and Functional consequences** **CHEN, Polly Leilei** (National University of Singapore, Singapore)	**Body Donation in Hong Kong** **CHAN, Lap Ki** (The University of Hong Kong, Hong Kong)
11:54 – 12:12	**Phenotypic spectrum of *PIK3R1* mutations in cancers** **CHEUNG, Lydia Wai Ting** (The University of Hong Kong, Hong Kong)	**Silent Mentors Program, Singapore - Our Journey** **NG, Yee Kong** (National University of Singapore, Singapore)
12:12 – 12:30	**Complement component 1, q subcomponent binding protein (C1QBP), an interacting partner of the Y-Box Binding Protein-1 (YB-1) Promotes Breast Cancer Progression** **BAY, Boon Huat** (National University of Singapore, Singapore)	**History of Body Donation for Medical Education in Korea** **LEE, Wang Jae** (Seoul National University College of Medicine, Korea)
12:30 – 13:45	**Lunch Break / Poster Session**	

Conference – Day 2

December 6, 2016 (Tuesday)

Time	Programme	Time	Programme
Lecture Theatre 3		**Lecture Theatre 4**	
SYM 4.1: **Advances in Aging Studies** **Chairperson:** **ZHOU, Zhongjun** (The University of Hong Kong, Hong Kong) **HUEN, Michael Shing Yan** (The University of Hong Kong, Hong Kong)		**SYM 4.2:** **Innovations in teaching Gross Anatomy** **Chairperson:** **KARTHIK, Harve Subramhanya** (National University of Singapore, Singapore) **ZHANG, Shaoxiang** (Third Military Medical University, China)	
13:45 – 14:15	**Guardians of Healthy Aging: Telomere Protection in Mammalian Cells** **SONGYANG, Zhou** (Sun Yat-sen University, China)	**13:45 – 14:03**	**Gross Anatomy Teaching in Southern Medical University** **OUYANG, Jun** (Southern Medical Unversity, China)
14:15 – 14:25	**Aging: Matrix Matters** **ZHOU, Zhongjun** (The University of Hong Kong, Hong Kong)	**14:03 – 14:21**	**Collaborative Ultrasound Objective Structural Practical Examination (OSPE) as a part of practical assessment in Gross Anatomy course** **PAWLINA, Wojciech** (Mayo Clinic College of Medicine, USA)
14:25 – 14:35	**Electrical stimulation enhances mood and memory functions in animal models** **LIM, Lee Wei** (The University of Hong Kong, Hong Kong)	**14:21 – 14:39**	**Gross Anatomy Education in Practical Anatomy Center, Korea University College of Medicine** **RHYU, Im Joo** (Korea University, Korea)
14:35 – 15:15	**Longevity mechanisms in long-lived mammals** **GORBUNOVA, Vera** (University of Rochester, USA)	**14:39 – 14:57**	**Learning to Contribute in an Inquiry-Based Curriculum** **TOPP, Kimberly** (University of California, USA)
		14:57 – 15:15	**Changing from passive to active learning: making dissection clinically relevant** **CHAN, Lap Ki** (The University of Hong Kong, Hong Kong)
15:15 – 15:45	**Coffee Break / Poster Session**		
Lecture Theatre 4			
Plenary Lecture 4			
15:45 – 16:30	**Chairperson: HERRUP, Karl** (The Hong Kong University of Science and Technology) **Imaging synapses in vivo – physiology and pathology of synapses and neural circuits** **OKABE, Shigeo** (The University of Tokyo, Japan)		
16:30 – 17:15	**Chairperson: CHAN, Lap Ki** (The University of Hong Kong, Hong Kong) **Innovations in anatomy teaching – a journey over 30 years through body painting, 3D printing and virtual reality** **MCMENAMIN, Paul G** (Monash University, Australia)		
17:15 – 17:30	**Closing Ceremony / Poster Awards**		

SO, Eric Chi Wai PhD
Professor, Department of Haematological Medicine,
King's College London, United Kingdom

Professor Eric So obtained his BSc degree (1st honour) and PhD (Gold medal award) from the University of Hong Kong before moved to Stanford University in 2000 for his postdoc with Professors Michael Cleary on MLL leukaemia. In 2004, he joined the Institute of Cancer Research in London to setup his research group studying transcriptional and epigenetic deregulation in acute leukaemia. In 2009, Professor So joined King's College London as the chair professor in leukaemia and stem cell biology. Over the years, Professor So has made many seminal discoveries and publishes extensively in high impact scientific and medical journals including Cancer Cell, Nature Medicine, Cell Stem Cell, and Nature Cell Biology. He has won many international personal awards including the Croucher Foundation (HK) Scholarship and Fellowship Awards, the Leukemia and Lymphoma Society (LLS, US) Special Fellowship Award, the Association for International Cancer Research (AICR, UK) International Fellowship Award, the European Molecular Biology Organization (EMBO) Young Investigator Award, and the Pezcollar Foundation and European Association for Cancer Research (EACR) Cancer Researcher Achievement Award. His current research focus is to identify novel molecular targets in particular those involved in epigenetic regulation and stem cell function for development of effective cancer therapeutics.

Transcriptional memory from cells-of-origin bypasses the requirement of β-catenin mediated self-renewal in cancer stem cells

Self-renewal is a unique and essential property that distinguishes (cancer) stem cells from their downstream progenies. While very little is known about the molecular pathways that govern this critical property, canonical Wnt signaling mediated by β-catenin has emerged as an essential molecule for self-renewal of various normal stem cell as well as cancer stem cells. Intriguingly, β-catenin is largely dispensable for normal hematopoietic stem cells but is required for leukemic stem cells. This suggests a different molecular network regulated by β-catenin in hematopoietic stem cells, and more importantly highlights a therapeutic opportunity to target β-catenin for eradication of leukemic stem cells. However, the majority of the studies and clinical treatments mostly only focus on cancer genetics without considering the epigenetic or transcriptional memory from their cells-of-origin. It is possible that phenotypically and genetic indistinguishable cancers driven by same oncogenic events but derived from different cellular origins can utilize alternative pathways to overcome highly specific personalized medicine targeting particular essential self-renewal mechanisms. To address this question, we will examine how cells-of-origin can determine the biology of cancer stem cells, and discuss a novel functional crosstalk between key self-renewal pathways mediated β-catenin and Hox in leukemic stem cells, and their implication in designing effective leukemia treatment. If time allows, I will also reveal our recent progress in identifying the potential origin of human AML stem cells, and their impacts to our current understanding of human cancer.

TSENG, Guo-Fang
National Taiwan Normal University, BS
National Taiwan University, MS
University of Wisconsin-Madison, PhD
Stanford University Medical Center, Postdoc
Professor, Department of Anatomy, College of Medicine,
Tzu Chi University, Taiwan

Dr. Tseng is a professor of Anatomy of the Tzu Chi University in Hualien, Taiwan. He started training as an anatomist in 1977 in the National Taiwan U (MS) and later at the University of Wisconsin-Madison (PhD). He then received training in neurology at Stanford Medical Center (Postdoc). He was formerly a professor and head of the Anatomy of National Taiwan University and received numerous teaching awards.

He joined Tzu Chi University's anatomy teaching in 1997 and leads a unique humanistic-based Silent Mentor Program. The program has won praises all over the world and received front page coverage in the Wall Street Journal in 2009, 04, 22 by Ian Johnson, a Pulitzer journalist. He sets up and directs the Medical Simulation Center of the Tzu Chi University that uses un-embalmed donated bodies to train medical students and surgeons on invasive clinical skills and surgeries. Currently, he is also the vice president and dean of the Research and Development of the University.

The silent mentor program of Tzu Chi University: body donors are altruistic mentors of the students

We taught gross anatomy in a humanistic and interaction-based setting. Donors were silent mentors for the selfless offering. Students visited the surviving family and learned about the silent mentors' lives and the donation before the class. Brief ceremonies attended by families, students and volunteers, were held at the beginning and end of the class. At the start of the class, students also shared their views of the silent mentors with surviving families and classmates. Interaction bound students with families and help the students to better appreciate the body donation.

The curriculum inspired students to deliberate over the selfless act of donors and families, and brought up their empathy toward others and future patients. Students appreciated the program and were self-driven to study harder. The program was highly esteemed by donors and families and overcame the reluctance to body donation in Taiwan. The university received abundant donations and pledges to donate in the future. The program was adopted in various way medical schools in Taiwan and Asia as well. In short, by integrating a "gratitude, respect and love" philosophy to gross anatomy courses, anatomy teaching can in addition help to cultivate empathy in medical students. This brought anatomy teaching to a new level of significance in the training of medical professionals.

LEE, Wang Jae MD
President of Korean Association of Anatomists
Professor, Department of Anatomy,
Seoul National University College of Medicine, Korea

He is a current president of Korean Association of Anatomists. His research major is the tumor immunology and he has a vivid academic interest in cancer treatment using high dose of vitamin C. Actually he has published more than 40 papers on anti-tumor effect of vitamin C. He was also a former president of Korean Association of Immunologists (KAI) and an Editor-in-Chief of the Immune Network published by KAI. In early 2000, he was a vice chancellor for academic affairs of Seoul National University and also an associate dean for research affairs of Seoul National University, College of Medicine.

SVCT-2, an indicator biomarker for treatment of breast cancer by L-ascorbate

L-ascorbate (vitamin C) clearly has an inhibitory effect on cancer cells. However, the mechanism underlying differential sensitivity of cancer cells from same tissue to L-ascorbate is yet to be clarified. Here, we demonstrate that L-ascorbate has a selective killing effect, which is influenced by sodium-dependent vitamin C transporter 2 (SVCT-2) in human breast cancer cells. Treatment of human breast cancer cells with L-ascorbate differentially induced cell death, dependent on the SVCT-2 protein level. Moreover, knockdown of endogenous SVCT-2 via RNA interference in breast cancer cells expressing high levels of the protein induced resistance to L-ascorbate treatment, whereas transfection with SVCT-2 expression plasmids led to enhanced L-ascorbate chemosensitivity. Surprisingly, tumor regression by L-ascorbate administration in mice bearing tumor cell xenograft also corresponded to the SVCT-2 protein level. Interestingly, SVCT-2 expression was absent or weak in normal tissues, but strongly detected in tumor samples obtained from breast cancer patients. In addition, enhanced chemosensitivity to L-ascorbate occurred as a result of caspase-independent autophagy, which was mediated by beclin-1 and LC3 II. In addition, treatment with NAC, a Reactive Oxygen Species (ROS) scavenger, suppressed the induction of beclin-1 and LC3 II, implying that the differential SVCT-2 protein-dependent L-ascorbate uptake was attributable to intracellular ROS induced by L-ascorbate, subsequently leading to autophagy. These results suggest that functional SVCT-2 sensitizes breast cancer cells to autophagic damage by increasing the L-ascorbate concentration and intracellular ROS production. Conclusively, SVCT-2 in breast cancer may act as an indicator for commencing L-ascorbate treatment.

MA, Stephanie Kwai Yee PhD
Assistant Professor, School of Biomedical Sciences,
The University of Hong Kong, Hong Kong

Dr. Ma obtained her Bachelor's and Master's degrees from the University of British Columbia in Vancouver, Canada, and her PhD from the University of Hong Kong. She is currently an Assistant Professor in the School of Biomedical Sciences and a Principal Investigator at the State Key Laboratory for Liver Research in the LKS Faculty of Medicine at the University of Hong Kong. Dr. Ma's research interest is on exploiting stemness as a cancer cell vulnerability. She is the recipient of numerous honors and awards including the 2006-2007 Li Ka Shing Prize for the Best PhD Thesis (The University of Hong Kong), the 2008 Hong Kong Young Scientist Award in Life Sciences (Hong Kong Institution of Science), the 2011 Faculty Outstanding Research Output Award (The University of Hong Kong), the 2012-2013 Outstanding Young Researcher Award (The University of Hong Kong), the 2014 Croucher Innovation Award (Croucher Foundation) as well as the 2014 Higher Education Institution of China – Scientific Research Outstanding Achievement Award (Second-class Award in Science and Technology Section). She is listed as the top 1% most cited scholars in 'Clinical Medicine' and 'All Fields' ranked by the ISI's Essential Science Indicators. Her original research findings have been published in leading international journals including Cell Stem Cell, Gastroenterology, Hepatology, Cancer Research, Oncogene and Stem Cell Reports.

Exploiting 'stemness' as a cancer cell vulnerability

Cancer is a major public health problem and remains the leading cause of disease-related deaths in the world. While cancer is a disease commonly associated with adulthood and aging, it can also be viewed as a stem cell and developmental disorder. Our group is interested in understanding the general mechanism of tumorigenesis and developing strategies to target cancer through understanding 'cancer stemness', a property that is closely associated with tumor relapse, drug resistance and the general unfavorable outcome of the disease. We are conducting systematic multidisciplinary studies, involving omics profiling strategies coupled with molecular, cellular, functional and disease modeling approaches, to identify and characterize functional genes and pathways that are operational for cancer stemness. Gastrointestinal tumor types particularly prevalent in Southeast Asia and Hong Kong, including hepatocellular carcinoma and esophageal squamous cell carcinoma are currently used as model systems in our studies. Findings from our work may open up the possibility of personalized intervention strategies specifically targeted at cancer stemness, ultimately leading to the improved quality of life of cancer patients. In this talk, a summary of our recent work will be shared.

CHEN, Polly Leilei MD, PhD
Cancer Science Institute of Singapore, National University of Singapore, Singapore

Dr. Chen Leilei Polly received her PhD degree from the University of Hong Kong (HKU) in 2010. After 2-year postdoctoral training at HKU, she was recruited as a Research Assistant Professor and Special Fellow at the Cancer Science Institute of Singapore (CSI Singapore) where she directs a research lab studying human cancers. Currently she is a Principal Investigator in CSI and a President Assistant Professor in the Department of Anatomy, Yong Loo Lin School of Medicine, National University of Singapore (NUS). Dr. Chen's research has centered on transcriptome instability of human cancers and in particular on understanding how RNA editing contributes to cancer initiation and progression. Her scientific achievements include being the first group to describe RNA editome imbalance in human gastrointestinal (GI) cancer and decipher several "driver" RNA editing events in GI cancer.

Understanding RNA Editing in Gastrointestinal Cancer: Causes and Functional consequences

Gastrointestinal (GI) cancer is responsible for more cancer-related deaths than any other types of cancers. Conventionally, all cancers arise from an acquired genomic hit/s (mutation or genomic abnormality) in "driver" genes, resulting in malignant transformation. RNA editing, one of co- or post-transcriptional modifications, introduces changes in the RNA sequences encoded by the genome, which refutes the Central Dogma that DNA dictates each nucleotide in RNA. RNA editing may lead to tumor-specific "editing/epigenetic mutations (epimutations)". In humans, the most frequent type of editing is the conversion of adenosine to inosine (A-to-I), which is catalyzed by RNA editing enzymes, the double-stranded RNA (dsRNA)-specific ADAR (Adenosine DeAminase that act on RNA) family of protein, ADAR1 and ADAR2. Knowledge gleaned from our recent research has highlighted a link between RNA editing dysregulation and GI cancer initiation and progression, raising the complexity of cancer to a new level once again. Uncovering new functional "epimutations" in tumors may contribute to personalized medicine by better classifying and diagnosing cancers and potentially reveal new targets for therapeutics.

CHEUNG, Lydia Wai Ting PhD
Assistant Professor, School of Biomedical Sciences,
The University of Hong Kong, Hong Kong

Dr. Cheung received her BSc and PhD at The University of Hong Kong. She obtained further training in cancer genomics research as post-doctoral fellow in the MD Anderson Cancer Center (Houston, US) and was then promoted to instructor in the same Institute. She is now an assistant professor in the School of Biomedical Sciences at the LKS Faculty of Medicine, HKU. The focus of her research is functional characterization of genomics abnormalities in cancers, with a goal of ultimately devising the optimal therapeutic strategies for cancer patients. She is the first to comprehensively characterize PI3K pathway aberrations in endometrial cancer and the first to report high-throughput functional genomics analysis of an epithelial cancer. Her recent publications demonstrate that mutations in cancer patients can be neomorphs (gain of novel function) that activate unexpected pathways and are functionally distinct from the wild-type gene. This revolutionizes the concepts underlying precision cancer therapy where the impact of each specific mutation in a cancer gene has to be considered rather than the cancer gene alone. Dr. Cheung has also received multiple scholarships, grants and awards in both Hong Kong and US.

Phenotypic spectrum of *PIK3R1* mutations in cancers

The canonical action of the p85α regulatory subunit of phosphatidylinositol 3-kinase is to inhibit the activity but also the degradation of the p110 catalytic subunit by forming heterodimer with p110. *PIK3R1* (encodes p85α) is the 12th most frequently mutated gene across tumors sequenced by The Cancer Genome Atlas. Functional characterization of some of these *PIK3R1* mutations revealed loss-of-function mutants that are unable to inhibit p110α catalytic activity. Recently, we have also developed a molecular model of how p85α homodimer stabilizes PTEN protein. We demonstrated that some *PIK3R1* mutants disrupt the formation of p85α homodimer, leading to PTEN destabilization and activation of PI3K pathway. Further, I will give example of neomorphic mutants that induce signaling pathways not predictable by the canonical functions of p85α. These different oncogenic mechanisms of mutations suggest the need to functionally characterize specific mutations in cancer genes for effective genome-informed precision medicine.

BAY, Boon Huat MBBS, PhD
Professor, Department of Anatomy, Yong Loo Lin School of Medicine,
National University of Singapore, Singapore

Dr. Boon Huat Bay's research interest is in Cancer Biology, in particular the utility of biological markers of malignancy and molecular-targeted cancer therapy. He also has an interest in nanotoxicology, especially, investigating the cytotoxic and genotoxic effects of gold, silver and zinc oxide nanoparticles, using various microscopy and technological platforms in different model systems. A medical graduate, he has taught gross anatomy to medical, dental, pharmacy, nursing and life science students since 1990. In terms of administrative contributions, he was Assistant Dean (Graduate Studies) from 2003-2008 and Head of Anatomy from 2008-March 2016.

Complement component 1, q subcomponent binding protein (C1QBP), an interacting partner of the Y-Box Binding Protein-1 (YB-1) Promotes Breast Cancer Progression

C1QBP is known to play important roles in cancer progression, including proliferation and metastasis. C1QBP has also been previously reported to interact with Y-Box-Binding Protein 1 (YB-1), a multifunctional protein which affects many fundamental cellular processes. In this study, we evaluated the interactome of C1QBP by quantitative SILAC proteomic analysis and verified that YB-1 is an interacting partner of C1QBP. This was further confirmed by co-immunoprecipation experiments. We further show a positive correlation of YB-1 mRNA and C1QBP mRNA in a panel of breast cancer tissues by real time PCR and protein expression in clinical breast cancer samples by immunohistochemical staining. C1QBP and YB-1 gene and protein expression (by Western blot) were also observed to correlate well in breast cancer cell lines. Co-localization of both proteins was demonstrated by confocal immunofluorescence microscopy. Knockdown of C1QBP inhibited cell proliferation, migration and invasion in MDA-MB-231 breast cancer cells in vitro. However, double knockdown of C1QBP and YB-1 did not show a synergistic decrease for cell proliferation, migration and invasion. When C1QBP was diminished in YB-1-overexpressing cells, there was a decrease in cell proliferation, migration and invasion, but it did not return to the basal levels. Taken together, the interaction of C1QBP with YB-1 alone was not sufficient to induce cell proliferation, migration or invasion, implying that perhaps there are other factors which may also influence these functions.

CHOMPOOPONG, Supin BSc, MSc, PhD
President of The Anatomy Association of Thailand
Associate Professor, Faculty of Medicine Siriraj Hospital, Mahidol University, Thailand

Supin Chompoopong (Menayotin) is the present president of the Anatomy Association of Thailand (AAT). She received her PhD in 1988 through the Mahidol University's Anatomy Program (Neuroscience).

From 1998 to present she has worked as Associate Professor in Department of Anatomy, Faculty of Medicine Siriraj Hospital, Mahidol University.

Her neuroscience research interests are anti-inflamation, anti-apoptotic and anti-oxidant property of herbal medicine, Cerebral Ischemia of Hippocampus and cognitive impairment in rats, Schwann cell culture and Spinal cord injury animal model, Estrogen and Phytoestrogen as Neuroprotection.

Her ongoing researchs is to study the neurological role of Thai medicinal herbs and especially germinated brown rice in cerebral ischemic and Parkinson's like animal models.

Body Donation in Thailand

Body donation for medical studies brings many benefits in terms of education, health care, and morality, which will lead to future social development. Medical studies and researches need to learn from human bodies in order to research new life-saving medical and surgical procedures and techniques. The benefits of studies on donated bodies or cadavers are obvious in many areas such as medical and other health care professionals' education, medical specialists' training, medical research and the anatomy museums. At least two factors that have helped Thai people make a decision in the body donation. One is that King Bhumibol has officially approved body donation a strong incentive in Thai society and students organized the Royal Cremation Event to give the privilege honesty back to donors' family. The second factor is that donors attain the highly regarded status of *ajarn-yai*, great teacher. Teachers in Thailand are respected to an extent unfamiliar to Westerners. This respect is formalized in a ceremony called *wai-khru* (honour the teacher), which takes place annually in Thai schools and universities. At Siriraj Hospital, Mahidol University over the past seven years (in 2009-2015), the average number of donation is 7192.29±1411.29 donors/year (33.61%±1.26% for male, 66.39%±1.26% for female). The average number of donated body gathered to Anatomy department is 323±50.41 bodies/year (58.39%±4.73% for male, 41.60%±4.73% for female). Therefore, most of Medical school in Thailand still has no problem in number of the donated bodies and expresses the deepest gratitude and appreciation for the spiritual intention of those who donate their bodies for medical study.

Keywords: Body donation, Cadaver, Anatomy

OUYANG, Jun MD
Professor, Southern Medical University, China

Jun Ouyang, MD, is director of Department of Anatomy & Guangdong Provincial Medical Biomechanical Key Laboratory, Southern Medical University, Guangzhou, China.

Professor Ouyang obtained his bachelor's degree in clinical medicine from the First Military Medical University in 1990 then subsequently earned his MD at the same university in 1996. He had his post-doctor fellow training in West Virginia University, USA from 2000 to 2002.

As chief editor of *Chinese Journal of Clinical Anatomy*, Prof. Ouyang's research interests focus on musculoskeletal biomechanics. He has authored or co-authored numerous articles that have appeared in peer-reviewed journals, including Spine, Journal of Trauma, Clinical Biomechanics, Clinical Orthopedics and Related Research, Acta of Biomengineering and Biomechanics, Surgical Radiology Anatomy, Journal of Gene Medicine, American Journal of Physiology et al.

He translated some classical works and atlas of anatomy into Chinese, include *Grant's Dissector* (Fifteenth Edition), *Atlas of Anatomy* (Lippincott Williams & Wilkins), *Lippincott's Concise Illustrated Anatomy* (3 Volumes), *Thieme Atlas of Anatomy* (Third Edition).

Body Donation in Southern Medical University

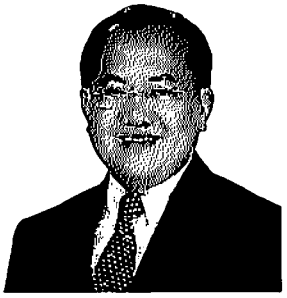
The presentation would give you an introduction on how the body donation works in China and what we have done in Southern Medical University. Body donation should be guided by Red Cross in local province and/or city and abidance by local regulations. As one of four universities that authorized by Guangzhou branch Red Cross Society of China, we started our body donation work since 2001. In 2013, Southern Medical University became the only one authorized university for body donation by Guangdong Provincial Red Cross Society. Southern Medical University designed a website (http://web3.fimmu.com/ytjz/) to promote body donation and memorize those who donated their body. We also built the community of body donation promotion among undergraduate students. The community visited the registered body donation people regularly, and held a "Twinkling Stars" commemoration in campus annually. The community also go into the street to promote body donation during the weekend or special days, such as, World Red Cross Day.

CHAN, Lap Ki
BSc, MBBS, FHKCOS, FHKAM, FRCS Edinburgh, PhD, MEd
Associate Professor, School of Biomedical Sciences,
The University of Hong Kong, Hong Kong

Dr. Chan is an anatomist and medical educator, with a background in orthopedics and physical anthropology. He is currently an associate professor in the School of Biomedical Sciences, and the deputy director of the Bau Institute of Medical and Health Sciences Education, at the Li Ka Shing Faculty of Medicine at The University of Hong Kong. He has a background in orthopedics and physical anthropology and teaches gross anatomy to medical students. His research interests include innovative pedagogies in anatomy education, problem-based learning (PBL), team-based learning (TBL), interprofessional education (IPE), and faculty development. He is the director of the Staff and Professional Development Program. He is an educator for the Asia Pacific region for the AO Foundation (Arbeitsgemeinschaft für Osteosynthesefragen). His teaching excellence has been recognized by such awards as the Thomas Henry Huxley Instructorship from Duke University, and most recently, an Outstanding Teaching Award from The University of Hong Kong. He serves as an associate editor for Anatomical Sciences Education and has edited, with Professor Wojciech Pawlina, "Teaching Anatomy – A Practical Guide", published by Springer.

Body Donation in Hong Kong

Learning gross anatomy through the dissection of cadavers is the traditional foundation of medical education. Dissection is considered by many anatomists to be the most powerful way of presenting and learning anatomy. In the early years of the Li Ka Shing Faculty of Medicine of The University of Hong Kong (HKU), most of the cadavers for dissection were unclaimed bodies from hospitals. However, in recent years, the number of unclaimed bodies has been steadily declining. The need for cadavers has stimulated the HKU Body Donation Programme, which has a history of 40 years, to launch a series of activities to inform the public of the body donation programme as a way to contribute to the society, even after their death. But there were concerns over whether body donation can be accepted by te Hong Kong residents because of their cultural background. Students conducted interviews with registered body donors and their family members, in order to identify concerns specific to their cultural, social and religious backgrounds. A population-wide survey was also conducted in 2014. It was found that a very high percentage (84%) of the adult residents of Hong Kong have heard of body donation and many of them (51%) were willing to donate their body after death. After years of promotion, the impact of the programme's widely reported activities is demonstrated by the dramatic increase in the number of registered and actual donors.



NG, Yee Kong BDS, PhD
Associate Professor, Department of Anatomy,
National University of Singapore, Singapore

Yee-Kong NG was trained as a dentist, but made a big switch in his early professional career to teaching and research in anatomy. His research interest was in neuroscience particularly in degeneration and stroke. Lately, he spends more time in teaching and is heavily involved in the current medical curriculum rationalization. He is the Yong Loo Lin School of Medicine's Phase 1 director. His teaching excellence has been recognized by numerous education awards presented by the Dental, Medical and Science Faculties, as well as the University Teaching Excellence Award. In recent years, he championed and promoted Silent Mentors, Body Donation Program for the school as well as Singapore.

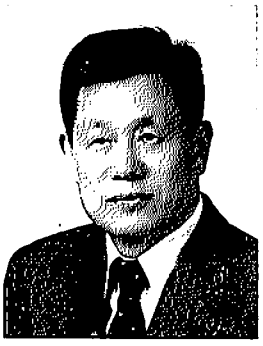
Silent Mentors Program, Singapore - Our Journey

It has been 7 years since we made the 1st initiative in looking at the possibility of getting cadavers mainly from the willed body programme for the teaching of human anatomy. It all started from the trip we made to Tzu Chi University (TCU) at Hualien, Taiwan in 2009. To better understand the whole body donation programme for medical education and research, we had also made several other visits to institutions in Australia, China, Germany, Thailand, UK and USA. We had learned and benefitted much from these visits, and adopted some of these best practices upon returns.

We had also adapted the term "Silent Mentors", which was used and popularised by TCU to our body donation program. Among some other things that we do to promote body donation was the emphasis of responsibility in handling and managing the bodies and their parts. We also stress the importance of gratitude and respect towards donors and their families for their selfless gifts and sacrifices. At the beginning of our human anatomy courses, an introductory lecture on silent mentors and values that we emphasized above was highlighted. We concluded our anatomy courses with appreciation ceremonies organised separately by our medical and dental students.

We had since made some but significant progress in bringing awareness as well as breaking the steep eastern cultural hindrances of whole body donation. In this seminar, we would like to share and elaborate on our humble experience of the journey.

Yee-Kong NG, Harve S KARTHIK, Owen OC CHAN, Samuel SW TAY, Boon-Huat BAY
Department of Anatomy, Yong Loo Lin School of Medicine, National University of Singapore



LEE, Wang Jae MD
President of Korean Association of Anatomists
Professor, Department of Anatomy,
Seoul National University College of Medicine, Korea

He is a current president of Korean Association of Anatomists. His research major is the tumor immunology and he has a vivid academic interest in cancer treatment using high dose of vitamin C. Actually he has published more than 40 papers on anti-tumor effect of vitamin C. He was also a former president of Korean Association of Immunologists (KAI) and an Editor-in-Chief of the Immune Network published by KAI. In early 2000, he was a vice chancellor for academic affairs of Seoul National University and also an associate dean for research affairs of Seoul National University, College of Medicine.

History of Body Donation for Medical Education in Korea

Importance of dead body in medical education, especially in anatomy education cannot be overemphasized. In Korea, since launching the modern medicine introduced by the medical missionary mainly from United States of America at the end of 19th century, more than 100 years have passed and now great progress in modern medicine has been achieved through modernized medical education, at the center of which anatomy education stands high. In Korea, actually modern medical education has started before or after the Korean war occurred in 1950. At the beginning, so many unclaimed dead bodies for medical education have provided by the district offices, where they have been managed officially. However, the number of unclaimed dead bodies has been decreased drastically before or after the Seoul Olympic 1988, so that anatomy education has been fallen in big trouble. Since then, the movement for dead body donation has started from Seoul area, expanded nation-widely thereafter. Unclaimed dead bodies have been no more available for medical education. At the beginning of the movement, there have been very few donated dead bodies so that it has been very difficult to teach the medical students the normal body structures effectively. Now, the dead body donation system has been settled well so that the sufficient dead bodies donated make anatomy education performed successfully in Korea.

SONGYANG, Zhou PhD
Professor and Dean of School of Life Sciences, Sun Yat-sen University, Guangzhou, China

Professional Experience

1995	PhD in Molecular Physiology, Tufts University, USA.
1995-1996	Postdoctoral Fellow, Harvard University, Cambridge, MA.
1996-1998	Postdoctoral Fellow, Massachusetts Institute of Technology, Cambridge, MA
1998-2007	Assistant and Associate Professor, Department of Biochemistry and Molecular Biology, Baylor College of Medicine, Houston, TX
2008-	Professor, Department of Biochemistry and Molecular Biology, Baylor College of Medicine, Houston, TX
2009-	Director, Institute of Healthy Aging Research, School of Life Sciences, SYSU, Guangzhou

Dr. Songyang is interested in the molecular mechanisms that regulate cell survival, genome stability, stem cell pluripotency, and cancer initiation through proteomic and functional genomic approaches. Dr. Songyang's major research areas include telomeres and telomerase, DNA damage repair signaling, and embryonic stem cell self-renewal and differentiation. Dr. Songyang has also developed several technology platforms to study protein-protein interactions and signaling pathways. He has published more than 120 papers in leading journals such as Nature, Science, Cell, Nature Cell Biology, NSMB, Molecular Cell, PNAS, Curr. Biol., and JCB. His publications were cited more than 13,000 times.

Guardians of Healthy Aging: Telomere Protection in Mammalian Cells

Telomeres have been implicated in cancer and aging. In mammalian cells, the telomeres are bound by the core telomeric proteins TRF1, TRF2, RAP1, TIN2, TPP1, and POT1. These six telomeric proteins form large protein complexes and recruit different signaling molecules for telomere length control and end protection. Through proteomic approaches, we systematically identified proteins that associate with telomeres and regulate telomerase. The function of these proteins in telomere maintenance, aging, and cancer will be discussed. Furthermore, defects in telomere protection could lead to premature aging. We report here the generation of human cells that conditionally knockout (KO) each of the six telomeric proteins using the CRISPR-Cas9 system. KO of individual telomeric proteins generated distinct DNA damage responses, metabolic profiles, and telomere length changes. Our results reveal unique features of the human telomeric complexes and indicate that human telomeric proteins differ in telomere maintenance and metabolic control.

ZHOU, Zhongjun PhD
Professor, School of Biomedical Sciences,
The University of Hong Kong, Hong Kong

Zhongjun Zhou is currently a professor at the School of Biomedical Sciences, The University of Hong Kong. He graduated from Xiamen University with a BSc in Biochemistry, PhD in Pathophysiology from Peking Union Medical College and PhD in Medical Biochemistry from Karolinska Institute. He joined the biochemistry department in HKU as a Research Assistant Professor in Dec 2002 and became Associate Professor in 2005.

Prof Zhou is interested in understanding the biological functions of extracellular and nuclear matrix proteins. He works on signaling regulation in development, aging, and tumorigenesis using genetically modified mice and human patient tissues. He is well recognized in the field of biology of aging for his work on genomic instability and chromatin dynamics in progeria (HGPS). He has also made significant contributions to the understanding of membrane-type 1 matrix metalloproteinase (MT1-MMP) function as a signaling regulator in the development of bone, lymphocytes, blood vessels and lymphatic vessels.

He is the recipient of Outstanding Research Award of HKU and Croucher Senior Research Fellowship. He is the founding Chair of Asian Society of Aging Research and chief editor of Translational Medicine of Aging and associate editor of Mutation Research.

Aging: Matrix Matters

Abnormal splicing of *LMNA* gene gives rise to a truncated prelamin A termed as progerin which is accumulated in patients suffering from Hutchinson-Gilford progeria Syndrome. Lamin A interacts with and activates a variety of nuclear factors including histone modifying enzymes such as MOF, SUV39H1, SIRT1 and SIRT6. The presence of progerin compromises the proper association of these important nuclear proteins with nuclear matrix, leading to defective chromatin remodeling in response to DNA damage. The nuclear lamin A also serves as activators for SIRT1 and SIRT6 that are critical in stem cell maintenance and DNA damage repair. Targeting the epigenetic changes significantly rescue the cellular senescence and extend lifespan in progeroid mice. Our studies suggest a profound role for lamin A in regulating nuclear architecture, chromatin dynamics and stem cell potency that all contribute to the aging processes.

Acknowledgements
These works are supported by grants from Research Grant Council (CRF/GRF) of Hong Kong and Natural Science Foundation of China.

LIM, Lee Wei MD, MSc, PhD
Assistant Professor, School of Biomedical Sciences,
The University of Hong Kong, Hong Kong

Dr LIM Lee Wei is currently an Assistant Professor in the Li Ka Shing Faculty of Medicine, the University of Hong Kong. At the same time, he is also an Adjunct Associate Professor in Sunway University and a researcher in Maastricht University, the Netherlands. Dr Lim completed his Master of Affective Neuroscience and PhD studies at Maastricht University; and subsequently, he spent several years of research scholarships including the Marie Curie Fellowship and Kootstra Top-Talent Fellowship in Maastricht University and Oxford University. In 2011, he was awarded the Lee Kuan Yew Research Fellowship as a principal investigator for neuromodulation research in Nanyang Technological University, Singapore.

Dr Lim's research focuses on the basic and translational neuroscience using the deep brain stimulation for neurodegenerative and neuropsychiatric disorders. More specifically, his interests are directed towards understanding the neural basis of mood and anxiety disorders, as well as learning and memory behavior in neurodegenerative disorders. His research plan of investigation consists of different inter-related sections using cellular and molecular techniques, electrophysiology, behavioral and neurochemical methods, which together provide a multidisciplinary approach to understand how electrical stimulation interfaces with the brain networks, by controlling the neuronal activity and cellular microenvironment.

Electrical stimulation enhances mood and memory functions in animal models

Memory dysfunction is the main symptom of dementia-related disorders. In this study, we tested the hypotheses that electrical stimulation of the medial prefrontal cortex (mPFC) enhanced memory- and mood-related behaviors in animal models. Our data demonstrated that stimulation of the mPFC evoked antidepressant effects. Further, we have shown that mPFC stimulation enhanced neurogenesis in the hippocampus, mitigating the deleterious effects of memory dysfunction conditions and improved the learning and memory functions in the Morris water maze and object recognition tests. Our results showed a remarkable increase of neural progenitors, surviving BrdU-positive cells, and dendritic arborization after chronic mPFC stimulation as compared to the sham. To support these findings, immunofluorescence studies revealed co-localization of c-Fos immediate-early gene activation with the doublecortin and the BrdU-labeled cells in the hippocampal dentate gyrus, indicating their pivotal roles on memory functions. These effects were further supported by the hippocampi upregulated neuroplasticity-related gene expression involving proliferation, differentiation, and migration using microarrays and quantitative real-time PCR techniques. Besides, we have also found that mPFC stimulation evoked a specific neurocircuitry modulation of the serotonergic pathways, linking the dorsal raphe nucleus in regulation of mood-related and hippocampal-dependent memory behaviors. Overall, our findings suggested that mPFC stimulation has the potential to be developed into a therapy to treat patients suffering from dementia as well as treatment-resistant depression.

GORBUNOVA, Vera PhD
Professor, Department of Biology, University of Rochester, USA

Vera Gorbunova is a Professor of Biology at the University of Rochester and a co-director of the Rochester Aging Research Institute. Her research is focused on understanding the mechanisms of longevity and genome stability and on the studies of exceptionally long-lived mammals. Dr. Gorbunova earned her BSc degrees at Saint Petersburg State University, Russia and her PhD at the Weizmann Institute of Science, Israel. Dr. Gorbunova pioneered comparative biology approach to study mechanisms of longevity. Dr. Gorbunova also investigates the role of Sirtuin proteins in maintaining genome stability. More recently the focus of her research has been on the longest-lived rodent species the naked mole rats and the blind mole rat. Dr. Gorbunova identified high molecular weight hyaluronan as the key mediator of cancer-resistance in the naked mole rat. Her work received awards of from the Ellison Medical Foundation, the Glenn Foundation, American Federation for Aging Research, and from the National Institutes of Health. Her work on cancer-resistance in the naked mole rat was awarded the Cozzarelli Prize from PNAS for outstanding scientific excellence and originality. Most recently she was awarded a prize for research on aging from ADPS/Alianz, France, Prince Hitachi Prize in Comparative Oncology, Japan, and Davey prize from Wilmot Cancer Center.

Longevity mechanisms in long-lived mammals

Animals have evolved a dramatic diversity of aging rates. Even within mammals, lifespans differ over 50-fold from four years in a mouse to 211 years in a bowhead whale. This natural diversity of lifespan can be exploited to understand the mechanisms of longevity. With modern technological advances now available, it became possible to undertake comparative study of aging at molecular level. Our goal is to identify mechanisms that allow such exceptionally long-lived animals to live long and healthy lives and then use these mechanisms to benefit human health. I will discuss our recent progress in the studies of tumor suppressor mechanisms, and DNA repair in short and long-lived rodent species.

OUYANG, Jun MD
Professor, Southern Medical University, China

Jun Ouyang, MD, is director of Department of Anatomy & Guangdong Provincial Medical Biomechanical Key Laboratory, Southern Medical University, Guangzhou, China.

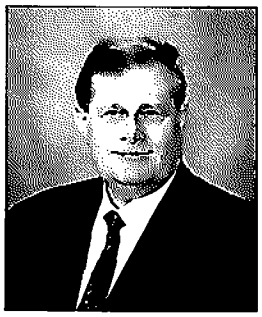
Professor Ouyang obtained his bachelor's degree in clinical medicine from the First Military Medical University in 1990 then subsequently earned his MD at the same university in 1996. He had his post-doctor fellow training in West Virginia University, USA from 2000 to 2002.

As chief editor of *Chinese Journal of Clinical Anatomy*, Prof. Ouyang's research interests focus on musculoskeletal biomechanics. He has authored or co-authored numerous articles that have appeared in peer-reviewed journals, including Spine, Journal of Trauma, Clinical Biomechanics, Clinical Orthopedics and Related Research, Acta of Biomengineering and Biomechanics, Surgical Radiology Anatomy, Journal of Gene Medicine, American Journal of Physiology et al.

He translated some classical works and atlas of anatomy into Chinese, include *Grant's Dissector* (Fifteenth Edition), *Atlas of Anatomy* (Lippincott Williams & Wilkins), *Lippincott's Concise Illustrated Anatomy* (3 Volumes), *Thieme Atlas of Anatomy* (Third Edition).

Gross Anatomy Teaching in Southern Medical University

As the basic discipline of medicine, anatomy teaching is always facing innovation challenges whenever new teaching methods have been practiced. No matter what we will do, the truth is that we have to teach the same anatomy knowledge within lesser hours than before. Effectiveness and quality are two important factors for evaluation. This presentation would introduce different anatomy course for different major in Southern Medical University, and related innovation.



PAWLINA, Wojciech MD
Professor of Anatomy and Medical Education, Department of Anatomy,
Mayo Clinic College of Medicine, Rochester, MN, USA

Wojciech Pawlina, MD, is a Professor of Anatomy and Medical Education at Mayo Clinic College of Medicine in Rochester, Minnesota, USA. He earned his medical degree from the Jagiellonian University College of Medicine in Cracow, Poland, where he worked in the Department of Anatomy while completing his residency in Obstetrics and Gynecology. Since 1986 he worked in the Department of Anatomy and Cell Biology at the University of Florida College of Medicine. In 1999 he relocated to Mayo Clinic in Rochester, Minnesota and currently serves as Chair of the Department of Anatomy and Director of the Procedural Skills Laboratory. He teaches gross anatomy, histology, and embryology to medical and dental students, as well as residents and fellows. He received numerous teaching awards from both the University of Florida and Mayo Clinic including Distinguish Mayo Educator Award. His research interest is directed towards strategies of implementing innovative teaching methodologies in anatomical sciences education, teaching professionalism, leadership, and teamwork in early medical curriculum. He has co-authored several textbooks in the field of anatomy, histology and medical education, as well as numerous scientific papers. He serves on several editorial boards of scientific journals and is also Co-Editor-in-Chief of the Anatomical Sciences Education.

Collaborative Ultrasound Objective Structural Practical Examination (OSPE) as a part of practical assessment in Gross Anatomy course

In the last few years ultrasound imaging has been introduced to many anatomy courses in the United States. However, very few if any offer formal assessment of students' ultrasound skills and anatomical knowledge at the same time. Throughout the anatomy didactic block at Mayo Medical School, first-year medical students were provided with both didactic briefing sessions and hands-on training on ultrasound basics, image interpretation and scanning techniques. As part of the final practical examination, students were expected to complete a collaborative team-based, hands-on ultrasound skills assessment. During this collaborative OSPE, students in four-person teams were expected to (1) setup the ultrasound machine, then (2) obtain, (3) orient, and (4) label an obtained image on a live model. Teams were graded using the rubric scale for each of the four components and given an overall score as a team. Each student was given a unique role within the exercise corresponding to the four tasks. The entire OSPE was performed in the classroom at 4 stations with 4 live models. Rotating through all 4 stations, students obtained, oriented and labeled 4 different structures. Collaborative OSPE was organized and supervised by near-peer teachers, who were trained third-year medical students working as teaching assistants (TAs) in the course. Collaborative OSPE assessment demonstrated an effective method to evaluate ultrasound skills and anatomy knowledge learned in the anatomy course.

RHYU, Im Joo MD, PhD
Professor, Department of Anatomy, College of Medicine,
Korea University, Korea

Rhyu IJ is a neuroanatomist with a background in medicine. He is interested in macroscopic and microscopic plastic response of the nervous system in response to various stimulations, especially motor activities. He demonstrated macroscopic brain plasticity of athletes with MRI and he investigates ultrastructure of the nervous tissue with various microscopic technique. With wide spectrum of morphological research tools, he is contributing to understand brain structure and function.

Gross Anatomy Education in Practical Anatomy Center, Korea University College of Medicine

Practical Anatomy Center is a gross anatomy laboratory of Korea University College of Medicine opened in 2012. The center was designed to provide anatomy education from a practical perspective not only for medical and undergraduate students but also for medical doctors including private practitioners as well as interns, residents, and faculty members of the university hospitals. The ventilation system of the center was carefully designed to minimize the odor during the dissection.

For first-year medical students, each session of cadaver dissection follows the class room lecture, and watching anatomical dissection videos for the corresponding chapter. During and after the cadaver dissection, students can observe plastic models and digital human body using virtual dissection table. By virtual dissection, students can make various sections required to understand sonographs, CT and MRI images. Before and after each cadaver dissection session, students are required to perform brief memorial ceremony for the deceased to improve attitude for professionalism as future medical doctors. As an elective course for family medicine during the clinical clerkship, "The Anatomical Basis of Physical Examination" class is provided to correlate the various landmarks used for physical examination in clinical practices with underlying organs, therefore to provide anatomical grounds for routine physical examination.

Multimodality anatomy laboratory with video watching, cadaver dissection combined with ethical education, and dry anatomy education module enhanced not only understanding the human body, but also building basis of professionalism in medicine.

Im Joo RHYU, Sun-Hwa Park, Chang-Sub UHM
Department of Anatomy, Korea University College of Medicine

TOPP, Kimberly PT, PhD, FAAA
President of American Association of Anatomists
Professor, Dept of Anatomy, University of California, San Francisco, USA

Dr. Kimberly Topp is Professor and Chair of the Department of Physical Therapy and Rehabilitation Science and Professor in the Department of Anatomy at the University of California San Francisco. She completed her PhD in Anatomy and Cell Biology at the University of California Davis and postdoctoral training in Neurobiology at UCSF. She oversees the anatomy curriculum for the doctor of medicine with 650 students, and the full curriculum for the doctor of physical therapy with 150 students. The Department of Physical Therapy includes the PhD in Rehabilitation Science, and outpatient physical therapy practices with 27 physical therapists. Dr. Topp's research interests are in neurobiology, and she has studied biomechanical properties of peripheral nerve and neuropathic pain. She is collaborating with scientists at UCSF to quantify the effects of chemotherapeutic drugs on the sensory and motor systems of patients with cancer. Dr. Topp has been the recipient of the Kaiser Award for Excellence in Teaching and the Sexton Sutherland Endowed Chair in Human Anatomy. She is a member of UCSF Academy of Medical Educators, a Fellow of American Association of Anatomists, and an honorary Fellow of The Anatomical Society. Dr. Topp is the current President of the American Association of Anatomists.

Learning to Contribute in an Inquiry-Based Curriculum

Gross anatomy education within health professions education is evolving rapidly, apace with larger curricular change. A program in Inquiry, in which students grapple with clinical questions for which there are no clear answers, now occupies one-quarter of the 4-year medicine curriculum at the University of California San Francisco. A similar approach is under development in the schools of pharmacy and dentistry. Where are the homes for foundational gross anatomy in such drastically modified curricula? How do we teach a foundational science in an educational environment striving to inform students of clinical uncertainly and the fragility of knowledge? How do we strategically hone our curricular "time" while also helping our students (and other faculty) to understand that gross anatomy has changed in the last 100 years, and that today's clinical questions and practice require that clinicians dive independently into anatomy far beyond that presented in the introductory course. This presentation will describe our honing of introductory gross anatomy and our move of deep-dives and creative approaches to anatomy to periods of time in close proximity to clinical practice. I will also describe our development of application-only assessments to facilitate the culture of inquiry, and lastly, the student's dashboard used to track progress and to guide self-directed learning in preparation for full-time clinical practice and life-long learning.

CHAN, Lap Ki
BSc, MBBS, FHKCOS, FHKAM, FRCS Edinburgh, PhD, MEd
Associate Professor, School of Biomedical Sciences,
The University of Hong Kong, Hong Kong

Dr. Chan is an anatomist and medical educator, with a background in orthopedics and physical anthropology. He is currently an associate professor in the School of Biomedical Sciences, and the deputy director of the Bau Institute of Medical and Health Sciences Education, at the Li Ka Shing Faculty of Medicine at The University of Hong Kong. He has a background in orthopedics and physical anthropology and teaches gross anatomy to medical students. His research interests include innovative pedagogies in anatomy education, problem-based learning (PBL), team-based learning (TBL), interprofessional education (IPE), and faculty development. He is the director of the Staff and Professional Development Program. He is an educator for the Asia Pacific region for the AO Foundation (Arbeitsgemeinschaft für Osteosynthesefragen). His teaching excellence has been recognized by such awards as the Thomas Henry Huxley Instructorship from Duke University, and most recently, an Outstanding Teaching Award from The University of Hong Kong. He serves as an associate editor for Anatomical Sciences Education and has edited, with Professor Wojciech Pawlina, "Teaching Anatomy – A Practical Guide", published by Springer.

Changing from passive to active learning: making dissection clinically relevant

The dissection laboratory is a small-group learning environment, which offers much opportunities for interactions, feedback, and reflection. However, in traditional dissection classes, students follow step-by-step instructions, such as those in popular dissection guidebooks. In order to utilize the active learning opportunities afforded by the interactive learning environment of the gross anatomy laboratory, clinically relevant team exercises were introduced into the dissection classes of the second year medical students in an undergraduate-entry, system-based, problem-based medical curriculum in Hong Kong. At the beginning of the traditional dissection class, a clinical problem was presented to the students (e.g. what is a safe level for inserting a chest drain, does the appendix lie at the McBurney's point). The cadavers represented the patients on which students need to solve the problem. The results of the team exercises of all student groups were then summarized by the teacher at the end of the class. As survey was conducted and students rated these statement the most highly (4.2 out of 5 on the Likert scale): the clinical exercises "made me more interested in anatomy", "increased my understanding of the relevance of cadaveric dissection to clinical medicine", and "The teachers in charge has made the learning points of these team exercises clear and relevant". Such team exercises seemed to enhance the clinical relevance of dissection to students' clinical education.

OKABE, Shigeo MD, PhD
Professor, Department of Cellular Neurobiology,
The University of Tokyo, Japan

I started my academic career as an assistant professor in Prof. Nobutaka Hirokawa's laboratory in the University of Tokyo, where I studied microtubule dynamics in neurons. As a postdoc in the laboratory of Prof. Ron McKay in NINDS/NIH, I switched my subject to neuronal stem cells. In 1996, I came back to Japan and became a Principal Investigator of National Institute of Bioscience and Human-Technology in Tsukuba and started my new research project on synapse dynamics. In 1999, I moved to Tokyo Medical and Dental University and then back to the University of Tokyo in 2007.

My current research interest is on the mechanisms of synapse development and remodeling in the mammalian cortex. Our research strategy is based on real-time imaging of synaptic molecules using modern fluorescence imaging techniques, such as in vivo two-photon microscopy.

Recent publications:
1) Nature Communications 3, 722, 2012., 2) Neuron 76, 549-564, 2012., 3) Nature Communications 4, 1440, 2013., 4) Nature Communications 5, 4742, 2014.

Imaging synapses in vivo – physiology and pathology of synapses and neural circuits

Proper regulation of synapse formation and elimination is a key feature in the development of neuronal circuits. Connectivity between neurons may be redundant in the early stage of development. Regulated pruning of excess and unnecessary synaptic connections takes place in the postnatal period and extrinsic instructive signals are thought to play important roles in this process.

By using in vivo two-photon imaging of GFP-labeled postsynaptic proteins, remodeling of individual synapses can be visualized directly and analyzed quantitatively. In the early postnatal period, cortical pyramidal neurons show higher turnover of synapses, which may reflect ongoing process of circuit remodeling. Pathophysiology of juvenile-onset psychiatric disorders may be related to impairments in this postnatal circuit remodeling. To test this possibility, we analyzed in vivo dynamics of spine synapses in mouse models of autism spectrum disorders (ASDs). We found up-regulation of spine turnover in multiple ASD mouse models with different genetic backgrounds. Neurons in the somatosensory cortex of ASD mouse models responded poorly to whisker stimulation, suggesting dysfunction of cortical information processing. Selective impairment in spine dynamics and less precise wiring in the neocortical circuits may be a core pathology of juvenile-onset psychiatric disorders and may be useful as a potential target of early intervention.

MCMENAMIN, Paul G BSc, PhD, MSc(Med Sci), D.Sc.(Med)
Professor, Department of Anatomy & Developmental Biology, School of Biomedical and Psychological Sciences, Monash University, Australia

Professor Paul McMenamin was born and raised in Glasgow, Scotland and originally did his PhD in eye research in 1978-1981 at Glasgow University. After post-doc training he moved to The Anatomy Department at Glasgow University and trained there for several years before moving to Perth, Western Australia to teach anatomy to undergraduate medical students and postgraduate specialist trainees. He continued to run a productive eye research laboratory in which immune mediated diseases of the eye became a focus. Paul moved to Monash University in 2010 to take up the position of Director of 'The Centre for Human Anatomy Education'. Throughout his career he has tried to be innovative in the way he teaches and has used audience response systems, body painting and more recently has been behind the development of a 3D printed human anatomy series that is commercially available. A recent trip to teach in Liberia in West Africa has strengthened his resolve to develop new resources which are accessible to developing countries or those without access to cadavers. He has recently converted the data from the 3D Printed Anatomy into a Virtual Reality experience. Meanwhile Paul continues to pursue his research in ocular immunology and neuroimmunology.

Innovations in anatomy teaching – a journey over 30 years through body painting, 3D printing and virtual reality

Whilst many medical schools, universities and teaching institutions charged with the task of teaching anatomy to medical students and allied health professionals have access to human cadaver material many institutions cannot access this resource for a number of reasons. Use of human cadavers in teaching involves special ethical and legal considerations as well as resources or facilities which have an inherent often hidden cost. In many countries cultural and religious factors also act as an impediment to cadaver-based anatomy teaching. In addition, many student groups simply do not have time to perform dissection yet they still have to learn human anatomy. Prof McMenamin will share some of his experiences from over 35 yrs of teaching in which he has transitioned body painting from life drawing art classes to his medical classes. He will also describe the processes behind the recently completed Monash 3D printed anatomical replica series and how these perform in a trial of student learning compared to prosections. He is currently exploring Virtual Reality as a means of taking students inside the body using real human data not 'cartoon' anatomy.

About Poster Awards

We will have Poster Award of HK$200 each to be awarded for the best 6 posters in the Closing Ceremony on December 6.

Adjudication panel for the poster sessions

HING, Hiang Lian
Universiti Kebangsaan, Malaysia

CHAN, Lap Ki
The University of Hong Kong, Hong Kong

CHAN, Leung Franky
The Chinese University of Hong Kong, Hong Kong

CHAN, Sun On Hector
The Chinese University of Hong Kong, Hong Kong

CHAN, Wood Yee
The Chinese University of Hong Kong, Hong Kong

CHANG, Raymond Chuen Chung
The University of Hong Kong, Hong Kong

CHEN, Hong
Fudan University, China

CHEUNG, Annie Lai-Man
The University of Hong Kong, Hong Kong

CHEUNG, Martin Chi-Hang
The University of Hong Kong, Hong Kong

CHUNG, Sookja Kim
The University of Hong Kong, Hong Kong

HUEN, Michael Shing Yan
The University of Hong Kong, Hong Kong

KARTHIK, Harve Subramhanya
National University of Singapore, Singapore

MA, Stephanie Kwai Yee
The University of Hong Kong, Hong Kong

WU, Wutian
The University of Hong Kong, Hong Kong

ZHOU, Zhongjun
The University of Hong Kong, Hong Kong

Poster Abstract
11
Cancer Biology

Anticancer Properties of Icariside II in Human Oral Squamous Cell Carcinoma Cells

Hae-Mi Kang [1,2], Su-Bin Yu [1,2], Young-Seok Kim [1], Jong-Jin Kil [1], Tae-Hyun Bang [1], Nak-Eun Choi [1], Hyun-Ho Kwak [1,2], In-Ryoung Kim [1], Bong-Soo Park[1,2*]
[1] Department of Oral Anatomy, School of Dentistry, Pusan National University
[2] BK21 PLUS Project, School of Dentistry, Pusan National University, Yangsan, Korea

OSCC is currently the most common malignancy of the head and neck, with tens of thousands of patients affected by this cancer per year worldwide. Natural flavonoids from plants are potential sources for novel anti-cancer drugs. Icariin is the active ingredient of flavonol glycoside, which is derived from the medical plant Herba Epimedii. A metabolite of icariin, icariside II has been demonstrated to exhibit a variety of pharmacological purposes, including anti-rheumatic, anti-depressant, cardiovascular protective, and immunomodulatory uses. However, the exact mechanism causing the apoptosis-inducing effect of icariside II in OSCC is still not fully understood. Thus, in the present study, we assessed the effect of icariside II as an anti-cancer agent against OSCC cell lines by measuring its effect on cell viability, cell proliferation, and MMP. Icariside II treatment of OSCC cells was found to result in a dose- and time-dependent decrease in cell viability. Hoechst staining indicated that the icariside II-treated HSC cells were undergoing apoptosis. Icariside II inhibited cell proliferation and induced apoptosis in HSC cells, in particular, significantly increasing all of the present data in HSC-4 cells. These findings clearly suggest that icariside II-induced apoptosis via activation of intrinsic pathways and caspase cascades in HSC-4 cell lines. Icariside II could be a potential treatment for OSCC, and it could provide valuable data for the development of a novel anti-cancer strategy.

Hae-Mi Kang : khaemi90@naver.com
Su-Bin Yu : ysb0811@gmail.com
Young-Seok Kim : stonexx@hanmail.net
Jong-Jin Kil : kiljj82@naver.com
Tae-Hyun Bang : bbang-t@daum.net
Nak-Eun Choi : na-geun@hanmail.net
Hyun-Ho Kwak : kwakhh@pusan.ac.kr
Bong-Soo Park: parkbs@pusan.ac.kr

All authors are affiliated Department of Oral Anatomy, School of Dentistry, Pusan National University
Address: School of Dentistry, Pusan National University 49, Busandaehak-ro, Mulgeum-eup, Yangsan-si, Gyeongsangnam-do, Korea

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Future Directions for Development in Anatomical Sciences

3-D Imaging of Embryonic Development of Xenopus Using Synchrotron Radiation microCT

Hongtae Kim, Sung-Mi Han, Mae-Ja Park, Jae-Hong Lim
Catholic University of Daegu School of Medicine, Korea

Understanding developmental processes requires accurate visualization and parameterization of three-dimensional embryos. In Xenopus laevis, the South African clawed frog, cell and tissue movements have been studied in explants, in fixed embryos, in vivo using fluorescence microscopy or microscopic magnetic resonance imaging. None of these methods allows cell behaviors to be observed with micrometer-scale resolution throughout the optically opaque embryos over developmental time. Here we use non-invasive synchrotron radiation microCT, based on single distance phase contrast and/or combined with simple staining methods, to examine the course of embryonic development. We demonstrate that this powerful three-dimensional imaging technique provides high resolution views of developmental processes of each stage in wild-type X. laevis embryos, including cleavage, gastrulation and neurulation. Synchrotron radiation microCT provides a useful tool for comparative developmental studies, embryo phenotyping, and quantitative modeling of development.

Autophagy Inhibition Promotes Quercetin Induced Apoptosis in MG-63 Human Osteosarcoma Cells

Su-Bin Yu [1,2], Hae-Mi Kang [1,2], Lee Ki-hyun[1], Jong-Jin Kil [1], Tae-Hyun Bang [1], Nak-Eun Choi [1], In-Ryoung Kim [1], Bong-Soo Park[1,2*]
[1] Department of Oral Anatomy, School of Dentistry, Pusan National University
[2] BK21 PLUS Project, School of Dentistry, Pusan National University, Yangsan, Korea

Quercetin is a natural flavonoide phytochemical that is extracted from various plants. Quercetin is used to treatment many disease, because of its biological activities, such as anti-inflammation, antivirus, antioxidant and anticancer. Recently, autophagy has been reported that may play a key role in anticancer therapy. Therefore, we set to this study to investigate the anticancer activities and molecular mechanisms of Quercetin against human osteosarcoma cells. As a result of these experiments, Quercetin inhibited cell proliferation and induced cell death through apoptotic process that is regulated by mitochondrial pathway and caspase-cascade. Quercetin also induced autophagy, this autophagy was inhibited by 3-MA known as an autophagy inhibitor. Blockade of autophagy promoted Quercetin-induced apoptosis, confirming that autophagy acts as a pro-survival process. Thus, these finding demonstrated that Quercetin is a great anticancer agent, but combination application of Quercetin and autophagy inhibitor is expected to enhance the effect of anticancer therapy.

Su-Bin Yu : ysb0811@gmail.com
Hae-Mi Kang : khaemi90@naver.com
Ki-Hyun Lee: skydocs@naver.com
Jong-Jin Kil : kiljj82@naver.com
Tae-Hyun Bang : bbang-t@daum.net
Nak-Eun Choi : na-geun@hanmail.net
Bong-Soo Park: parkbs@pusan.ac.kr

All authors are affiliated Department of Oral Anatomy, School of Dentistry, Pusan National University
Address: School of Dentistry, Pusan National University 49, Busandaehak-ro, Mulgeum-eup, Yangsan-si, Gyeongsangnam-do, Korea

Mechanism underlying Shikonin-induced apoptosis and cell cycle arrest on SCC25 human tongue squamous cell carcinoma cell line

Hae-Mi Kang [1,2], Su-Bin Yu [1,2], Young-Seok Kim [1], Sang-Hun Oh[1],
Jong-Jin Kil [1], Tae-Hyun Bang [1], Nak-Eun Choi [1], In-Ryoung Kim [1], Bong-Soo Park[1,2*]
[1] Department of Oral Anatomy, School of Dentistry, Pusan National University
[2] BK21 PLUS Project, School of Dentistry, Pusan National University, Yangsan, Korea

Shikonin, a major ingredient in the Chinese traditional herb Lithospermum erythrorhixon, exhibits multiple biological functions including antimicrobial, anti-inflammatory, and antitumor effects. More recently, shikonin has shown antitumor properties in many cancers. This study was undertaken to investigate the underlying mechanisms giving rise to modulation of cell cycle-related proteins and induction of apoptosis induction by shikonin in the SCC25 human tongue squamous cell carcinoma cell line.

The effects of shikonin on the viability and growth of SCC25 cells were assessed by the MTT assay and clonogenic assays, respectively. Hoechst staining and DNA electrophoresis indicated that the shikonin-treated SCC25 cells were undergoing apoptosis. Western blotting, immunocytochemistry, confocal microscopy, FACScan flow cytometry, MMP activity and proteasome activity also supported the induction of apoptosis by shikonin.

Shikonin treatment of SCC25 cells resulted in a time- and dose-dependent decrease in cell viability, a dose-dependent inhibition of cell growth, and apoptotic cell death. The treated SCC25 cells showed several lines of apoptotic manifestation such as nuclear condensation, DNA fragmentation, the reduction of MMP and proteasome activity, the decrease of DNA contents, the release of cytochrome c into cytosol, the translocation of AIF and DFF40 (CAD) onto nuclei, a significant shift of Bax/Bcl-2 ratio, and the activation of caspase-9, caspase-7, caspase-6, caspase-3, PARP, Lamin A/C and DFF45 (ICAD). Shikonin treatment also resulted in down-regulation of the G1 cell cycle-related proteins and up-regulation of $p27^{KIP1}$.

Taken together, our present findings demonstrate that shikonin strongly inhibits cell proliferation by modulating the expression of the G1 cell cycle-related proteins and it induces apoptosis via the proteasome, mitochondria, and caspase cascades in SCC25 cells.

Hae-Mi Kang : khaemi90@naver.com
Su-Bin Yu : ysb0811@gmail.com
Young-Seok Kim : stonexx@hanmail.net
Sang-Hun Oh: jahaoh@yahoo.co.kr
Jong-Jin Kil : kiljj82@naver.com
Tae-Hyun Bang : bbang-t@daum.net
Nak-Eun Choi : na-geun@hanmail.net
Bong-Soo Park: parkbs@pusan.ac.kr

All authors are affiliated Department of Oral Anatomy, School of Dentistry, Pusan National University
Address: School of Dentistry, Pusan National University 49, Busandaehak-ro, Mulgeum-eup, Yangsan-si, Gyeongsangnam-do, Korea

XIAP inhibitor Embelin induces autophagic and apoptotic cell death in human oral cancer cells

Su-Bin Yu [1,2], Hae-Mi Kang [1,2], Young-Seok Kim [1], You-Jin Lee[1],
Jong-Jin Kil [1], Tae-Hyun Bang [1], Nak-Eun Choi [1], In-Ryoung Kim [1], Bong-Soo Park[1,2*]
[1] Department of Oral Anatomy, School of Dentistry, Pusan National University
[2] BK21 PLUS Project, School of Dentistry, Pusan National University, Yangsan, Korea

Embelin is an active ingredient of traditional herbal remedies for cancer and other diseases. Recently, it has been suggested that autophagy may play an important role in cancer therapy. However, little data are available regarding the role of autophagy in oral cancers. Therefore, we conducted this study to examine whether Embelin modulates autophagy in CA9-22. Our results showed that Embelin had anticancer activity against the CA9-22 human tongue squamous cell, and we observed that autophagic vacuoles were formed by MDC and AO. We also analyzed Embelin-treated CA9-22 cells for the presence of biochemical markers and found that it directly affected the conversion of LC3-II, the degradation of p62/SQSTM1, full-length cleavage formation of ATG5-ATG12 complex and Beline-1, and caspase activation. Rescue experiments using an autophagy inhibitor showed Embelin-induced cell death in CA9-22, confirming that autophagy acts as a pro-death signal. Furthermore, Embelin exhibited anticancer activity against CA9-22 via both autophagy and apoptosis. These findings suggest that Embelin may potentially contribute to oral cancer treatment and provide useful information for the development of a new therapeutic agent.

Su-Bin Yu : ysb0811@gmail.com
Hae-Mi Kang : khaemi90@naver.com
Young-Seok Kim : stonexx@hanmail.net
You-Jin Lee: jahaoh@yahoo.co.kr
Jong-Jin Kil : kiljj82@naver.com
Tae-Hyun Bang : bbang-t@daum.net
Nak-Eun Choi : na-geun@hanmail.net
Bong-Soo Park: parkbs@pusan.ac.kr

All authors are affiliated Department of Oral Anatomy, School of Dentistry, Pusan National University
Address: School of Dentistry, Pusan National University 49, Busandaehak-ro, Mulgeum-eup, Yangsan-si, Gyeongsangnam-do, Korea

Poster Abstract
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Neuroscience

Pituitary Adenylate Cyclase-Activating Polypeptide Enhances Saliva Secretion via Direct Binding to PACAP Receptors of Major Salivary Glands in Mice

Naoko Nonaka, Masanori Nakamura
NN: choku@dent.showa-u.ac.jp, MN: masanaka@dent.showa-u.ac.jp
Department of Oral Anatomy and Developmental Biology, Showa University School of Dentistry, Tokyo, Japan

Xerostomia, or dry mouth, is a common syndrome that is generally treated with artificial saliva; however, no other effective methods have yet been established. Saliva secretion is mainly under the control of the autonomic nervous system. Pituitary adenylate cyclase-activating polypeptide (PACAP) is recognized as a multifunctional neuropeptide in various organs. In this study, we examined the effect of PACAP on saliva secretion, and detected the distribution of the PACAP type 1 receptor (PAC1R) in major salivary glands, including the parotid, submandibular, and sublingual glands, in 9-week-old male C57BL/6 mice. Intranasal administration of PACAP 38 increased the amount of saliva secreted, which was not inhibited by atropine pretreatment. Immunohistochemical analysis showed that PAC1R was distributed in the three major salivary glands. In the parotid and sublingual glands, PAC1R was detected in striated duct cells, whereas in the submandibular gland, a strong PAC1R immunoreaction was detected in tall columnar epithelial cells in the granular ducts (i.e., pillar cells), as well as in some striated duct cells. PACAP significantly increased the concentration of epidermal growth factor in saliva. These results suggest that PACAP directly regulates saliva secretion by controlling the absorption activity in the ducts, and that pillar cells regulate the function of granular epithelial cells in the granular duct, such as the secretion of growth factors into the saliva. Collectively, these results suggest the possibility of PACAP as a new effective treatment of xerostomia

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Advanced Biomedical Platforms

Study of ancient human DNAs from remaining bones

Kyung Yong Kim, Kijeong Kim, Seung-Ho Han, Yoon-Hee Chung, Won-Bok Lee
Department of Anatomy, College of Medicine, Chung-Ang University, South Korea

Ancient human body remains have provided useful information about past human livings. Mummies are the best evidences of the ancient human lives. But most of human remains are bones. With times, the human bones also disappear by chemical decomposition. Human skeletal remains are well preserved in cold and dry area. DNAs can be extracted from ancient human skeletons. The human DNAs are fragmented in short sequences, contaminated with humic acid from soil, and reduced in amount of DNAs. These three are the limiting factors for the study of ancient human DNAs. Ancient human DNA analysis has growingly been the fields of many researchers including molecular and evolutionary anthropology, human history, medical genetics, genetic genealogy, forensic science, and other fields including paleontology (microbiology, botany, and etc), We have studied ancient human DNAs form bones up to 3000 years ago from Asia. We would like to introduce the molecular human anthropology with some successful methods.

Protective Effect of Rhus verniciflua Stokes Extract Against Renal Ischemia-Reperfusion Injury Enhancing Nrf2-mediated Induction of Antioxidant Enzymes

Du Ri Choi, Ji Heun Jung, Kwang Sik Yu
Department of Anatomy, KonYang University, Daejeon, South Korea

The ischemia-reperfusion injury (IRI) often causes acute kidney disease (AKD) by mediating oxidative stress-induced apoptosis of parenchymal cells. The extract of Rhus verniciflua Stokes (RVS) is used as a traditional herbal medicine and is known to exert the anti-oxidative, anti-apoptotic, and anti-inflammatory properties. Herein, we investigated the therapeutic effect and the underlying mechanism of RVS on IRI-induced AKD by performing in vivo and in vitro experiments, which were established by surgical modeling of renal IRI on mice and chemical induction of hypoxia on human renal tubular epithelial cell line, HK-2, respectively. We demonstrated that the IRI-induced elevation of blood urea nitrogen, serum creatinine, and lactate dehydrogenase were significantly attenuated by intra-oral (i.o) administration of RVS (20 mg/kg/day) for 14 days before surgery. We observed that IRI surgery drastically induced the histological damages and cellular apoptosis on renal parenchyma, which were attenuated by i.o RVS pretreatment. Furthermore, we found that RVS treatment significantly inhibited the cell death and reactive oxygen species (ROS) production of HK-2 cells which were damaged by chemical hypoxia triggered by incubation with 300 μM cobalt chloride. Interestingly, we observed that RVS treatment upregulated the level of endogenous antioxidant enzymes e.g., heme oxidase-1 and catalase, as well as their upstream regulator, nuclear factor erythroid 2–related factor 2 in HK-2 cells. Taken together, these results suggested that i.o intake of RVS has a therapeutic effect on IRI-induced AKD and these effects are, at least partly, due to the attenuation of ROS production through upregulating the antioxidant defense system inside the renal tubular cells.

A Test of Methods for Age Estimation by using Acetabulum in a Thai Population

Phruksachat Singsuwan[a], Apichat Sinthubua[a,b,d], Sukon Prasitwattanaseree[a,c,d] ,and Pasuk Mahakkanukrauh[a,b,d,*]
[a]Forensic Osteology Research Center, Faculty of Medicine, Chiang Mai University, 50200, Thailand
[b]Department of Anatomy, Faculty of Medicine, Chiang Mai University, 50200, Thailand
[c]Department of Statistics, Faculty of Science, Chiang Mai University, 50200, Thailand
[d]Excellence Center in Osteology Research and Training Center (ORTC), Chiang Mai University, 50200, Thailand
*Corresponding author. E-mail: pasuk034@gmail.com
E-mail addresses: phruksachat_sing@cmu.ac.th (Singsuwan P.), apichat_nine@yahoo.com (Sinthubua A.), sprasitwattanaseree@gmail.com (Prasitwattanaseree S.)

Age estimation is an important step in the biological identification of human remains. The os coxa, especially acetabulum is a reliable age indicator. Besides, this region often preserves better than the pubis. This study aimed to test Rissech *et al.*'s original scoring method and to derive regression equation for age estimation in a Thai population. A Thai sample of 220 skeletons (110 males and 110 females) from the Forensic Osteology Research Center, Faculty of Medicine, Chiang Mai University were observed. The age range was between 22 to 90 years with an average age of 61.6 years for males, and 62.7 years for females. The seven variables were considered as follows: (i) acetabular groove (AG), (ii) acetabular rim shape (ARS), (iii) acetabular rim porosity (ARP), (iv) apex activity (AA), (v) activity on the outer edge of the acetabular fossa (AOAF), (vi) activity of the acetabular fossa (AAF), and (vii) porosities of the acetabular fossa (PAF). Correlation between each variable and known age at death by Kruskal–Wallis test were all significant. The derived regression equation for estimating age at death as follows: Age = 18.33 + 2.38(ARS) + 4.17(ARP) + 2.31(AA) + 2.40(AOAF) + 2.12(PAF). The *R* square value revealed 0.466. The standard error of the estimate was 12.11 years. The current study could be supplemented in the future by testing on the holdout samples and modified by adjusting the variables and the equation model for application to age estimation from acetabulum in Thailand.

Keywords: Forensic anthropology, Age estimation, Acetabulum, Os coxa, Thai population

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Poster Abstract
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Aging

Effects of Caloric restriction on hepatic steatosis and hepatic metabolism in db/db mice

Kyung Eun Kim[1], Eun Ae Jeong[1], Chin-ok Yi[1], Jong Youl Lee[1], Eun Ha Jang[1], Hwajin Kim[1], Sang Won Park[2], Dong Hoon Lee[1], Hyun Joon Kim[1], Sang Soo Kang[1], Gyeong Jae Cho[1], Wan Sung Choi[1], Gu Seob Roh[1]
[1]Department of Anatomy and Convergence Medical Science, Bio Anti-aging Medical Research Center, Institute of Health Sciences, Gyeongsang National University School of Medicine, Jinju, Gyeongnam, 52727, Republic of Korea
[2]Department of Pharmacology, Bio Anti-aging Medical Research Center, Institute of Health Sciences, Gyeongsang National University School of Medicine, Jinju, Gyeongnam, 52727, Republic of Korea

Non-alcoholic fatty liver disease (NAFLD) is one of the most frequent causes of liver disease and its prevalence is a serious and growing clinical problem. Caloric restriction (CR) is commonly recommended for improvement of obesity-related diseases such as NAFLD. However, the effects of CR on hepatic metabolism remain unknown. We investigated the effects of CR on metabolic dysfunction in the liver of obese and diabetic db/db mice. We found that CR of db/db mice reverted insulin resistance, hepatic steatosis, body weight and adiposity to those of wild type mice. Moreover, western blot analysis showed that lipogenesis pathway enzymes in the liver of db/db mice were reduced by CR. In addition, CR reversed ketogenesis pathway enzymes and enhanced autophagy, mitochondrial dysfunction, fibrogenesis and endoplasmic reticulum stress in db/db mice. In particular, hepatic inflammation-related proteins including lipocalin-2 in db/db mice were attenuated by CR. Therefore, these findings showed that CR is a good non-genetic intervention therapy for attenuating the deleterious effects of obesity and diabetes-induced multiple complications.

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Future Directions for Development in Anatomical Sciences

Stature Estimation from Vertebral Column in a Thai Population

Apichat Sinthubua[a,b,c,*], Chirapat Inchai[a], Sitthiporn Ruengdit[b], Pasuk Mahakkanukrauh[a,b,c]
[a]Department of Anatomy, Faculty of Medicine, Chiang Mai University, 50200, Thailand
[b]Forensic Osteology Research Center, Faculty of Medicine, Chiang Mai University, 50200, Thailand
[c]Excellence Center in Osteology Research and Training Center (ORTC), Chiang Mai University, 50200, Thailand
*Corresponding author: E-mail: apichat_nine@yahoo.com
E-mail addresses: zest_vj@hotmail.com (Inchai C.), 4805236@gmail.com (Ruengdit S.), pasuk034@gmail.com (Mahakkanukrauh P.)

Stature estimation is one of the major components in forensic identification. Many literatures purposed the relationship and stature estimation equations between body height and long bone measurements. Vertebral column was also considered as a reliable indicator for stature estimation. The length of vertebral column contributes around 30% of the total skeleton stature. Several studies reported the regression formulae for stature estimation in a Thai population using measurements of long bones of the extremities. However, there is no study on stature estimation applying vertebral column in Thais. The purpose of this study is to estimate stature from 100 vertebral column belonged to Thai individuals with age ranged between 22-94 years housed in Forensic Osteology Research Center, Faculty of Medicine, Chiang Mai University. Measurement of the vertebral body, including anterior body height, middle body height and posterior body height obtained from C3to S1 vertebrae.
Pearson correlation analysis showed moderate to high correlation coefficients between stature and each measurement which are 0.459 to 0.717 (anterior body height), 0.593 to 0.710 (middle body height), and 0.522 to 0.725 (posterior body height). Regression formular showed the standard error of estimation (SEE) ranged from 5.796 to 6.831 centimeters. The measurements from each vertebra delivered relatively high relationship with stature. Therefore, it might aid in stature estimation even only a single vertebra is found. In conclusion, vertebral body height could be useful for stature estimation in personal identification in forensic circumstances.

Keywords: Stature estimation, Vertebral column, Thai population

(Acknowledgement: The authors are grateful to Forensic Osteology Research Center, Faculty of Medicine, Chiang Mai University for access to skeletal collection)

Poster Abstract
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Neuroscience

Resveratrol Reduces Retinal Ischemia/Reperfusion Injury through the Down-regulation of Caspase-3 mRNA in Mouse

Jinhyun Ryu[1], Naeun Park[1], Gyeong Min Kang[1], Seong Wook Seo[2], Jong Moon Park[2], Sang Soo Kang[1]
[1]Department of Anatomy and Convergence Medical Science, Center for Anti-aging industrializing platform, Gyeongsang National University, Jinju, South Korea
[2]Department of Ophthalmology, Institute of Health Science

Ischemia/reperfusion (I/R) injury is a common animal model for the study of ischemic cell death in retina leading to blind. I/R injury increases caspase-3 expression. The expression of caspase-3 is highly related with activation of apoptotic pathway. However, the mechanism of cell death in retinal I/R injury is not clear. Resveratrol (Res) is one of the effective natural compounds in many ocular diseases by the regulation of transcription factor such as NF-κB and p53. And Res also related with the regulation of apoptosis pathway. The expressions of caspase-8 and caspase-3 are the major markers for apoptosis. Therefore, the present study aims to reveals whether Res regulates the expression of caspase-8 and caspase-3 after retinal I/R injury. Res was administered to male C57BL/6 mice for 5 days (pre-treated for 2 days and post-treated for 3 days, 20 mg/kg/day, i.p.). Retinal I/R injury was induced by high intra-ocular pressure for 1 h. The change of retinal morphology was observed by H&E staining. The mRNA was extracted 2 days later after I/R injury and the levels of caspase-8 and caspase-3 mRNA were measured by RT-PCR. The retinal I/R injury increased loss of retinal thickness and retinal ganglion cell numbers. The expressions of caspase-8 and caspase-3 mRNA were also increased. The Res treatment reduced the change of retinal morphology and decreased the expression of caspase-8 and caspase-3 mRNA. The changes of retinal morphology suggest the increase of retinal cell death after I/R injury. And the expression of caspase-8 and caspase-3 mRNA reveals the increase of apoptosis. This study demonstrates that Res reduce retinal I/R injury by suppression of caspase-3 expression in mouse model.

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Cancer Biology

O-linked N-acetylglucosamine transferase promotes cervical cancer tumorigenesis through human papillomaviruses E6 and E7 oncogenes

Minjun Kim, Akhtar Ali,Dong Hoon Lee, Gu Seob Roh, Hyun Joon Kim, Sang Soo Kang, Gyeong Jae Cho, Wan Sung Choi*
Department of Anatomy and Convergence Medical Science,
Gyeongsang National University School of Medicine, Korea

Human Papillomaviruses (HPVs) are the primary cause of cervical cancer and two immediate early genes, E6 and E7, are critical for tumorigenesis. The O-linked N-acetylglucosaminylation (O-GlcNAcylation), a posttranslational modification of proteins catalyzed by O-linked N-acetyl-glucosamine (O-GlcNAc) transferase (OGT), is increased in various cancers. Host cell factor-1 (HCF-1) is a co-transcription factor required for viral infection. We investigated if the O-GlcNAcylated HCF-1 increases E6 and E7 expression in HPV16/18-positive cervical cancers. We examined expression of O-GlcNAc, OGT, and HCF-1 in cervical cancer patients or normal subjects by western blotting and immunohistochemistry. We analyzed the effect of O-GlcNAcylation of HCF-1 on E6 and E7 expression and tumor formation in HeLa cells of OGT overexpression or knock-down. We found that expression of O-GlcNAc, OGT, and HCF-1 was increased in cervical cancer, with increased E6 and E7 levels. HPV16/18-positive cervical cancer cells exhibited increased O-GlcNAc and OGT expression, compared to controls. When cells were grown in high glucose or transfected with OGT, the levels of O-GlcNAc, HCF-1, E6, and E7 were increased. The interaction between OGT and HCF-1 was also increased and HCF-1 was O-GlcNAcylated. OGT shRNA (shOGT)-mediated knockdown decreased levels of HCF-1, E6, and E7, and reduced proliferation, invasion, and metastasis. Xenograft studies showed decreased tumor size in shOGT-infected cells. The results suggest that OGT increases E6 and E7 expression through O-GlcNAcylation of HCF-1, and promotes cervical carcinogenesis. We propose that inhibition of OGT prevents reactivating E6 and E7 oncogenes and serve as a strategy for treating cervical cancer.

Poster Abstract
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Developmental Biology

Primary cilia is a marker of kidney mass reduction

Sang Jun Han, Jee In Kim, Kwon Moo Park
Kyungpook National University School of Medicine, Republic of Korea

Primary cilium length changes are associated with various diseases including acute and chrorinc kidney injury. Here, we investigated whether renal mass reductions affect primary cilia length and its molecular mechanisms. Mice were subjected to either unilateral nephrectomy (UNx) or unilateral ischemia for 30 min (UI) and 9 days after those operations primary cilia were observed. Some mice were administered Mn(III) Tetrakis (1-methyl-4-pyridyl) porphyrin (MnTMPyP, a ROS scavenger). Primary cilium was observed by immunofluorescence staining using anti-acetylated-a-tubulin antibody and its length was determined under microscope. UNx increased primary cilium length in kidney tubule cells and parietal cells of the remaining kidney. UI also increased primary cilium length in tubule cells and parietal cells of the UI-exposed kidneys. Treatments of MnTMPyP prevented those elongations of primary cilia. UNx resulted in the hypertrophy of the remaining kidneys. UI induced tubular cell damage and fibrosis in the UI-induced kidneys. Treatments of MnTMPyP prevented the hypertrophy of the remaining kidney after UNx and post-UI kidney tubule cell damage and fibrosis. These results indicate that renal mass reduction change primary cilia length via ROS production.

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Cancer Biology

OGT-knockdown enhances sensitivity to cisplatin through the decreased sCLU expression by hypo-O-GlcNAcylated LXRs and down-regulated SREBP expression in HeLa cells

Minjun Kim, Akhtar Ali, Mi Young Choi, Dong Hoon Lee, Gu Seob Roh, Hyun Joon Kim, Sang Soo Kang, Gyeong Jae Cho, Yoon Sook Kim, Wan Sung Choi*
Gyeongsang National Univ. School of Medicine, Jinju, Korea

O-linked N-acetylglucosamine Transferase (OGT) expression is increased in the various types of cancers and O-GlcNAcylation might be a modification process in cancer formation and progression. Secretory Clusterin (sCLU) is also involved in cancer cell proliferation and drug resistance. Recently, it was reported that Liver X receptors (LXRs) and Sterol response element binding protein (SREBP) regulates CLU transcription and O-GlcNAcylated LXRs transports into nucleus to enhance the transcriptional activity. Here, we found that sCLU was significantly increased in cervical cancer cell lines which showed higher expression levels of O-GlcNAc and OGT, compared to normal keratinocyte. OGT-knockdown decreased the expressions of LXRs and SREBP as well as sCLU. And also, increased O-GlcNAcylation by Thiamet-G treatment enhanced sCLU expression. Treatment of Don, Glutamine fructose-6-phosphate amidotransferase (GFAT) inhibitor, decreased levels of O-GlcNAcylation and sCLU in HeLa cells. Moreover, levels of LXRs, SREBP and sCLU in HeLa cells exposed to high glucose which is the highly O-GlcNAcylated condition were significantly elevated as comparing to low glucose condition. Moreover, OGT targeting decreased the interaction between OGT and LXRs and down-regulated the level of O-GlcNAcylated LXRs. Finally, OGT-knockdown decreases cell proliferation and increases apoptosis induced by cisplatin treatment in HeLa cells. Taken together, these findings suggest that OGT, O-GlcNAcylated LXRs and SREBP-1 increases sCLU which contributes to drug resistance in cervical cancer cells.

5-Aminosalicylic Acid Protects Epithelial Tight Junction Thereby Attenuating Intestinal Barrier Defect in Colitic Mice

Ji Heun Jeong. Kwang Sik Yu, Duri Choi, Nam Seob Lee, Young Gil Jeong, and Seung-Yun Han
Departments of Anatomy, College of Medicine, Konyang University, 158 Gasuwon-dong, Seo-gu, Daejeon 302-718, Republic of Korea.
E-mail: jjh4110@konyang.ac.kr

Intestinal barrier defects are involved in the pathogenesis of inflammatory bowel disease (IBD). While the oral supplement of 5-Aminosalicylic Acid (5-ASA) is currently used as a first-line therapy for IBD, underlying therapeutic mechanisms are largely unknown. The present study investigated the ameliorative effects of 5-ASA on epithelial tight junction (TJ) barrier defects using a mice model of IBD. For these, mice were divided into 3 groups: the control (the group were administered water for 5 days), the DSS [3.5 % dextran sulfate sodium (DSS) in combination with phosphate buffered saline as a vehicle for 5 days], and the DSS+5-ASA group [3.5 % DSS in combination with 5-ASA (20 mg/kg/day) for 5 days]. DSS group showed severe colon damage, as indicated by colon shortening and increased disease activity index i.e., loss of body weight and increases of fecal occult blood as well as stool consistency. In DSS group, histologic alterations were also evident as demonstrated by goblet cell denudation and diffuse mural thickening with submucosal edema. Furthermore, DSS group showed the functional impairment on intestinal barrier integrity, as demonstrated by marked translocation of fluorescein sodium (F-Na) into the blood at 4 hrs after the oral intake. On the contrary, all these symptoms were significantly attenuated in DSS+5-ASA group. Notably, observation under electron microscopy (EM) revealed that DSS group showed the ultrastructural aberration on the colonic epithelial TJ, which was markedly diminished in DSS+5-ASA group. Immunohistochemical and semi-quantitative analysis for detecting the TJ proteins (zonula occludens-1, occludin, and claudin-5) were corroborated the EM finding well. Finally, it was demonstrated that DSS group exhibited the substantial elevation on colonic gene expressions of matrix metalloproteinase-2 and -9, both enzymes responsible for TJ degradation, which were significantly decreased in DSS+5-ASA group. Taken together, it might suggest that protective effects of 5-ASA on DSS-induced colitis at least partially due to protection of the TJ barrier in colonic epithelium.

Poster Abstract
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Cancer Biology

Deregulation of RNA N6-adenosine methylation promotes liver carcinogenesis

Mengnuo Chen, Wei Lai, Chuck-Ting Law, Felice Ho-Ching Tsang, Iris Ming-Jing Xu, Joyce Man-Fong Lee, Carmen Chuk-Lui Wong, Irene Oi-Lin Ng, Chun-Ming Wong.
The State Key Laboratory for Liver Research, Department of Pathology, Li-Ka Shing Faculty of Medicine, The University of Hong Kong

Epigenetic alterations profoundly contribute to human carcinogenesis. Traditionally, epigenetic studies are primarily focused on DNA methylation, histone modifications, and chromatin remodeling. Recently, emerging evidences suggest that diverse and revisable chemical modifications on RNAs, also known as "epi-transcriptome", represent a new layer of epigenetic regulation. N-6-methyladenosine (m6A) is the most abundant modification on eukaryotic mRNA. m6A modification is involved in regulating mRNA stability, alternative splicing, and translation efficiency. However, the roles of m6A deregulation in human carcinogenesis remain to be explored. By whole-transcriptome sequencing (RNA-Seq), we first identified that METTL3, the major m6A methyltransferase, was significantly upregulated in human liver cancer and other solid tumors. Clinically, overexpression of METTL3 was associated with poor patent survival. Functionally, we proved that knockdown of METTL3 remarkably inhibited cancer cell proliferation, migration, and colony formation *in vitro* and substantially suppressed tumor formation in nude mice. On the contrary, using CRISPR-dCas9 activation system, we demonstrated that overexpression of METTL3 significantly augmented *in vivo* tumorigenicity. To delineate the underlying molecular mechanisms, we interrogated the transcriptome changes of two METTL3 knockdown cell lines by RNA-Seq. To this end, we identified SOCS2, a prominent tumor suppressor, as a down-steam target of METTL3 mediated m6A modification. With m6A-RNA immunoprecipitation (RIP) assay, we confirmed that SOCS2 mRNA was subject to m6A modification in cancer cells. Inactivation of METTL3 by RNAi or treatment of methylation inhibitor (DAA) impaired m6A mediated mRNA degradation and thereby stabilized SOSC2 mRNA. In conclusion, our findings suggested that deregulation of METTL3 and its associated m6A modification could contribute to human carcinogenesis by imposing an epigenetic control on the stability and expressions of critical tumor suppressor genes.

Thrombocytes Modulate Neuroinflammation, Neuronal Plasticity and Recovery during Traumatic Brian Injury

M. Dukhinova, I. Kuznetcova, T. Veremeyko, E. Kopeykina, A. Yung, E.D. Ponomarev
School of Biomedical Sciences, Faculty of Medicine, The Chinese University of Hong Kong

Platelets (thrombocytes) are recognized as innate immune cells, which promote inflammation and tissue recovery on early stages after the trauma. Blood-brain barrier disruption occurs during large set of neurodegenerative and neurological disorders, including traumatic brain injury (TBI) and allows blood and immune cells enter the CNS. However, the role of platelets in neuroinflammation remains unclear.

Our research group has found previously that platelets could specifically recognize CNS-specific sialated glycolipids (gangliosides) within neuronal and astroglial lipid rafts [1]. ST3GalV knock-out (ST3KO) mice that are lack of major CNS-specific gangliosides exhibit impaired platelet - brain lipid raft interaction and reduced neuroinflammation during the onset of experimental autoimmune encephalitis. In the current study we investigated the role of platelet - CNS-specific ganglioside interaction in neuroinflammation and recovery in wild type (WT) and ST3KO mice TBI.

We found that ST3KO mice exhibited reduced level of microglia and macrophage activation, as well as lymphocyte infiltration to the brain after TBI. TBI was associated with more severe hemorrhage and neuronal damage at the brain trauma site of ST3KO, than WT, animals. Intracerebral injection of WT brain lipid rafts increased neuroinflammation and reduced hemorrhage initiated by TBI in ST3KO mice. Our results suggested that platelet-derived factors, such as serotonin, histamine and platelet-activating factor, were likely important mediators of platelet effect during TBI. Moreover, platelets could directly interact with cortical neurons exhibiting neurotrophic and neuroprotective features. Platelets activated with WT, but not ST3KO, brain lipid rafts increased the expression of neuroplasticity markers PSD95, synapsin-1 and Trkβ and dendritic spine number in neuronal and brain organotypic culture and reduced the loss of these markers after TBI.

Our study demonstrated the essential role of thrombocyte – ganglioside interaction in regulation of neuroinflammation, hemorrhage, neuronal plasticity and recovery after TBI. In conclusion, we suggest that platelets can contribute to other neuroinflammatory and neuropsychiatric disorders, such as Alzheimer's disease, stroke and epilepsy.

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Correspondence address: 323R, Lo Kwee-Seong Integrated Biomedical Sciences Building, Area 39, The Chinese University of Hong Kong, Shatin, New Territories, Hong Kong SAR, China

Telephone number: 3943 9621

Poster Abstract
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Cancer Biology

HELLS promotes aberrant chromatin remodeling and epigenetic silencing of tumor suppressor genes in liver cancer

Cheuk Ting LAW, Lai WEI, Felice HC TSANG, Iris MJ XU, Robin KH LAI, Daniel WH HO, Joyce MF LEE, Carmen CL WONG, Irene OL NG and Chun-Ming WONG
The State Key Laboratory for Liver Research and Department of Pathology, Li Ka Shing Faculty of Medicine, The University of Hong Kong

Epigenetic regulation is mediated by the orchestration of DNA methylation, histone modifications, and chromatin remodeling. Dysregulation of epigenetic control is a common trait of human cancers. While the critical involvement of aberrant DNA methylation and histone modifications in carcinogenesis have been recently emphasized, our knowledge on chromatin remodeling is still very limited. Exactly how deregulation of chromatin remodeling implicates in human carcinogenesis remains largely elusive. In a systematic profiling analysis of known epigenetic modifying genes, we herein unveiled a frequently up-regulation of chromatin remodeling enzyme HELLS (helicase, lymphoid-specific) in liver cancer and other solid tumors. Up-regulation of HELLS was associated with more aggressive cancer phenotypes and poor survival of liver cancer patients. Overexpression of HELLS significantly promoted cancer cell growth. In contrast, genetic ablation of HELLS by shRNA or CRISPR/Cas9 system drastically suppressed cancer cell proliferation and migration *in vitro* as well as abolished tumorigenicity and lung metastasis in orthotopic implantation model in vivo. Up-regulation of HELLS also facilitated metabolic reprogramming in cancer cells. By MNase-Seq and RNA-Seq, we delineated the downstream targets and molecular mechanisms of HELLS mediated epigenetic silencing in liver cancer. We showed that actively transcribed genes are typically associated with a nucleosome-free region (NRF) at their transcription start site (TSS). Formation of such NRF increases the DNA accessibility and enables the binding of transcription factors. We demonstrated that HELLS imposed epigenetic silencing by increasing nucleosome occupancy at NRF or enhancer regions of its target genes. Up-regulation of HELLS disrupted the formation of NFR and therefore precludes the binding of transcript factors for gene activation. This mechanism leads to the epigenetic silencing of multiple tumor suppressor genes, including E-Cadherin, FBP1, IGFBP3, XAF1 and CREB-H in liver cancer. In conclusion, our findings suggested that chromatin remodeling enzyme HELLS is a key epigenetic driver of human cancers. By promoting nucleosome occupancy on NRF, HELLS epigenetically silences multiple tumor suppressor genes to promote human carcinogenesis.

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Neuroscience

Study the effect of brain-derived neurotrophic factor on microglial activation

Wen-Ting Li, Shih-Ying Wu, Yu-Min Kuo
College of Medicine, National Cheng Kung University, Taiwan

Microglial activation may contribute to neuroinflammation and has been suggested to play an important role in the pathogenesis of several neurodegenerative diseases. Brain-derived neurotrophic factor (BDNF) is a member of the neurotrophin family, which is related to the canonical nerve growth factor. BDNF has been shown to reduce the levels of proinflammatory cytokines in animal models of spinal cord injury and stroke. However, whether the reduced inflammation was caused by a direct anti-inflammation effect or an indirect neuroprotection effect of BDNF is still unclear. Here, we showed that BDNF receptor, tropomyosin-related kinase B (TrkB), was abundantly expressed in neurons and microglia. Pretreatment, but not posttreatment, of BV2 cells (a microglial cell line) with BDNF inhibited lipopolysaccharide (LPS)-induced activations of p38 and JNK and NF-κB and productions of proinflammatory cytokines. The effect of BDNF on microglial activation was also investigated in the NF-kB-luc transgenic mice. Our preliminary results showed that LPS-induced upregulation of NF-kB-luc signals in brain could be inhibited by peripheral injection of BDNF.

Perilla seed oil ameliorates nonalcoholic fatty liver in mice fed with high-fat diet via the augmented lipolytic activity

Sora Kim, Naeun Pak, Theodomir Dusabimana, Sang Won Park, Kyuho Jeong, Jihyun Je, Su Ji Kim, Hye Jung Kim, Ki Churl Chang
Gyeongsang National University School of Medicine, South Korea

Nonalcoholic fatty liver disease is the most prevalent chronic liver disease and increases the risk of cardiovascular disorders, such as atherosclerosis. Recently, the animal oils for supplementing n-3 polyunsaturated fatty acids are being replaced to the plant-natured oils. This study was designed to investigate the effect of perilla seed oil on development of nonalcoholic steatosis. Male C57BL/6J mouse (5 weeks old) were fed with 45% high-fat diet for 90 days to develop nonalcoholic steatosis and metabolic disorder, and the mice were administered orally with perilla oil or palm oil (200 or 1,000 mg/kg/day) for 90 days. There were any differences on food intake and body weight between perilla oil and palm oil groups. Perilla oil-treated mice significantly lowered serum cholesterol level compared to the palm oil-treated or untreated mice. Treatment with perilla oil for 90 days significantly reduced hepatocellular lipid droplet and triglyceride accumulation compared to palm oil-treated or untreated mice. Palm oil- or perilla oil-treated mice significantly increased the protein levels and the phosphorylation of AMP-activated protein kinase and acetyl-CoA carboxylase in the liver compared to untreated mice, with significantly higher levels in the perilla oil-treated mice. In addition, treatment with perilla oil increased the adipose triglyceride lipase protein level in the liver, whereas decreased the protein levels of fatty acid synthase. Finally, perilla oil-treated mice presented less aortic lipid accumulation compared to palm oil-treated or untreated mice at 90 days after high-fat diet feeding. These results indicate that perilla seed oil attenuates the development of nonalcoholic steatosis and aortic lipid accumulation induced by high-fat diet, and this effect is mediated by the enhanced lipolytic activity in the liver.

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Aging

A Preliminary Study on Age Estimation from Cranial Suture Closure in a Thai Population

Pasuk Mahakkanukrauh[1,2,3,*], Sittiporn Ruengdit[1], Sukon Prasitwattanseree[1,2,4], Karnda Mekjaidee[1,2,5], Apichat Sinthubua[1,2,3]

[1]Forensic Osteology Research Center, Faculty of Medicine, Chiang Mai University, 50200, Thailand

[2]Excellence in Osteology Research and Training Center)ORTC(, Chiang Mai University, Chiang Mai, 50200, Thailand.

[3]Department of Anatomy, Faculty of Medicine, Chiang Mai University, 50200, Thailand

[4]Department of Statistics, Faculty of Science, Chiang Mai University, 50200, Thailand

[5]Department of Forensic Medicine, Faculty of Medicine, Chiang Mai University, 50200, Thailand

*Corresponding author: E-mail: pasuk034@gmail.com

E-mail addresses: 4805236@gmail.com (Ruengdit S.), sprasitwattanaseree@gmail.com (Prasitwattanaseree S.), turquoise0401@gmail.com (Mekjaidee K.), apichat_nine@yahoo.com (Sinthubua A.)

Age estimation is a vital and challenging method in constructing a biological profile of unknown skeletal remains. Cranial suture closure have been widely used and studied as age indicator for a long time, in contrast, its reliability is still debated. However, anthropologists have attempted to improve and develop this method because skull is usually found in forensic contexts. Various literatures reported that closure of cranial sutures is population specific. In addition, there is no standard aging method evaluating cranial suture closure in a Thai population. Consequently, this study aims to examine Thai cranial suture closure by using Meindl and Lovejoy scoring method. Closure of 48 suture positions including vault, lateral-anterior, facial, maxillary, sphenomaxillary and endocranialsutures was investigated. Principle Component Analysis revealed that two components derived from the suture closure scores can separate between a group of individuals under 40 years old and individuals aged over 40 years old. These two components composed of the scores evaluated from both ectocranial sutures (pterion and transverse palatine suture) and endocranial sutures (coronal bregmatica, coronal pterica, lambdoidal intermediate, and lambdoidalasterica). The result indicated that a group of particular sutures, using together, can deliveran efficient age classification. This preliminary study suggested that cranial suture closure could be applied as age indicator in Thais. Hence, further study is needed to establish a standard aging method using cranial suture closure in this population.

Keywords: Aging method, Cranial suture, Skull, Thais, Principle Component Analysis

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Poster Abstract
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Stem Cells Research

Shape control of tenogenesis

Fang-Yu Lo, Yang-Kao Wang
Department of Cell Biology and Anatomy College of Medicine, National Cheng Kung University

Tendon tissues are highly prone to injury, often resulting in the development of tendinopthy. Therefore, tendon regeneration has emerged as promising therapeutic strategy for tendinopathy. The classic method to control tendon cell differentiation is the application of soluble factors to wound site for healing and induction of tendon repair. In addition to these soluble cues, growing evidence has implied physical cues, such as geometric control is capable of directing stem cell commitment to tendon lineage. One of the methods to control cell geometry is micropatterning, which has been widely used to control cell shape to regulate stem cell fate. Interestingly, tenocyte reside within a niche that comprises parallel collagen fibers, suggesting a possible geometrical control in tenogenesis. In this regard, previous studies have demonstrated that well-aligned chitosan-based ultrafine fibers induce teno-lineage differentiation successfully. However, the molecular mechanisms underlying how cell shape regulates tenogenesis remain largely unknown. We hypothesized that changes of cell shape regulate tenogenesis. We performed tendon stem progenitor cells (TSPCs) to establish tenogenesis in extracellular matrix-coated line patterning to investigate tenogenesis by different geometric controls. From our preliminary data, we found that TSPCs exhibited proliferation ability and surface markers of mesenchymal cells. These TSPCs grown on 20-100/50-100 μm line pattern expressed tendon markers scleraxis and tenomodulin without soluble factor stimulation. This study suggests that the geometry of TSPCs may control the tenogenesis without the collaboration of soluble factors.

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Cancer Biology

Acetylshikonin inhibits invasion of chronically inflamed oral squamous cell carcinoma cells

Da Jeong Kim, Bong Hae Cho, Bok Hee Woo, Jeom Il Choi, Ji Hye Lee, Hae Ryoun Park
School of Dentistry, Pusan National University, South Korea

Periodontitis is the most common chronic inflammatory condition occurring in the human oral cavity, and Porphyromonas gingivalis is known as a major pathogen of chronic periodontitis. We tried to simulate chronic inflammation by repeatedly infecting oral squamous cell carcinoma (OSCC) cells with P gingivalis. Repeated infection of OSCC YD10B cells by P. gingivalis resulted in decreased expression of epithelial cell markers, but levels of CD44 and CD133, well-known cancer stem cell markers, were increased by chronic infection with P. gingivalis. The prolonged exposure to P. gingivalis also promoted migratory and invasive properties of OSCC cells, all of which are described as cellular characteristics undergoing EMT. In an effort to delineate the molecular players that control the invasiveness, we first assessed the level of various MMPs as well as IL-8, a well-known inflammatory cytokine, in P. gingivalis-infected YD10B cells. MMP-2 and IL-8 secretion were substantially increased. When IL-8 was directly applied to YD10B cells, their invasive ability and MMP level were significantly increased. Furthermore, knockdown of IL-8 in P. gingivalis-infected YD10B cells attenuated their invasive potentials and MMP levels. Taken together, our findings strongly suggest that chronic inflammation by P. gingivalis infection plays an important role in the promotion of the invasive potential of OSCC cells via the upregulation of IL-8 and MMPs. Acetylshikonin, a shikonin derivative from Lithospermum erythrorhizon, has been suggested as a potential chemotherapeutic agent in addition to their traditional anti-inflammatory use. In this study, we investigated the usefulness of acetylshikonin as a treatment regimen for oral cancers by observing inhibitory function of acetylshikonin in chronic inflammation-induced increased invasiveness of oral cancer cells and the involved mechanisms. Here, we report that Acetylshikonin significantly inhibited the P. gingivalis-induced invasion of human OSCC cells by downregulating MMP expression. In summary, our data suggest that acetylshikonin is a strong candidate for use as a selective chemotherapeutic agent for the treatment of OSCC.

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Neuroscience

High-fat diet downregulates expression of molecules involved in astrocytic lactate metabolism without impairing memory in the hippocampus

Sheng-Feng Tsai, Yu-Min Kuo
College of Medicine, National Cheng Kung University, Taiwan

High-fat diet downregulates expression of molecules involved in astrocytic lactate metabolism without impairing memory in the hippocampus Sheng-Feng Tsai1and Yu-Min Kuo1,2 1Institute of Basic Medical Sciences, College of Medicine, National Cheng Kung University, Tainan, Taiwan 2Department of Cell Biology and Anatomy, College of Medicine, National Cheng Kung University, Tainan, Taiwan Long-term high-fat diet (HFD) disturbs the energy metabolism and impairs the functions of brain. The lactate released from the astrocytes is known to fill the increased energy needs during neurotransmission. The lactate shuttled between astrocytes and neurons is essential to the hippocampal memory function. However, whether HFD effect the hippocampal astrocyte-neuron lactate shuttle (ANLS) and the role of ANLS in hippocampal memory functions of high-fat fed animals remain unclear. In this study, we fed male C57BL/6 mice with 12w HFD since their ages of 8w, and their hippocampal ANLS and memory functions were determined. Our results showed that HFD increased the body weight gain and induced insulin resistance by elevating fasting glucose and insulin. The hippocampal expression of astrocytic ANLS initiators, GLAST and GLT-1, and connexin-43, the astrocytic gap junction governing energy metabolites transport between astrocytes, was lower in HFD mice than control ones. Moreover, compared to the control group, the HFD mice had lower content level of lactate in the hippocampus. The regression and correlation analysis revealed that a significant positive correlation existed between the hippocampal lactate content level and GLAST/GLT-1 expression. The downregulated expression of astrocytic ANLS components was not caused by the hippocampal astrocyte loss which was determined by GFAP immunostaining. Although HFD altered ANLS in the hippocampus, we found the hippocampus-associated learning and memory functions were intact in HFD mice. Besides, the neuronal expression of molecules involved in lactate transport and synaptic plasticity was not changed by HFD. In conclusion, 12w HFD impaired the ANLS without hampering the memory functions in the hippocampus, and the astrocytes seemed to be more vulnerable to HFD than neurons.

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Cancer Biology

Anticancer potential of L-amino acid oxidase isolated from the venom of Crotalus mitchellii Pyrrhus

Kok Keong, Tan; Boon Huat, Bay; Gopalakrishnakone, Ponnampalam
Department of Anatomy, Faculty of Medicine, National University of Singapore, Singapore

Snake venom L-amino acid oxidase (svLAAO) catalyzes the oxidative deamination of L-amino acid (LAA) into the corresponding α-keto acid, releasing hydrogen peroxide (H2O2) as a by-product. In the present study, we investigated the anticancer potential of LAAO isolated from Crotalus mitchellii Pyrrhus (LAAOcmp) using LNCaP prostate cancer cell line as the model. We found that LAAOcmp reduces LNCaP viability in a concentration-dependent manner. As caspase-3 activity and the expression of cleaved caspase-9 and cleaved PARP were increased upon treatment with LAAOcmp, suggesting that cell death was mediated by apoptosis. Flow cytometry analysis of cell cycle also revealed abnormal DNA content wherein the G1 peak was distorted, suggesting cell cycle arrest. DNA laddering as demonstrated by gel electrophoresis showed that DNA fragmentation occurred in a time-dependent manner during LAAOcmp treatment. LNCaP treated with LAAOcmp had stronger fluorescence indicating increased ROS formation, resulting from oxidation of H2DCF. It would appear that an increase in oxidative stress is the cause of cytotoxicity. On the other hand, LAAOcmp could also induce a slow-acting cytotoxic effect on LNCaP by depleting LAA in the culture medium. Our findings suggest that svLAAO is a potential anticancer therapeutic.

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Cancer Biology

L-ascorbic Acid Can Abrogate SVCT2-dependent Cetuximab Resistance Mediated by Mutant KRAS in Human Colon Cancer Cells

Ji Hee Gong[a,b,*], Jai-Hee Moon[a,b,*], Yu Jin Shin [a], Jae-Sik Shin[a,b] , Eun Young Lee[a], Seul Lee[a,b], Hyun Jae Shim[a], Jimin Shin[a] , Seung-Mi Kim[a], Bonghyun Park[a,b], Joonyee jeong[a,b], Dong-Hoon Jin[a,b,#]
[a]Asan Institute for Life Science, [b]Department of Convergence Medicine,
University of Ulsan College of Medicine, Asan Medical Center, 86 Olympicro-43gil, Songpa-gu, Seoul, Republic of Korea
#Corresponding author: inno183@amc.seoul.kr (Dong-Hoon Jin)

Colon cancer patients with mutant KRAS are resistant to cetuximab, an antibody directed against the epidermal growth factor receptor, which is an effective clinical therapy for patients with wild-type KRAS. Numerous combinatorial therapies have been tested to overcome the resistance to cetuximab. However, no combinations have been found that can be used as effective therapeutic strategies. In this study, we demonstrate that L-ascorbic acid partners with cetuximab to induce killing effects, which are influenced by sodium-dependent vitamin C transporter 2 (SVCT-2) in human colon cancer cells with a mutant KRAS. L-ascorbic acid treatment of human colon cancer cells that express a mutant KRAS differentially and synergistically induced cell death with cetuximab in a SVCT-2-dependent manner. The ectopic expression of SVCT-2 induced sensitivity to L-ascorbic acid treatment in human colon cancer cells that do not express SVCT-2, whereas the knockdown of endogenous SVCT-2 induced resistance to L-ascorbic acid treatment in SVCT-2-positive cells. Moreover, tumor regression via the administration of L-ascorbic acid and cetuximab in mice bearing tumor cell xenografts corresponded to SVCT-2 protein levels. Interestingly, cell death induced by the combination of L-ascorbic acid and cetuximab resulted in both apoptotic and necrotic cell death. These cell death mechanisms were related to a disruption of the ERK pathway and were represented by the impaired activation of RAFs and the activation of the ASK-1-p38 pathway. Taken together, these results suggest that resistance to cetuximab in human colon cancer patients with a mutant KRAS can be bypassed by L-ascorbic acid in an SVCT-2-dependent manner. Furthermore, SVCT-2 in mutant KRAS colon cancer may act as a potent marker for potentiating L-ascorbic acid co-treatment with cetuximab.

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Cancer Biology

ABT-263 (Navitoclax) induces cell death in MDA-MB-231 but not in MCF-7 selectively and synergistically induces apoptosis with RAD001 (Everolimus) in MCF-7 human breast cancer cells

Eun Young Lee, Bonghyun Park, Hyun Jae Shim, Jimin Shin, Jae-Sik Shin, Jai-Hee Moon, Yu Jin Shin, Seul Lee, Seung-Mi Kim, Ji Hee Gong, Joonyee jeong, Dong-Hoon Jin
Asan Medical Center, University of Ulsan College of Medicine, Seoul, Korea

ABT-263 (Navitoclax), which is a Bcl-2 family protein inhibitor, was tested as an anti-cancer agent in clinic. However, the clinical trials were very limited and the mechanism of action was not fully understood. In present study Navitoclax showed dramatic apoptotic effects in MDA-MB-231 breast cancer cells time- and dose-dependently. Another type of breast cancer cells, MCF-7 has resistant effect to Navitoclax differently. More specially, Bcl-2 family proteins including Bcl-2, Bcl-w, Bcl-xL and Mcl-1 have been shown no changes with treatment of Navitoclax in both breast cancer cells, MDA-MB-231 and MCF-7. But RIF was decreased only in MDA-MB-231 dose-dependently. RIF is a member of ROI and which is controlled by mTOR. The combination treatment to MCF-7 cells with Navitoclax and Everolimus (mTOR inhibitor) and using siRNA-mediated mTOR knockdown showed apoptotic effects by control of RIF stability. Together, these findings suggest that Navitoclax induces apoptosis in MDA-MB-231 breast cancer cells through the inhibition of RIF and combi-treatment with Everolimus make synergic apoptotic effect in MCF-7 by RIF stability.

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Cancer Biology

Combined treatment with vitamin C and sulindac synergistically induces p53- and ROS-dependent apoptosis in human colon cancer cells

Joonyee jeong, Yu Jin Shin, Jai-Hee Moon, Jae-Sik Shin, Seung-Mi Kim, Ji Hee Gong, Eun Young Lee, Seul Lee, Hyun Jae Shim, Jimin Shin, Bonghyun Park, Dong-Hoon Jin
Asan Medical Center, University of Ulsan College of Medicine, Seoul, Korea

Sulindac has anti-neoplastic properties against colorectal cancers; however, its use as a chemopreventive agent has been limited due to toxicity and efficacy concerns. Combinatorial treatment of colorectal cancers has been attempted to maximize anti-cancer efficacy with minimal side effects by administrating NSAIDs in combination with other inhibitory compounds or drugs such as L-ascorbic acid (vitamin C), which is known to exhibit cytotoxicity towards various cancer cells at high concentrations. In this study, we evaluated a combinatorial strategy utilizing sulindac and vitamin C. The death of HCT116 cells upon combination therapy occurred via a p53-mediated mechanism. The combination therapeutic resistance developed in isogenic p53 null HCT116 cells and siRNA-mediated p53 knockdown HCT116 cells, but the exogenous expression of p53 in p53 null isogenic cells resulted in the induction of cell death. In addition, we investigated an increased level of intracellular ROS (reactive oxygen species), which was preceded by p53 activation. The expression level of PUMA (p53-upregulated modulator of apoptosis), but not Bim, was significantly increased in HCT116 cells in response to the combination treatment. Taken together, our results demonstrate that combination therapy with sulindac and vitamin C could be a novel anti-cancer therapeutic strategy for p53 wild type colon cancers.

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Aging

Foxp3 is a Key Downstream Regulator of p53-mediated Cellular Senescence

Seul Lee, Jae-Sik Shin, Jai-Hee Moon, Yu Jin Shin, Seung-Mi Kim, Eun Young Lee, Ji Hee Gong, Hyun Jae Shim, Jimin Shin, Joonyee jeong, Bonghyun Park, Dong-Hoon Jin
Asan Medical Center, University of Ulsan College of Medicine, Seoul, Korea

The downstream events and target genes of p53 in the process of senescence are not fully understood. Here, we report a novel function of the forkhead transcription factor Foxp3, which is a key player in mediating T-cell inhibitory functions, in p53-mediated cellular senescence. The overexpression of Foxp3 in mouse embryonic fibroblasts (MEFs) accelerates senescence, whereas Foxp3 knockdown leads to escape from p53-mediated senescence in p53-expressing MEFs. Consistent with these results, Foxp3 expression resulted in the induction of senescence in epithelial cancer cells, including MCF7 and HCT116 cells. Foxp3 overexpression also increased the intracellular levels of reactive oxygen species (ROS). The ROS inhibitor N-acetyl-L-cysteine rescued cells from Foxp3-expression-induced senescence. Furthermore, the elevated ROS levels that accompanied Foxp3 overexpression were paralleled by an increase in p21 expression. Knockdown of p21 in Foxp3-expressing MEFs abrogated the Foxp3-dependent increase in ROS levels, indicating that Foxp3 acts through the induction of p21 and the subsequent ROS elevation to trigger senescence. Collectively, these results suggest that Foxp3 is a downstream target of p53 that is sufficient to induce p21 expression, ROS production and p53-mediated senescence.

Poster Abstract
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Aging

Mutual relationship among perineal muscles; the bulbospongiosus, external anal sphincter, and superficial transverse perineal muscle

Satoru MURO, Phichaya BARAMEE, Janyaruk SURIYUT, Hisayo NASU, Masayo HARADA, Kumiko YAMAGUCHI, Keiichi AKITA
Department of Clinical Anatomy, Tokyo Medical and Dental University, Japan

It is generally accepted that perineal muscles attach at the perineal body in males and females. However, during dissection processes, it is pretty difficult to expose the perineal body and clearly identify the border of each perineal muscles. The aim of this study is to reinvestigate the perineal muscles especially the bulbospongiosus, the external anal sphincter, and the transverse perineal muscles. Eighteen formalin embalmed cadavers (9 males and 9 females, mean: 76.1 y/o) were used for macroscopic and histological examinations. The bulbospongiosus were adjoined to the lateral surface of the external sphincter and ran anteriorly to cover the bulb (of the penis in males or of the vestibule in females). In male specimens, some muscle fibers from the anteroinferior region of the external anal sphincter in the midsagittal plane extended anteriorly, and attached to the bulbospongiosus. However, this midsagittal attachment was not observed in females, although a lot of textbooks describe this attachment as the perineal body. In addition, the transverse perineal muscles broadly connected to the lateral surfaces of the bulbospongiosus and the external anal sphincter both in males and females, although a lot of textbooks describe the insertions of the transverse perineal muscles as the perineal body. In conclusion, the perineal body as the central tendinous attachment of the perineal muscles is not clearly identified. Each muscle of the perineal muscles is not shown as an independent muscle. Therefore, these perineal muscles should be considered to be morphologically continuous such as one muscle sheet.

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Developmental Biology

Molecular Insights on Cyclopia

Karthik Harve, Omid Iravani, Francis Voon Chee Tet, Boon Huat Bay
Yong Loo Lin School of Medicine, National University of Singapore, Singapore

Cyclopia is a culmination of a genetic insult during embryogenesis, due to possible teratogenic effects on the human genome, that leads to fatal developmental anomalies in the brain (alobar holoprosencephaly), eye (synophthalmia), midline facial structures, including the nose (proboscis), oral cavity (cleft lip/palate) and mandible (micrognathia), with a plethora of extracranial defects involving the heart, renal and gastrointestinal systems. . The 'cyclops' strikingly mentioned in the Greek epic Odyssey by Homer (800-700 BC) were legendary gigantic creatures that battled the Olympian gods. They were described as being single-eyed and macrosomic, and these are also characteristics of cylopic fetuses born to diabetic mothers. Several studies have shown that teratogens which target Sonic Hedgehog (SHH) signaling are able to induce cyclopia. Sonic hedgehog (SHH) is expressed in the neural ectoderm and notochord, both of which contribute to the development of the brain, eyes and face. We present here a review of the molecular mechanisms which point to potential genetic events that ultimately determine the phenotypic presentation of this developmental anomaly. Molecular insights can further help in the application of more effective screening diagnostic tools for early detection of this syndrome.

Poster Abstract
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Developmental Biology

Genetic approach for studying Kinesin family member 7 (Kif7) in mouse enteric nervous system (ENS) development

Tingwen ZHOU, Chi-Chung HUI, Elly Sau-Wai NGAN
Department of Surgery, LKS Faculty of Medicine, The University of Hong Kong, Hong Kong

Kinesin family member 7 (Kif7) is a motor protein regulating Hedgehog signalling transduction in cilium. Conditional deletion of Kif7 in neural crest cells (NCCs) interfere the growth and survival of mice, closely associated with colonic motility defect. Deletion of Kif7 disrupted neuronal differentiation and proliferation of enteric NCCs (ENCCs) at early developmental stages, accompanied by disorganization of nerve network in embryonic gut. Subsequent immunohistochemistry analyses with various glial and neuronal markers revealed the presence of giant myenteric ganglion in mutant gut. Moreover, a dramatic reduction in nNOS + inhibitory neurons in the myenteric plexuses of distal small intestine and colon, deficiency of submucosal plexuses and reduced neuronal/glial projections in intestinal villi were observed in mutants. All these data suggested that Kif7 regulates the formations of both myenteric and submucosal plexuses, probably also neuronal subtypes. Aberrant ganglion formation and loss of nNOS+ neurons may account for the gut motility defects in the mutants. To delineate the underlying molecular mechanism, microarray analysis was performed with control and Kif7KO ENCCs, where genes regulating the proliferation and differentiation of ENCC were found down-regulated in Kif7KO ENCCs, implying that loss of Kif7 interrupts the regulatory networks of these molecules and interferes the development of ENS.

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Developmental Biology

Human Neural Crest Model for Studying Vinculin in Enteric Nervous System Development and Hirschsprung Disease Pathogenesis

Reeson Xu WANG, Frank Pui-Ling LAI, Cynthia Sin-Ting LAU, Elly Sau-Wai NGAN
Department of Surgery, LKS Faculty of Medicine, The University of Hong Kong, Hong Kong

Hirschsprung Disease (HSCR) is a congenital disease characterized by the lack of ganglion cells in the colon. Most of the patients only present with mild phenotype with a short segment of the colon affected, namely S-HSCR. A novel mutation in Vinculin (VCL M209L) was recently identified in a S-HSCR patient. VCL is an adaptor protein in focal adhesion (FA), which is responsible for cell migration or axonal guidance. M209L mutation resides in the head domain of VCL and that may interfere the protein conformation. Overexpressions of wild-type VCL and VCL M209L constructs in HeLa cells showed that M209L mutation impairs FA assembly by reducing the interactions of VCL with its binding partners. Intriguingly, FA phenotype was nicely recapitulated in patient-specific iPSC-derived neural crest cells (NCCs) carrying VCL M209L mutation, where FA size was found significantly reduced. More importantly, iPSC-NCCs exhibited defects in migration and differentiation. With CRISPR-Cas9 genome editing system, correction of VCL M209L mutation in patient-specific iPSCs rescued FA phenotypes and restored the cellular functions of iPSC-NCCs. Taken together, our data suggest that VCL M209L mutation may account of HSCR phenotype and its roles in the development of enteric nervous system will be further studied using mouse model.

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Developmental Biology

Correlation between Interleukin-17 Serum and Goiter Gradation of Graves' disease patients

Dwitya Elvira, Eryati Darwin
Internal Medicine Department of Medical Faculty of Andalas University, Indonesia

Graves' disease (GD) is a thyroid autoimmune disease that systemically affect human metabolism. Graves' disease characterized as hyperthyroidism, diffuse goiter, opthalmopathy and/ dermopathy, increasing thyroid hormone (thyroxin), decrease of thyroid stimulating hormone (TSH) and increasing of autoantibody thyroid (TRAb). Etiology of GD remains elusive but imbalance of T-helper cells, regulatory T cells and cytokines were accused to cause this disease. The identification of new subpopulation of T helper (Th-17) produce IL-17 (Interleukin-17), that also crucial in development of autoimmune disease, particularly Graves' disease. Gradation of goiter were divide into three classifications: grade I, grade II and grade III. Aim of this study is to correlate between serum of IL-17 cytokine with goiter gradation of Graves' disease patients. Thirty patients diagnosed as Graves' disease based on clinical and laboratory measurement were included as object of this study. Age, gender, goiter gradation, thyroid function status was measured and noted as baseline characteristic of this study. Goiter gradation classified as three classifications: grade I, grade II and grade III. Serum of IL-17 were measured using ELISA method. As a result of this study, we found increasing of IL-17 cytokine serum compared to control, with mean values 11,961,73 pg/mL in patient group, and 7,477,07 pg/mL in control group. Goiter gradation found mostly in grade II (50%) followed by grade I and grade III. There is no correlation between IL-17 serum with goiter gradation. From this study we can conclude that there is no correlation between cytokine IL-17 serum with goiter gradation in Graves' disease patients but we found increasing of interleukin-17 serum in Graves' disease patients that suggested that Th-17 cells can play a central role in pathogenesis of Graves' disease.

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Developmental Biology

Effect of Fixed Orthodontic Appliance on the Levels of Matrix Metalloproteinase-8 in Gingival Crevicular Fluid

Eryati Darwin* and Aida Fitriana**
*Faculty of Medicine, Andalas University, ** Faculty of Dentistry, Andalas University, Padang-Indonesia
Email: eryatidarwin@fk.unand.ac.id

Fixed orthodontic therapy is the preferred therapeutic modality for treatment of malocclusions that affect mastication and facial appearance. Nevertheless Its may cause subgingival microbial composition, promotion of plaque development and soft tissue injuries. This mechanical injury can lead to development of inflammatory reactions in periodontal tissues, which promote tooth movement and biological processes, resulting in bone resorption. Matrix metalloproteinases (MMP) are thought to be responsible for the turnover and degradation of the extracellular matrix that act on pro-inflammatory cytokines. Matrix metalloproteinase-8 (MMP-8) is a member of MMP family of enzymes is seen in almost every human tissue in which inflammation is present.
The aim of this study was to assess effects of fixed orthodontic therapy on the level of matrix metalloproteinase
Twenty healthy patients who treated with a straight wire technique using brackets on the maxillary and the mandibular arches were divided into two group; one group with less than six months treated, and the other group with more than six months treated. Salivary samples were taken from gingival crevicular fluid and its MMP-8 level were determine using ELISA method. This study was approved by Research Ethic Committee.
Our study shows that the level of MMP-8 in group using more than six months fixed orthodontic appliance were higher than in group using less than six months with statistically significant differences ($p<0,05$) .
From our study, we conclude that orthodontic appliance may induced inflammatory process on the gingival tissue

Keywords: fixed orthodontic, inflammation, MMP-8

Poster Abstract
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Neuroscience

Chrysoeriol Induces Apoptosis in Rat C6 Glioma Cells

Suppanut Wongkularb[1], Patoomratana Tuchinda[2], Sukumal Chongthammakun[3]*
[1] Molecular Medicine Graduate Program, Faculty of Science, Mahidol University, Bangkok 10400, Thailand.
wi2012hola@gmail.com
[2] Department of Chemistry, Faculty of Science, Mahidol University, Bangkok 10400, Thailand.
patoomratana.tuc@mahidol.ac.th
[3] Department of Anatomy, Faculty of Science, Mahidol University, Bangkok 10400, Thailand.
sukumal.cho@mahidol.edu
* Correspondence: sukumal.cho@mahidol.edu

Glioblastoma multiforme (GBM) is the most common and most aggressive cancer that begins within the brain. Currently, the novel therapeutic strategies to target and kill GBM cells are desperately needed. Chrysoeriol is a flavonoid compound found in several tropical medicinal plants such as Alfafa seeds, Eurya cilliata leaves or rice seeds. It has been shown to possess antiinflammatory and antioxidant activities. However, only a few studies have reported its anticancer effects. This study assessed the in vitro anticancer activity of chrysoeriol extracted from *Phyllanthus niruri* against rat C6 glioma cell and its possible mechanism. We prepared different doses of chrysoeriol, 0-20 μM, and evaluated their ability to inhibit the proliferation of C6 glioma cells by using the MTT assay. The low doses of chrysoeriol (1.25-5 μM) for 24 h had no effect on cell viability but the high doses of chrysoeriol (10-20 μM) for 24 h had significantly reduced cell viability ($p < 0.05$) compared to control. The expression of pro-apoptotic Bax protein and anti-apoptotic Bcl-2 protein was evaluated by Western blotting of the protein levels and real-time quantitative PCR (RT-qPCR) measurement of mRNA levels. Chrysoeriol at a concentration of 2 μM upregulated the expression of Bax and downregulated the expression of Bcl-2. The Bax/Bcl-2 protein ratio was significantly increased in 2 μM-treated group ($p < 0.05$). However, the expression of Bax/Bcl-2 mRNA ratio did not reach statistical significance. These results demonstrate that the anticancer effect of chrysoeriol is associated with the induction of apoptosis. Chrysoeriol may have interesting potential in cancer chemoprevention and therapy.

Keywords: chrysoeriol; glioma; apoptosis

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Cancer Biology

Downregulation of Peroxiredoxin III modulates expression of microRNAs in breast cancer in vitro

Ow Suet Hui, Chua Pei Jou, Bay Boon Huat
Yong Loo Lin School of Medicine, National University of Singapore, Singapore

Peroxiredoxin III (Prx III) is an antioxidant enzyme that is exclusively localised in mitochondria. It contains an active cysteine which is responsible for the reduction of reactive oxygen species (ROS) such as H2O2, enabling the maintenance of mitochondrial homeostasis. A class of non-coding RNA known as microRNA (miRNA) has been known to negatively regulate gene expressions by mRNA degradation or translation inhibition. Multiple studies have reported aberrant PRXs and miRNAs expressions in various cancers, including breast cancer. In this study, we investigate the relationship between Prx III and miRNAs in breast cancer in vitro. Comprehensive miRNA profiling using Affymetrix miRNA 4.0 array detected a total of 6621 miRNAs in which 40 miRNAs were differentially expressed between Prx III knockdown and control in MDA-MB-231 breast cancer cells. Among the differentially expressed miRNAs, five were up-regulated and 35 were down-regulated. Based on Transcriptome Analysis Console (TAC) and DIANA-miRPath v3.0, the enriched KEGG pathways for the up-regulated and down-regulated clusters of miRNA were identified. Experimental validations of some of the miRNA targets in the dataset were carried out. It would appear that Prx III can mediate cellular processes via miRNAs.

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Cancer Biology

Tumor suppressor TP53INP1 negatively regulates ERK1/2 via DUSP10 to promote metastasis in hepatocellular carcinoma

Kai Yu Ng, Stephanie Ma
School of Biomedical Sciences, LKS Faculty of Medicine, The University of Hong Kong

Frequent tumor recurrence and high incidence of metastasis in hepatocellular carcinoma (HCC) contribute to its poor prognosis. We have previously found that the initiation, growth and self-renewal of CD133+ liver tumors are regulated by the balance of miR-130b overexpression and tumor protein 53 inducible nuclear protein 1 (TP53INP1) down-regulation, suggesting that TP53INP1 is a critical effector driving hepatocarcinogenesis. However, its role in metastasis is not known. Current study with a paired primary/metastatic HCC tissue microarray found a significantly lower TP53INP1 expression in metastatic tissues as compared to primary HCC counterparts. Functional studies in HCC cell lines MIHA and MHCC-97L with TP53INP1 stably suppressed demonstrated an enhanced ability to migrate and invade. In vivo orthotopic animal studies also confirmed this phenomenon with an enhanced ability of TP53INP1 suppressed cells to metastasize to the lung. To elucidate the downstream signalling mechanism by which TP53INP1 regulates HCC metastasis, a Proteome Profiler Human Phospho-Kinase Array was utilized to compare the differential phosphorylated protein profile in HCC cells with or without TP53INP1 suppressed. Phospho-ERK was found to be consistently upregulated in HCC cell lines with TP53INP1 knocked down, with its involvement in TP53INP1-mediated metastasis subsequently validated by rescue experiments using an ERK kinase inhibitor (U0126) or shERK1/2 knockdown approach. ERK1/2 is known to be negatively regulated by a family of dual-specificity MAPK phosphatase called DUSP/MKP, and screening of a panel of DUSP/MKP family members by qPCR identified DUSP10 to be commonly down-regulated in HCC cell lines with TP53INP1 suppressed. The important role of DUSP10 was also confirmed by rescue experiments whereby DUSP10 overexpressed in TP53INP1 knockdown cells reversed the upregulated phospho-ERK expression and enhanced aggressiveness of HCC cells. Further analysis of the DUSP10 promoter region by open-access database (JASPAR) showed 4 putative binding sites for p73, a known cofactor of TP53INP1. Luciferase assay demonstrated that both TP53INP1 and p73 are vital for DUSP10 promoter activities. Taken together, TP53INP1 negatively regulates HCC metastasis by cooperating with p73 to alter expression and activities of DUSP10 and ERK.

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Advanced Biomedical Platforms

Toxicity of Zinc Oxide Nanoparticles in *Drosophila Melanogastor*

Cheng Teng Ng[1,3§], Liang Qing Yong[1§], Choon Nam Ong[2], Liya Yu[3], Boon Huat Bay[1] and Gyeong Hun Baeg[1*]
[1] Department of Anatomy, 4 Medical Drive, Yong Loo Lin School of Medicine, National University of Singapore, Singapore 117597, Singapore
[2] NUS Environmental Research Institute, 5A Engineering Drive 1, National University of Singapore, Singapore 117411, Singapore
[3] Department of Civil and Environmental Engineering, 1 Engineering Drive 2, National University of Singapore, Singapore 117576, Singapore

ZnO NPs have been widely used in consumer products. However, there has been an increasing number of reports on ZnO NP-mediated toxicity. Here, we investigated the toxicological profiles of ZnO NPs in the fruit fly *Drosophila*. In *Drosophila* model, we first demonstrated that ZnO NPs were ingested and accumulated in the gut. ZnO NPs exposure caused a significant decrease in the viability and delay in the development of *Drosophila*, in a dose-dependent manner. There was also induction of ROS in the gut of the *Drosophila*, suggesting that the decreased viability was highly associated with ROS generation. Taken together, this study suggests that ZnO NPs can induce a significant ROS-related toxicity in *Drosophila*. More extensive studies would be needed to verify the safety issues related to the increased usage of ZnO NPs by consumers.

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Cancer Biology

Targeting ANXA3 in combination with sorafenib for the treatment of hepatocellular carcinoma

Man TONG, Steve LUK, Jin DING, Terence LEE, Stephanie MA
School of Biomedical Sciences, LKS Faculty of Medicine, The University of Hong Kong

Sorafenib is the only FDA-approved tyrosine kinase inhibitor for targeted therapy in advanced HCC. Nevertheless, its efficacy is limited with only a modest improvement in patient outcome, likely due to acquired resistance. In-depth understanding of the molecular mechanism of sorafenib resistance is warranted for the development of novel treatment strategies. Recent studies by us and others have characterized liver tumor-initiating cells (T-ICs) to be a possible source of resistant and recurrent tumors and a plausible target for HCC treatment. Our group has previously identified CD133 to be a functional marker of liver T-ICs and found annexin a3 (ANXA3) to regulate cancer and stem cell-like properties in this subset of cells. Interestingly, our recent observations also found CD133+ liver T-ICs to be more resistant to sorafenib. Sorafenib resistant clones, established in HepG2 and Huh7 cells by continuous exposure to increasing concentrations of sorafenib, displayed enhanced abilities to migrate, invade, self-renew, and initiate tumor formation in immunodeficient mice, as well as higher expression of stemness associated genes. These two sorafenib resistant cell lines and two other sorafenib resistant HCC patient-derived xenografts established in a similar manner were also found to be enriched for CD133 and ANXA3 expression. Sorafenib resistant clones with ANXA3 stably suppressed were re-sensitized to sorafenib treatment and had diminished ability to migrate, invade, self-renew and initiate tumor growth in vivo, further substantiating the role of ANXA3 in mediating sorafenib resistance in HCC. Mechanistically, an activated PKC/ERK/FRA2 signaling axis was found to be responsible for driving this phenomenon. Clinically, ANXA3 expression was also found to have prognostic value as a higher ANXA3 expression in HCC patients who have received sorafenib treatment was correlated with poor overall survival. The combinatorial use of a homemade ANXA3 neutralizing antibody and sorafenib on HCC patient derived xenografts is now being investigated as a potential new treatment regimen for combating sorafenib resistance in HCC.

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Developmental Biology

The Correlation of Prolactin Level and Oxytocin with Duration of Amenorrhea Lactation in Exclusive Breastfeeding Women

Arni Amir[1], Eryati Darwin[2]
[1]Department of Biology
[2]Department of Histology
Faculty of Medicine, Andalas University, Indonesia

Prolactin and oxytocin were hormones that play important roles in lactation process. When mothers exclusive breastfeeding, it presses ovulation causing no occurrence of menstruation, called amenorrhea lactation. After six months breastfeeding, it would make the mother had higher risk for menstruation and pregnancy. The aim of this research is determining the correlation between prolactin and oxytocin level with duration of amenorrhea lactation in exclusively breastfeeding mothers.
The design of the research was cross-sectional, observing 48 exclusively breastfeeding mothers in Puskesmas Padang Belimbing, September 2015 - June 2016. Samples were selected based on cluster random sampling. The examination of prolactin and oxytocin level was conducted at Biomedical Lab Faculty of Medicine Unand with human prolactin ELISA Kit and Human oxytocin Elisa Kit. Shapiro-Wilk was applied for normality test of the data, and Spearman's correlation was applied for analyzing prolactin and oxytocin level with duration of amenorrhea lactation.
The result is that there is a weak positive correlation and significance between prolactin level and duration of amenorrhea lactation ($r=0.331$; $p=0.022$); and there is a very weak negative correlation and insignificance between oxytocin level with the duration of amenorrhea lactation ($r=-0.085$; $p=0.565$).
The higher prolactin level, the longer amenorrhea lactation duration and the higher oxytocin level, the shorter amenorrhea lactation period.

Keywords: Prolactin, Oxytocin, Duration of Amenorrhea Lactation, Exclusive Breastfeeding

Poster Abstract
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Neuroscience

Maternal diabetes alters the expression of miR-26b and its putative targets Mecp2 and Psd-95 proteins in mouse embryonic neural stem cells

Seshadri Ramya, Sukanya Shyamasundar, Bay Boon Huat and S. Thameem Dheen*
Department of Anatomy, Faculty of Medicine, National University of Singapore, Singapore

It is well established that maternal diabetes is a teratogen which predisposes the developing brain to structural and/or functional defects, including neuropsychological disturbances, deficits in sensory perception and attention. However, the exact mechanism underlying glucose-induced defects remains unknown. Recently, we have shown that maternal diabetes alters epigenetic mechanisms including chromatin modifications, DNA methylation and microRNA (miRNA) expression in embryonic neural stem cells (NSCs). miRNA expression profiling in NSCs revealed that miR-26b, which has been shown to play roles in neurogenesis and neuronal differentiation was significantly upregulated in NSCs from diabetic pregnancy when compared to the control. Among the several predicted targets of miR-26b, methyl CpG binding protein2 (Mecp2) and post synaptic density 95 (Psd95) that are involved brain development and synaptic maturation and plasticity were selected for further study, since the expression levels of Mecp2 protein and Psd-95, were significantly downregulated in NSCs from diabetic pregnancy when compared to the control. Overexpression of miR-26b resulted in the downregulation of Mecp2 and Psd-95 proteins suggesting that they are putative targets of miR-26b. Taken together, this study reveals that maternal diabetes upregulates the expression of miR-26b resulting in downregulation of Mecp2 and Psd-95 proteins. Downregulation of expression level of Mecp2 and Psd-95 proteins that are crucial for brain development and synapse formation may underlie defective brain development and maturation, and manifest as neuropsychological disturbances that are observed in offspring of diabetic pregnancy.

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Cancer Biology

PRMT6-Dependent CRAF/ERK Signaling Regulates Cancer Stem Cell Plasticity in Liver Cancer

Lok Hei CHAN
School of Biomedical Sciences, Li Ka Shing Faculty of Medicine, The University of Hong Kong
Stephanie MA
School of Biomedical Sciences and State Key Laboratory for Liver Research, Li Ka Shing Faculty of Medicine, The University of Hong Kong

Liver cancer remains one of the most prevalent and deadliest cancer types in the world. Hepatocellular carcinoma (HCC) accounts for over 75% of all liver cancer cases. Contemporary challenge in treating HCC has been the common therapy resistance and recurrence after therapy, all of which have been reported to be associated with stem-like behavior of cancer cells. Elucidation of the mechanisms underlying these processes is fundamental for the development of new therapeutic treatments. Previous studies in our group and others have identified and characterized CD133+ cells in HCC as liver cancer stem cells (CSCs). By adopting a PCR array encompassing diverse human chromatin modifiers, protein arginine methyltransferase 6 (PRMT6) was found to be differentially down-regulated in the CD133+ subset of HCC cells. Consistently, PRMT6 was also preferentially down-regulated in liver chemoresistant hepatospheres as compared to its chemosensitive counterparts. Clinically, reduced PRMT6 expression was detected in HCC specimens and correlated with a higher risk of metastasis. Through lentiviral based gain and loss of function approach, PRMT6 was found to negatively regulate diverse in vitro cancer stem cell properties of HCC cells including clonogenicity, self-renewal, therapy resistance, metastasis and expression of CSC markers. In addition, PRMT6 also suppressed in vivo tumor initiation and serial transplantation. Surprising, contrary to its usual localization in the nucleus as a chromatin modification enzyme mediating histone H3R2 methylation, we found PRMT6 to be predominantly expressed in the cytoplasm in normal liver and HCC cells. Through tandem affinity purification and subsequent mass spectrometry profiling, we identified CRAF, a serine/threonine-protein kinase, as a novel cytoplasmic protein partner of PRMT6. Binding of PRMT6 to CRAF inhibited its kinase activity through site-specific arginine methylation, resulting in inhibition of ERK-mediated CSC plasticity in HCC. In summary, we found PRMT6 to regulate liver CSC plasticity via an unprecedented role in the cytoplasm through suppression of CRAF/ERK cascade.

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Advanced Biomedical Platforms

The Transition of Complex Periorbital Muscle Bundles with Their Adjacent Muscles

Natnicha KAMPAN[1], Itsuko OKUDA[2], Hisayo NASU[1], Kumiko YAMAGUCHI[3], and Keiichi AKITA[1]
[1]Department of Clinical Anatomy, Tokyo Medical and Dental University (TMDU), Japan
[2]Department of Diagnostic Radiology, International University of Health and Welfare, Mita Hospital, Japan
[3]Institute of Education, Tokyo Medical and Dental University (TMDU), Japan

Orbicularis oculi (OOc) is one of the facial muscles which various studies attempted to elucidate the anatomical variations. The malaris (Henle, 1858), the muscle surrounding OOc, is also considered to be consisted of the medial and lateral bundles. However, this complex muscle group has remained unclear. Hence, this study aimed to indicate the anatomical structures of the muscle bundles surrounding OOc, and investigate patterns of the attachments including the relations to the adjacent muscles by careful investigations of the fourteen hemi-faces of embalmed Japanese cadavers.
Medial and lateral bundles of OOc were found in all specimens. The medial bundles originated from the three different structures including the frontal process of maxilla, medial palpebral ligament, and depressor supercilii muscle, whereas the insertions were divided into three regions which were the inferior orbital part of OOc, the lateral bundle of OOc, and the adjacent muscles including the levator labii superioris alaeque nasi and levator labii superioris muscles. For the lateral bundle of OOc was found clearly originated from the superficial temporal fascia, while the insertions were noticed from the three regions including the medial bundle of OOc, zygomaticus minor, and zygomaticus major muscles. Remarkably, the reciprocal connection between the medial and lateral bundles of the OOc was obviously observed in all specimens.
Due to the varied variations and relations with adjacent muscles, these medial and lateral muscle bundles of OOc in this study were regarded as the transitional muscle bundles among the spherical muscle around the orbit, medial and lateral longitudinal muscles.

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Developmental Biology

The Effects of Nano Curcumin Toward Expression Head Shoch Protein 47 and Kidney Fibrosis Due to Unilateral Uteter Obstruction Rats

Yanwirasti Sofyan, Alvarino
Department of Anatomy, Faculty of Medicine, Andalas University, Indonesia

Upper urinary tract obstruction is one of the issue in urology, because it can occur in all phases of human life and can be located in all segmen of upper urinary trac. This condition lead to hydronephrosis which increased the pressure of intrahydrostaticren. As consequency. It will trigger.Molecul inflammation, such as TGF-β1. TGF-β1 will trigger the production of heat shock protein-47 (HSP-47) which produced synthesis of collagen. The result of this condition is renal fibrosis. Invitro, nanocurcumin has proved to decrease proinflamation cytokine. The aim of this research is to prove the effect of nanocurcumin toward expression of HSP-47 and kidney fibrosis due to unilateral ureter obstruction rats. Methods This experimental study used 34 male white rats (RattusNorvegicus) of 180-200 grams that were divided into two groups, control (I) and treated group (II). The rats were tied ureter unilaterally. The first group were given the solvent nanocurcumin, while the second group were given nanocurcumin for 28 days. After 28 days, relaparatomy were done and taken the cortex renal to examine expression HSP 47 and area fibrosis. HSP-47 were examined immunohistochemically and area of fibrosis were examined by using masson trachoma in histochemistry. Result The expression of HSP-47 in the kidney obstruction without administration ofnanocurcumin higher than which given nanocurcumin (22,29 ± 6,18: 13,06 ± 378). The statistical test shows there was significantly differences ($p < 0,05$). Rats which no given nanocurcumin had fibrosis area ≥ 80%,nwhile rats which given nanocurcumin had fibrosis area < 50%. The statical test shows, there was significantly differens ($p < 0,05$)
Conclusion
The result of study concluded that nanocurcuminsupress renal fibrosis by supress the expression of HSP-47 in rats with unilateral obstruction

Keyword: nanocurcumin, HSP-47, Kidney fibrosis

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Developmental Biology

Identification of Minangese Palatal Rugae Pattern

Nila Kasuma
Faculty of Dentistry, Andalas University, Indonesia

Indonesia is one of the countries that frequently suffers from mother nature disaster. Therefore, forensic odontology is consider as an essential entity in the forensic identification process. Palatal rugae is a very individualised characteristic anatomy in human and in certain population it may be descended through matrilineal lineage. Therefore, palatal rugae pattern has the potential to be used in identifying ethnic, one's race and lineage. Minangese as an ethnic group has unique kinship which is matrilineal-based. The aim of this study is to identify the pattern of Minangese palatal rugae based of shape and length of palatal rugae. This is a descriptive study. Total of 300 of Minangese people were recruited. Palatal rugae of each subject is marked using 2B pencil on the jaw mould accordingly. The method of identification of rugae pattern was adapted from Thomas and Kotze (1983) and Kapali et al (1997) which include the shape and length of rugae. The results showed that pattern of Minangese palatal rugae based of shape is wavy shape and pattern of Minangese palatal rugae based of length is primary rugae.

Keywords: Forensic odontology, Palatal rugae pattern, Rugoscopy, Minangese

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Neuroscience

Neuroprotective Effect of Chrysoeriol against MPP+-Induced Apoptotic Cell Death in SH-SY5Y Cells

Tanapol Limbunnrueng, Patoomratana Tuchinda, Sukumal Chongthammakun
Department of Anatomy and Structural Biology, Faculty of Science, Mahidol University, Bangkok, Thailand

Neuronal degeneration caused by mitochondrial apoptotic pathways implicates in many neurodegenerative diseases including Parkinson's disease (PD). Chrysoeriol, a flavonoid compound found in tropical plants, exhibits a variety of pharmaceutical activities including antioxidant and antiinflammatory properties. The protective effect of chrysoeriol extracted from Phyllanthus niruri in cellular models of PD has not been investigated. In the current study, we examined the protective effects along with the underlying mechanisms of chrysoeriol in an experimental PD model in vitro, in which SH-SY5Y cells were injured by 1-methyl-4-phenylpyridinium (MPP+). Our study showed that MPP+-induced cell death in SH-SY5Y cells was significantly reduced by chrysoeriol pretreatment in a dose-dependent manner, indicating the potent neuroprotective effects of chrysoeriol. The expression of pro-apoptotic Bax protein and anti-apoptotic Bcl-2 protein was examined by Western blotting of the protein levels and real-time quantitative PCR (RT-qPCR) measurement of mRNA levels. On the molecular level, we found that pretreatment with chrysoeriol significantly decreased the ratio of Bax to Bcl-2 at both the mRNA and protein levels. The results suggested that chrysoeriol exhibited significant neuroprotective effect against experimental PD models via regulation the balance of pro- and anti-apoptotic genes. The present study supports the notion that chrysoeriol may be a promising neuroprotective molecule for prevention of neuronal death in brain caused by neurodegenerative disorders such as PD.

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Cancer Biology

Stable knockdown of Y-box binding protein-1 (YB-1) inhibits breast cancer progression *in vitro*

Jia Pei Lim [1], Sukanya Shyama Sundar [1], Jayantha Gunaratne [1,2], Boon Huat Bay [1]
[1] Department of Anatomy, Yong Loo Lin School of Medicine, National University of Singapore, Singapore
[2] Quantitative Proteomics Group, Institute of Molecular and Cell Biology,
Agency for Science, Technology and Research, Singapore

Y-box binding protein-1(YB-1), an evolutionary conserved DNA or RNA binding protein, is known to be involved in a plethora of fundamental cellular processes. YB-1 has been reported to mediate chemoresistance and serve as an independent prognostic marker in breast cancer. Despite knowledge of the involvement of YB-1 in breast cancer progression, the exact mechanism by which YB-1 mediates metastasis is poorly understood. Hence, short hairpin RNA (shRNA)-mediated knockdown of YB-1 was carried out in the highly metastatic MDA-MB-231 breast cancer cell line, followed by functional analyses. Three stable YB-1 knockdown colonies (sq2(2E), sq4(4A) and sq4(5A)) were selected and silencing efficiencies were evaluated both at the mRNA and protein levels. The knockdown efficiency of *YBX1* gene were found to be approximately 37.8%, 65.9% and 66.8% in the selected colonies sq2(2E), sq4(4A) and sq4(5A) when compared to scrambled control cells. At the protein level, the YB-1 was decreased by approximately 33.5%, 63.5% and 68.4%. Using 3-(4,5-dimethylthiazol-2-yl)-5-(3-carboxymethoxyphenyl)-2-(4-sulfophenyl)-2H-tetrazolium (MTS) and alamarBlue assay, a reduction in cell proliferation was observed in the stable knockdown MDA-MB-231 cell colonies compared to scrambled control cells. Cell cycle analysis further revealed that stable knockdown of YB-1 in the MDA-MB-231 cells led to a G1 phase cell cycle arrest. Moreover, stable knockdown of YB-1 in MDA-MB-231 cells decreased both cell migration and invasion. The findings suggest that YB-1 could modulate breast cancer progression and serve as a potential biomarker for breast cancer metastasis.

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Cancer Biology

Peroxiredoxin 3 (PRDX3) promotes breast cancer progression through the modulation of cytoskeletal dynamics

Pei-Jou Chua[1], Heike Wollmann[2], Jing-Xian Zhang[2], Jayantha Gunaratne[1,2], Boon-Huat Bay[1]
[1]Department of Anatomy, Yong Loo Lin School of Medicine, National University of Singapore, Singapore
[2]Institute of Molecular & Cell Biology, Agency for Science, Technology & Research, Singapore
Email address: pei_jou_chua@nuhs.edu.sg; wheike@imcb.a-star.edu.sg; jxzhang@imcb.a-star.edu.sg; jayanthag@imcb.a-star.edu.sg; boon_huat_bay@nuhs.edu.sg

Reactive oxygen species (ROS) has a Janus face- on the one hand, too much ROS can be lethal to cells but on the other hand, low levels of ROS act as signaling molecules. As a redox sensor that is essential in maintaining mitochondrial homeostasis, peroxiredoxin 3 (PRDX3) has been observed to be significantly up-regulated in several types of common malignancies, including breast cancer. Our previous published data has shown that knockdown of PRDX3 reduced cell proliferation and induced cell cycle arrest, suggesting that PRDX3 is involved in breast tumorigenesis. By generating stable knockdown and overexpression of PRDX3 in MDA-MB-231 breast cancer cell line, we demonstrate that downregulation of PRDX3 increased cell adhesion to collagen I and inhibited tumor cell migration and invasion, while overexpression increased cell motility. By using RNA sequencing, a total of 462 genes were differentially expressed upon PRDX3 knockdown. Functional categorization of differentially expressed genes (DEGs) using DAVID analysis showed that DEGs were highly enriched in immune response, cell proliferation and developmental process as well as cell adhesion biological processes. Immunofluorescence staining of vinculin and F-actin indicated that PRDX3 is involved in the dynamic changes of cellular actin cytoskeleton and focal adhesion. Our data suggests that PRDX3 plays an important role in promoting breast tumourigenesis and could be a potential therapeutic target for novel or synergistic treatment approaches in breast cancer.

Deciphering the role of GRAM domain-containing protein 1B (GRAMD1B) in the JAK/STAT signaling cascade

Khanna P[1], Chua PJ[1], Thike AA[2], Tan PH[2], Bay BH[1], Baeg GH[1]
[1]Department of Anatomy, Yong Loo Lin School of Medicine, National University of Singapore, Singapore
[2]Department of Pathology, Singapore General Hospital, Singapore

The JAK/STAT signaling cascade is a key signal transduction pathway involved in the regulation and maintenace of a wide range of biological processes, such as cellular proliferation and immune responses. Consistently, the dysregulation of this pathway is associated with several human diseases, including immune disorders and cancers. However, the exact molecular mechanism of how JAK/STAT signaling integrates and transduces numerous extracellular cues to trigger specific genetic programs is not clear. One of the keys to filling this gap is by means of identifying and characterizing novel molecules involved in JAK/STAT-mediated biological processes. A genome-wide RNA interference (RNAi) screen was carried out to identify novel components of the JAK/STAT pathway in the fruit fly *Drosophila*. The RNAi-mediated knockdown of *Drosophila* GRAMD1B (dGRAMD1B) showed a significant increase in JAK/STAT reporter activity, suggesting that dGRAMD1B negatively regulates JAK/STAT signaling. Furthermore, our studies also showed that *dGRAMD1B* was transcriptionally up-regulated by JAK/STAT signaling, thereby generating a negative feedback loop. In mammalian cell culture system, pSTAT3 levels were greatly increased and showed accumulation in the nucleus on siRNA-mediated knockdown of hGRAMD1B, proposing disruption of pSTAT3 nuclear accumulation as a possible mechanism of negative regulation of JAK/STAT signaling by hGRAMD1B. In addition, our data also showed that JAK/STAT signaling induced by IL-6 treatment increases *hGRAMD1B* transcription, while AG490-mediated inhibition of JAK/STAT signaling decreased *hGRAMD1B* transcription; thereby supporting the existence of the negative feedback loop. More importantly, we also found that dGRAMD1B plays a vital role in JAK/STAT-mediated tumorigenesis *in vivo* (*Drosophila*). To further check for clinical significance of hGRAMD1B in gastric adenocarcinoma where dysregulated JAK/STAT signaling has been implicated, we carried out immunohistochemical staining of 138 malignant gastric tumor tissues and matched adjacent normal tissue. Our studies revealed a loss of hGRAMD1B expression in gastric tumor samples ($p<0.0001$), suggesting a tumor suppressor role of GRAMD1B in gastric tumorigenesis. All these fundamental new knowledge obtained about JAK/STAT-GRAMD1B regulation of cellular processes is expected to advance not only the general field of cell biology, but also provide an essential foundation for the identification of new therapeutic target(s).

Keywords: GRAMD1B, JAK/STAT signaling, pSTAT3, gastric cancer

Poster Abstract
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Aging

Effect of Cissus quadrangularis Linn extract on the development of osteopenia induced by ovariectomy in rats

Bhagath Kumar Potu
Department of Anatomy, College of Medicine & Medical Sciences, Arabian Gulf University, Bahrain
Email: potubk@agu.edu.bh

The aim of our study was to see the efficacy of petroleum ether extract of Cissus quadrangularis (CQ) on development of osteopenia in ovariectomy induced Wistar rats.The female Wistar rats were ovariectomized or Sham operated. The rats were anesthetized with pentobarbital sodium (40 mg/ kg b.w, i.p.), the ovaries were removed bilaterally. Sham-operation was performed in the same manner but only exposing the ovaries (sham operated (SHAM) group). A day later, the ovariectomized rats were randomly divided into four groups of eight animals each. The groups are 1. Sham operated (SHAM), 2. Ovariectomized (OVX), 3. Ovariectomized and treated with 25 mg/kg b.w of raloxifene (OVX+RAL), 4. Ovariectomized and treated with 500 mg/kg b.w of petroleum ether extract of CQ (OVX+CQ). The treatment continued for 30 days. At the end of the treatment, rats in all groups were sacrificed by cervical dislocation. Before sacrifice, blood was collected for the estimation of serum ALP, TRAP, Calcium and hydroxyproline; whereas the left femur was used for histomorphometrical analysis. The findings assessed on the basis of animal weight, morphology of femur, histomorphometry and biochemical analysis. As compared to SHAM group, OVX group animals showed a significant rise in serum ALP, TRAP and hydroxyproline levels at the end of 1 month following ovariectomy while no significant change was seen in the serum calcium levels. ALP and TRAP levels of OVX + RAL and OVX + CQ groups showed a further increase following administration of raloxifene and Cissus quadrangularis. The serum hydroxyproline content was found to be increased in the OVX + CQ compared to SHAM group. CQ significantly increased the thickness of both cortical ($p < 0.001$) and trabecular bone ($p < 0.001$). This action of CQ is comparable to action of Raloxifene.) These data suggest a strong anti-osteoporotic activity of CQ. The results confirm, at least in part, for the use of Cissus quadrangularis in folk medicine to treat osteoporosis.

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Neuroscience

Changes of morphological features of the extraforaminal ligaments of the spinal nerve roots in cervico-thoracic transitional zone

Pawaree Nonthasaen[1], Hisayo Nasu[1], Eiichiro Kagawa[1,2], Akimoto Nimura[3], Keiichi Akita[1]
[1] Department of Clinical Anatomy, Tokyo Medical and Dental University, Tokyo, 1138519, Japan
[2] JA Kyosai Research Institute
[3] Department of Functional Joint Anatomy

The Extraforaminal ligaments (EFLs) in the cervical and thoracic levels have been reported (Kraan et al., 2011; Shi et al., 2015). According to these reports, EFLs in the transitional zone might be quite different between the cervical and thoracic levels. In this study, we focused to examine EFLs in the transitional zone between both levels. Fourteen vertebral columns from the Japanese cadavers were carefully examined the EFLs from C4 to T4 levels. After dissection of EFLs, the anterior tubercles of cervical vertebrae and the proximal parts of the ribs were removed to investigate the relationships between EFLS and nerve roots. In addition, histological examinations were applied. EFLs at cervical levels consisted of the radiating ligaments which directly attached from uncovertebral joint and transverse process to spinal nerve roots, and the transforaminal ligaments which connected between two anterior tubercles of the cervical vertebrae. At the thoracic levels, EFLs consisted of the superior and inferior parts of costotransverse ligaments which connected between the rib and the transverse process, running ventral to the nerve and dorsal to it respectively. Interestingly, we could observe the EFLs characteristics of both cervical and thoracic levels around the T1 level. Interestingly, in C7-T1 levels, it was observed that nerve fibers connected between nerve roots passing through the foramen transversarium and the foramen costotransversarium. In the transitional zone between the cervical and thoracic vertebral levels, patterns of EFLS composed of the characters of both levels. In addition, connecting nerve fibers were observed only in this zone.

Induction of mouse induced pluripotent stem cells into neural crest cells *in vitro*

Y.Z. Niu, B. Feng, H. Zhao and W.Y. Chan
School of Biomedical Sciences, Faculty of Medicine, The Chinese University of Hong Kong, Shatin, Hong Kong SAR, P.R. China

Neural crest cells (NCCs) are multipotent embryonic cells which form a wide variety of derivatives during embryonic development. Abnormal migration or differentiation of NCCs would lead to many congenital birth defects such as Hirschsprung's disease, Axenfeld-Rieger syndrome and Waardenburg syndrome. Although extensive studies of NCCs have been performed over decades, expansion and propagation of NCCs from their primary culture remain a challenge. Recently, with the four Yamanaka transcription factors, we were able to generate mouse induced pluripotent stem cells (iPSCs) from embryonic fibroblasts. As iPSCs possess an almost unlimited ability to self-renew, we may establish a continuous source of NCCs if we can induce iPSCs to form NCCs *in vitro*. In the present study, we sought to develop an efficient protocol to specifically induce differentiation of mouse iPSCs into NCCs *in vitro*.

The protocol consisted of a three-step induction programme. After the reprogramming of mouse embryonic fibroblasts into iPSCs by transfection with the four Yamanaka factors, mouse iPSCs were firstly induced in a medium containing B27, N2 and FGF4 to form clusters of cells known as crestospheres. An addition of BMP4 to the medium enhanced the gene expression level of NCC markers Sox10, Ap-2α, Slug and Snail, and also induced more than 50% of the iPSCs to become p75 immunoreactive 6 days after induction. The next step involved emigration of NCCs from the crestospheres on a laminin-coated culture surface in the NCC induction medium (NCIM) containing FGF2 and EGF. Expression of NCC markers Sox10, Ap-2α, Slug, Snail, id2, id3 and nestin were upregulated when cells emigrated from crestospheres. The last step was expansion and maintenance of NCCs in an undifferentiated stage in the NCCs maintenance medium containing GDNF. Real-time PCR and immunostaining results showed that 50 ng/ml GDNF in the medium could keep NCCs undifferentiated with the continuous expression of NCC markers Sox10, Ap-2α, nestin and FoxD3. Induced NCCs were able to differentiate into Schwann cells, neurons, smooth muscle cells, chondrocytes and osteocytes *in vitro* under different differentiation conditions. When transplanted to E13.5 hindgut explants *ex vivo*, induced NCCs gave rise to TuJ1 immunoreactive neurons 2 days after transplantation. Hence, our results indicated that mouse iPSCs can be induced to form NCCs *in vitro* without any gene modifications, and the induced NCCs so derived would be useful in the NCC research and cell-based transplantation studies.

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The Role of Transforming Growth Factor-β Signalling in Persistent EBV Infection of Nasopharyngeal Epithelial Cells

V Sharmila[1], SW Tsao[2], CM Tsang[2], C. Dawson[3], IC Paterson[1], LF Yap[1]
[1] Department of Oral Biology and Craniofacial Sciences, Faculty of Dentistry, University of Malaya, Kuala Lumpur, Malaysia
[2] Department of Anatomy, Li Ka Shing Faculty of Medicine, University of Hong Kong, Hong Kong
[3] School of Cancer Sciences, University of Birmingham, UK

Undifferentiated nasopharyngeal carcinoma (NPC) is consistently associated with Epstein-Barr virus (EBV) infection. However, the molecular events that regulate the establishment of stable EBV infection in nasopharyngeal epithelial (NPE) cells remain largely undefined. Transforming growth factor-β (TGF-β) signalling regulates a variety of cellular processes and functions as a tumour suppressor in the early stage of epithelial carcinogenesis by inhibiting cell proliferation and promoting differentiation. We have preliminary evidence to show that undifferentiated epithelial cells which have lost their growth inhibitory response to TGF-β are more amenable to stable EBV infection compared to cells with an intact TGF-β response. Therefore, to test this hypothesis, we used two different approaches to disrupt TGF-β signalling in the premalignant NPE cell lines, NP361hTert and NP550hTert; (1) treatment of cells with a TGF-β receptor type I (TGF-βRI) kinase inhibitor (SB431542) and (2) overexpression of a kinase-deficient truncated form of the TGF-β receptor type II (dnTGF-βRII). Functional disruption of the TGF-β signalling pathway was confirmed in SB431542-treated and dnTGF-βRII-expressing cells by the demonstration of a reduced growth inhibitory response to TGF-β1, inhibition of ligand-induced phosphorylation of SMAD2 and loss of SMAD2/3-dependent signalling upon TGF-β1 stimulation. These cells were then infected with a GFP-tagged recombinant EBV (Akata strain) and the percentage of GFP-positive cells was determined by FACS. Compared to the controls, treatment with SB431542 and forced expression of dnTGF-βRII resulted in a longer persistence of EBV infection in both cell lines. Further, higher copy numbers of the EBV episomes were demonstrated in *NP550hTert cells treated with* SB431542, compared to the vehicle controls. Of note, serum-induced differentiation was suppressed in the SB431542-treated and dnTGF-βRII-expressing NP550hTert cells. To determine whether disruption of TGF-β signalling would protect cells from EBV-induced senescence, the activity of the senescence-associated β galactosidase (SA-β-Gal) was determined following EBV infection. The results showed that the SB431542-treated and dnTGF-βRII-expressing cells had lower percentages of senescent cells compared to the controls. Similarly, the expression of p16 (a marker of senescence) and p21 (a marker for growth arrest) in the controls was stronger than those in the dnTGF-βRII-expressing cells and SB431542-treated cells. Taken together, our data indicate that *defects in the TGF-β signalling pathway support and prolong EBV persistence in nasopharyngeal epithelial cells, possibly by suppressing cellular differentiation and/or EBV-induced senescence.*

Poster Abstract
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Neuroscience

Anti-inflammatory effects of Sea Cucumber Extracts (Holothuria scabra) on Cognitive Impairment of Vascular Dementia Mice

Farhana Padungraksart, Thanaporn Rungruang, Narawut Pakaprot, Thongchai Taechowisan, Prasert Sobhon, Supin Chompoopong
Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand

Sea Cucumber Extracts (Holothuria scabra extracts or HsE) is a valuable source of several kinds of substances that can serve as natural health products, contain saponins or triterpene glycosides. Pharmacological studies indicate anti-inflammatory and anticancer properties of the sea cucumber saponins. It may enhance learning and memory and promote brain functions. To determine the therapeutic potential of HsE as an alternative treatment for vascular dementia in mice, the chronic cerebral hypoperfusion was induced by carotid artery occlusion. In forty ICR mice, after the occlusion, HsE was injected intraperitoneally for ten days. The Morris Water Maze (MWM) was then performed after ten days of HsE treatment. At the end of experiment, all mice were sacrificed, and the brains were removed for histopathological determination of neuronal cell death and ELISA of TNF-α. The results showed that cerebral hypoperfusion increased the pyramidal cell death in the hippocampal CA1 area, impaired the spatial learning and memory and increase in TNF-α level. HsE improved memory retention in MWM, The CO+HsE group swam up to the platform with escape latency time (15.91±2.46 sec, $p<0.05$) compared with injury group (18.17±0.49 sec, $p<0.05$). Therefore, HsE could attenuate the memory deficits, the pyramidal cell death in hippocampal CA1 area related to decrease in TNF-α level in serum and brain. Role of HsE to decrease neuronal cell death may be described by the anti-inflammatory effect of HsE. Therefore, HsE improved the cognitive impairment, suggesting the therapeutic effects of HsE against the cerebral hypoperfusion.

Keywords: TNF-α, vascular dementia, Cognitive impairment, Holothuria scabra extracts.

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Cancer Biology

Elucidating the role of DLC1 in Metastatic Melanoma

Chris Xintao Yang, May Pui Lai Cheung, Michael Rui Liang, Martin Cheung
School of Biomedical Sciences, Li Ka Shing Faculty of Medicine, The University of Hong Kong

Deleted in liver cancer 1 (DLC1) is a Rho GTPase-activating protein (RhoGAP) that is generally recognized as a tumor suppressor. Whether it functions, as a tumor suppressor in melanoma is largely unknown. In this project, we revealed that cytoplasmic DLC1 was detected in human metastatic melanoma cells characterized by dermal invasion. More importantly, we found SOX10, member of SOX family transcription factor, which is crucial for melanoma growth and progression, was initially localized in the nuclei of benign melanocytic nevus and became co-localized with DLC1 in the cytoplasm of metastatic melanoma, suggesting a correlation of their expression and possible functional interaction with melanoma progression. In addition, there are five DLC1 isoforms corresponding to different transcript variants due to alternative splicing. qPCR and Western blot analysis demonstrated that transcript variant 2 was predominantly expressed in a panel of melanoma cell lines compared to other variants, indicating that DLC1 isoform 2 could play a role in the development of melanoma. To investigate this issue further, we have established clones stably expressing shRNA targeting isoform 2 in melanoma cell lines and found DLC1-silencing resulted in increased the level of active RhoA-GTP and promoting the formation of stress fibers. More significantly, in vitro functional assays revealed dramatic promotion of proliferation, migration and invasion in DLC1-knockdown cell lines. In vivo assessment of the effects of DLC1-knockdown on tumor formation and metastasis using nude mice model is currently ongoing. By far, our pilot findings suggest a unique paradigm for DLC1 in metastatic melanoma.

Enlarged Perivascular Spaces Related to AQP4 and GFAP expression of Mice Cerebral Malaria

Lanaprai Kwathai, Thanaporn Rungruang, Panapat Uawithya, Supin Chompoopong
Science and Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand

Cerebral malaria (CM) is a major cause of mortality and morbidity in severe Plasmodium falciparum. Post mortem analyses of brain of CM patients show adherence of parasitized red blood cells and inflammatory cells to the microvascularature of the brain. Loosening of tight junctions and degrading of the basal lamina located between the astrocytic endfeet and endothelia cause the breakdown of the BBB leading to brain edema after the upregulation of proinflammatory cytokines. The aquaporin-4 (AQP4) related to brain edema, therefore, this study was aimed to evaluate the perivascular space related to the expression of AQP4 and GFAP at astrocyte end feet in CM mice model, Mice were inoculated intraperitoneally with Plasmodium yoelii lethal (PyXL). Then observed sign of CM and recorded % of parasitaemia daily. Immunoperoxidase staining for AQP-4 and GFAP in brain were performed in 3 μm thickness of coronal sections. The results showed that infected CM mice without any treatments died within 8 days after inoculation and they showed clinical signs of neurological involvement such as convulsion and ataxia before death. Moreover, vessel congestion and enlargement of perivascular space, the potential sensor of systemic inflammation were also found in CM mice. The expression of AQP4 at astrocyte foot process was higher in CM group than non-infected group. Average number of reactive astrocyte of hippocampus CA1 areas in CM group (41.33 cells/145 μm2) was shown two times more expression than sham group (16.33 cells/145 μm2) and in ventricular areas (82 and 39.67 cells/145 μm2 in CM and sham groups, respectively). In conclusion, the present morphological study may support the prominent role of AQP4 related to astrocyte activation and enlargement of the perivascular spaces which is a consequence of increased vascular permeability. Therefore, AQP4 might be a target for therapeutic intervention to prevent CM.

Keywords: Cerebral malaria, AQP4, Brain edema, Astrocyte, Blood-brain barrier.

A Study on the Medical Student Learning Experience of Surface Anatomy and its Clinical Correlation through Standardized Patient

Satish R.L, Department of Anatomy, Yong Loo Lin School of Medicine, National University of Singapore, Singapore-117594
Aishwarya K, School of Medicine, University College of Cork, Cork, Ireland

Background: Surface anatomy is a branch of gross anatomy that examines shapes and markings on the surface of the body as they relate to underlying deeper structures in the human body. Understanding surface anatomy is one of the preclinical cornerstones of medical education. Surface anatomy is essential in enabling medical students locate and identify anatomical structure in studying gross anatomy. Furthermore, a clear understanding of surface anatomy helps medical students diagnose medical conditions with abnormal clinical signs (E.g. tracheal deviation, abnormal heart sounds, irregular pulses, organomegaly, caput medusa etc.,) and in treating patients (E.g. inserting a chest tube or drain). There is a general feeling that medical students have inadequate knowledge of clinically relevant anatomy. Standardized Patients (SP) provide medical students with a more authentic environment to learn; allowing for experiential learning which enable medical students to better understand and retain anatomical knowledge as well as emulating real time patients that they will encounter which emphasis the underlying clinical relevance of anatomy in the future medical practice. A clear understanding of anatomy is pivotal. We are studying the effect of SPs in medical students' experience of better understanding anatomy and appreciating the clinical relevance of anatomical structures of the cardiovascular, respiratory and gastrointestinal (mainly abdominal region) systems. Using SPs has greater impact on cognitive, affective, and psychomotor learning in medical students. Material & Methods: Survey results from students who are in their preclinical years (1st year of medical school) undergoing human anatomy education will be collected. 22 first year medical students participated in the ongoing study involving this teaching method. The students will attend 2-hour anatomy tutorials followed by 30-minutes hands-on sessions using SPs over a total of 7 sessions. After this session student will be administered with survey questionnaires. Anatomy knowledge prior and post introduction of SPs will evaluated for improvements made in clinical examination skills. Medical Students rely mainly on 4 techniques during their 30- minute hands on session: 1) Visual inspection : Medical students will directly observe the structure and markings of surface features (E.g. Observing the jugular venous pressure (JVP) along the border of the sternocleidomastoid muscle) 2) Palpation: Medical students will feel with a firm pressure or perceive by their sense of touch to precisely locate and identify anatomical features under the skin (E.g. Locating the sternal angle and palpating second rib to further locate the apex beat). 3) Percussion: Medical students will firmly tap specific body sites to detect resonating vibrations (E.g. Percussing the abdominal region for splenic and liver dullness) 4) Auscultation: Medical students will listen to sounds emitted from organs (E.g. Listening to normal heart sounds and recognizing murmurs if any) Results: Students attending tutorial session in its enhanced form of teaching through SPs will be compared based on their performance. The survey results will give us an insight as to how engaging, clinically relevant and effective the sessions with the SPs will be for medical student in understanding Anatomy. The survey questionnaire will also focus on the difficulty students face in deciphering the Normal heart, Lung & Bowel sounds and most clinically challenging signs (E.g. JVP) with the presence or absence of SPs. We will study such impacts through our survey questionnaire. We anticipate that traditional anatomy tutorials combined with SPs teaching session will be useful to preclinical first year medical students in learning clinically relevant anatomy.

Impact of amyloid beta precursor protein gene *APP* overexpression on the cell growth and migration/invasion of Down syndrome trophoblast

Oscar GW Wong, Claire LY Cheung, KK Chan, Philip Ip, Annie NY Cheung
Department of Pathology, Faculty of Medicine, The University of Hong Kong, Hong Kong

Introduction: Down syndrome (DS) is the most common congenital abnormality in humans. Trisomy 21 is the major cause accounting for about 95% of DS. Placentas of DS exhibit a number of development al defects. The molecular basis underlying the dysregulation of trophoblast differentiation and hCG signa lling in DS placentas is largely unknown. Overexpression of dosage sensitive genes present on human c hromosome 21 as the results of the extra copy may contribute to the abnormal development of DS pla centas. We have previously demonstrated that the gene *APP* coding for Amyloid beta precursor protein is significantly overexpressed in the trophoblasts of DS placentas.

Objectives: To investigate the functional impact of *APP* overexpression on various trophoblast functions.

Methodology: Inducible APP expression systems were established in the model trophoblast cell lines HT R-8/SVneo and BeWo. Induced expression of APP was verified by western blot. Cell growth was monit ored by MTT assay. Cell migration and invasion were evaluated by transwell migration/invasion assay. The impact of APP expression on trophoblast differentiation was examined by syncytialization marker α/ β-hCG and syncytin expression as well as confocal imaging of syncytia formation upon induction by fo rskolin.

Results & Outcome: APP induction in HTR-8/SVneo dose-dependently decelerated cell growth and conc omitantly decreased β-hCG secretion into the culture medium. Moreover, HTR-8/SVneo expressing APP migrated and invaded slower than the control uninduced cells. Forskolin treatment induced α/β-hCG and syncytin expression in BeWo but these markers was inhibited by APP expression. E-cadherin immunoflu orescence staining showed that there was a decrease in the number of syncytia formed in forskolin trea ted BeWo expressing APP. Taken together, APP overexpression observed in DS placentas produced nega tive impact on trophoblast functions and may contribute to the abnormal phenotypes of DS placentas.

Establishment and characterization of immortalized nasopharyngeal epithelial cells with stable infection of Epstein-Barr virus

Lin Jia, Chi Man Tsang, Yim Ling Yip, Wendy Law, Jun Zhang, Wen Deng, George SW Tsao
School of Biomedical Sciences, Faculty of Medicine, The University of Hong Kong

Nasopharyngeal carcinoma (NPC), with a remarkably distinctive ethnic and geographic distribution, has the highest incidence among Southern Chinese. Unlike other types of head and neck cancers, a unique feature of NPC is its close association with Epstein-Barr virus (EBV). EBV infection represents an early event during the pathogenesis of NPC. Latent EBV infection is found in every cancer cell of virtually all cases of undifferentiated NPC in endemic regions. Genetic alterations in the premalignant epithelium may support the EBV latency. Although EBV-encoded proteins in its latent infection, including LMP1, LMP2A, etc., have been identified with crucial functions to initiate or promote oncogenesis, to date the exact role of EBV infection in the malignant transformation of nasopharyngeal epithelial (NPE) cells remains unclear. We have previously generated a panel of immortalized NPE cells by ectopic overexpression of hTert (catalytic human telomerase). However, reliable in vitro models of stable EBV infection are also necessary for further elucidating the regulatory network supporting the persistent latency of EBV in NPE cells, and also the potential roles of EBV in the pathogenesis of NPC. In the present study, we established NP361hTert-EBV cells with stable EBV infection via co-culture with Akata B cells harboring EBV, followed by the effective enrichment of EBV-infected cells, indicated by positive GFP, by FACS sorting. The loss of EBV episome in NPE cells was fast initially upon infection, as well as the first and second enrichment of EBV-positive cells. The relatively stable maintenance of EBV could be achieved after enrichment for three times. Results of western blotting analysis showed that the protein expression level of cyclin D1 was increased in NP361hTert-EBV cells, as compared to the non-infected NP361hTert cells. Meanwhile, we also observed increased expression of phosphorylated protein kinase Akt (Ser473) in NP361hTert-EBV cells, suggesting the Akt signaling was activated, and such activation might contribute to the persistence of EBV in NP361hTert cells. This stable EBV-infected NPE cells will serve as a powerful in vitro model for further study in elucidating the functional role of EBV in the carcinogenesis of NPC.

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Poster Abstract
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Neuroscience

Decrease in Interleukin 1 beta and White Matter against Vascular Dementia in Mice by Sea Cucumber Extracts

Thitipong Naowasiri, Thanaporn Rungruang, Narawut Pakaprot, Thongchai Taechowisan, Prasert Sobhon, Supin Chompoopong
Department of Anatomy, Faculty of Medicine Siriraj Hospital, Mahidol University, Thailand

Vascular dementia (VD) is the second most prevalent type of dementia with involved in white mater (WM) damage and related to memory deficits. Sea Cucumber Extracts (Holothuria Scabra or HsE), one of marine source, used as supplementary food and transitional medicine in Asia. HsE composed of nutritional benefits, triterpenoid saponins which may act as a natural antioxidant and help in damage pathway. To determine the effects of HsE on VD using voice-cued fear (VCF) conditioning test, non-vision dominant. The chronic cerebral hypoperfusion was induced by carotid artery occlusion in forty ICR mice. After the occlusion, HsE was injected intraperitoneally for ten days, and VCF conditioning test was then performed after HsE treatment. Proinflammatory cytokines, both serum and brain interleukin-1 beta (IL-1β) level were decreased in CO-HsE group. HsE also improved WM damage with myelin index from 40.13±7.99 to 45.13±6.11 and memory retention with increased freezing behavior from 20.99 ± 2.34 % to 46.2 ± 3.55%, at $p<0.001$. The conditioned fear is often measured with freezing, a period of watchful immobility. Mice learned to stop moving as fear response, when the learning and memory process was interrupted with damaged white matter such as corpus callosum, the freezing behavior was decreased. In conclusion, the anti-inflammatory effect of HsE was shown by decreased IL-1 β. HsE also attenuated the memory deficits, more freezing in VCF, and improved WM damage, it may be suggested for using as drug in the alternative medicine.

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Neuroscience

Reactive Astrocytes Changed after Combined Chondroitinase ABC and 17beta-Estradiol treatments in Mild Cervical Spinal Cord Contusion

Bamrungsuk K[1], Chompoopong S[2*]
[1]PhD student, Department of Physiology, [2]Department of Anatomy, Faculty of Medicine Siriraj hospital, Mahidol University, Bangkok, 10700, Thailand.
**Corresponding author: E-mail: supin.cho@mahidol.ac.th,*

A mild contusion of the spinal cord may cause the loss of only some function below the site of the injury which can be evaluated the recovery function. The injury also results in more condroitin sulfate proteoglycan (CSPG) expression from accumulated reactive astrocytes at the lesion site which can cause glia scarring and block the axonal regeneration. Therefore, the successful restoration of sensory and motor functions will probably require a combination of approaches to address different aspects of the problem. To determine the effects of combined chondroitinase ABC (ChABC) and 17beta-estradiol (E2) on reactive astrocyte accumulation, the mild C5 hemicontusion injury (SCIM) was performed in rats. For SCIM-Ch-E group, 0.1 U of ChABC was applied immediately, day 3, day 7, and day 11 after injury. In addition, 300 µg/kg of E2 was intraperitonelly injected at 30 min after injury, daily for two weeks and then weekly until sacrificed. The GFAP immunoperoxidase and C-4-S immunofluorescence stained section were evaluated the number of astrocytes and expression of degraded CSPG, respectively by using computer image analysis. The combined ChABC and E2 treatment could significantly decrease the number of astrocytes (SCIM, 12.35±0.51, SCIM-Ch-E, 7.30±0.27) and showed more degraded CSPG activity (SCIM, 3.90±0.67, SCIM-Ch-E, 20.30±3.75) after injury. The GFAP staining showed hypertrophic astrocytes with extended processes aggregated at lesion in SCIM. The survival neurons in SCIM-Ch-E were surrounded by activated astrocytes with short processes. In conclusion, E2 treatment may reduce proinflammatory cytokines and decreased astrocytes aggregation. ChABC treatment could abolish the binding to CSPG receptor results in glial scar degradation which provides physical and biochemical characteristics of architecture in glia scar tissue promoting for neuronal growth.

Keywords: Chondroitinase ABC, Estrogen, Glial scar, Spinal cord injury

Investigating the impacts of Parkinson's Disease Dementia (PDD) by using an animal model in PD

Cindy Chi-Ching Pang [1,2], Wendy Noble[2], Raymond Chuen-Chung Chang[1,3,4]
[1]Laboratory of Neurodegenerative Diseases, School of Biomedical Sciences, LKS Faculty of Medicine, Pokfulam, The University of Hong Kong
[2]Institute of Psychiatry, Psychology and Neuroscience, Department of Basic and Clinical Neuroscience, King's College London, London, UK
[3]State Key Laboratory of Brain and Cognitive Sciences, The University of Hong Kong, Pokfulam, Hong Kong
[4]Research centre of Heart, Brain, Hormone, and Healthy Aging, LKS Faculty of Medicine, The University of Hong Kong, Pokfulam, Hong Kong
E-mail Correspondence: rccchang@hku.hk

Parkinson's disease (PD) is the second most common age-related neurodegenerative disease. The symptoms in PD are not limited to motor impairments, with increasing focus on the cognitive aspects of the disease. Cognitive functions typically involve executive functions, working memory and visiospatial memory. There is increasing awareness that cognitive dysfunction in Alzheimer's disease is different to that in PD. With approximately 80% of PD patients developing Parkinson's Disease Dementia (PDD) in the course of disease progression, we aim to establish a PDD model. The establishment of such a model will allow further understanding of disease progression, the molecular basis and pathogenesis of disease in relation to cognitive impairment, and can also be used to test for potential therapeutics.

Male Sprague-Dawley rats were injected with the neurotoxin 6-hydroxydopamine (6OHDA) into the medial forebrain bundle (MFB). Controls were injected with saline. Motor deficits were assessed using cylinder, rotarod and corner tests. Visuospatial impairment, which is part of the executive domain, was assessed using the Morris watermaze test. An induced apomorphine rotation test was used to confirm depletion of dopamine in the nigrostriatal pathway.

The behavioral problems of motor and cognitive dysfunctions shown in our animal model match with the motor and cognitive symptoms in patients with PDD, in particular visiospatial deficits. 6OHDA injected animals all showed significant deficits of $p < 0.05$ when compared to controls on the cylinder, corner and induced apomorphine rotation test.

From the established PDD model using 6OHDA injection into the MFB, motor deficits and cognitive deficits were apparent. Rotarod test was less sensitive in showing motor deficits compared to the corner and cylinder tests. This PDD model is a possible model for screening potential therapeutics in the future.

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Poster Abstract
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Neuroscience

Tetratricopeptide repeat domain 9A modulates anxiety-like behavior in female mice

Shawn Zheng Kai Tan [1], Lee Wei Lim [1 #], Smeeta Shrestha [2], Yu Zuan Or [2], Hwa Hwa Chung [2], Yang Sun [2], Chew Leng Lim [2], Sharafuddin Khairuddin [1], Thomas Lufkin [3], Valerie CL Lin [2 #]
[1] School of Biomedical Sciences, Li Ka Shing Faculty of Medicine, The University of Hong Kong, Hong Kong, China
[2] School of Biological Sciences, Nanyang Technological University, Singapore
[3] Department of Biology, Clarkson University, Potsdam, New York, USA
[#] Authors for correspondence: Lee Wei Lim (limlw@hku.hk) and Valerie CL Lin (cllin@ntu.edu.sg).

Tetratricopeptide repeat domain 9A (TTC9A) expression is abundantly expressed in the brain. Previous studies in TTC9A knockout (TTC9A-/-) mice have indicated that TTC9A negatively regulates the action of estrogen. In this study we investigated the role of TTC9A on anxiety-like behavior through its functional interaction with estrogen using the TTC9A-/- mice model. A battery of tests on anxiety-related behaviors was conducted. Our results demonstrated that TTC9A-/- mice exhibited an increase in anxiety-like behaviors compared to the wild type TTC9A+/+ mice. This difference was abolished after ovariectomy, and administration of 17-β-estradiol benzoate (EB) restored this escalated anxiety-like behavior in TTC9A-/- mice. Since serotonin is well-known to be the key neuromodulator involved in anxiety behaviors, the mRNA levels of tryptophan hydroxylase (TPH) 1, TPH2 (both are involved in serotonin synthesis), and serotonin transporter (5-HTT) were measured in the ventromedial prefrontal cortex (vmPFC) and dorsal raphe nucleus (DRN). Interestingly, the heightened anxiety in TTC9A-/- mice under EB influence is consistent with a greater induction of TPH 2, and 5-HTT by EB in DRN that play key roles in emotion regulation. In conclusion, our data indicate that TTC9A modulates the anxiety-related behaviors through modulation of estrogen action on the serotonergic system in the DRN.

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Developmental Biology

Dlc1, a Rho GTPase-activating protein, is essential for cranial neural crest development

Yanxia Rao, Jessica Aijia Liu, May Pui Lai Cheung, Martin Cheung
School of Biomedical Sciences, Li Ka Shing Faculty of Medicine, The University of Hong Kong, Hong Kong, China

An important feature of neural crest is its ability to undergo epithelial to mesenchymal transition(EMT), After EMT, neural crest cells migrate extensively throughout the body to differentiate into a variety of cell types. Here, we identified that two different isoforms of *Dlc1*, a Rho-GTPase-activating protein (RhoGAP) that negatively regulates specific Rho family proteins (RhoA-C), were expressed at cranial neural crest cells(CNCCs). Isoform3(the short one) was predominately expressed at the onset neural crest cells and maintained its expression in migrating CNCCs, while the isoform1(the longest one) was only expressed at migrating CNCCs. At cranial level, loss of isoform 3 function in early stage prevented neural crest normal delamination, the neural crest specifier gene *Snail2 and Sox9* was abnormally retained in the dorsal neuroepithelium, indicating Dlc1 isoform3 plays a critical role in promoting the neural crest cell state transition. In addition, knockdown of isoform1 caused a reduction of Sox10, which is normally required for emigration, as well as migrating neural crest marker HNK1. Moreover, isoform1 perturbation in neural crest cells impacted trigeminal ganglia assembly. Together, these findings demonstrate a novel function of two different isoforms of Dlc1 in cranial neural crest development. To further understand the mechanism underlying Dlc1controling cranial neural crest development. We will adopt series experiments to solve the issues as follow:1) As a well-studied RhoGAP protein, Dlc1 negatively regulates Rho activity. Previous studies demonstrate Rho ability to influence cell polarity and motility. Thus it is important to understand how Dlc1 controls Rho activity during cranial neural crest delamination and migration. 2) A complete EMT process of cranial neural crest cells is controlled by a complex net-work. thus it necessary to find the protein co-factors associated with Dlc1involing in EMT.

Poster Abstract
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Innovations in Teaching Gross Anatomy

Innovations in medical anatomy education with massive open online course technology

Pei Wang and Chia-Chuan Wang
School of Medicine, Fu Jen Catholic University, New Taipei City, Taiwan, ROC

The educational technology of massive open online courses (MOOCs) has been successfully applied in learner around the globe and medical teachers are now looking to MOOC technology as a means to improve professional medical education. We first designed an anatomy MOOC and launched on the Taiwan MOOCs platforms in 2015-2016. Major topics of the course focus on the skeletal and muscular systems, and the contents include not only basic structural and functional knowledge but also the performance of body fitness. The anatomy MOOC has attracted over 1,600 learners and campus-based undergraduate students through four rounds. Results of the questionnaire indicated that learners are favorable to the MOOC in effective teaching, proper learning objectives, interesting assignments, and high-quality videos. Furthermore, students in classroom teaching strongly agree that this MOOC help their consolidation and revision and can be a supplement to the existing traditional course. Additionally, the young employs, retiree and senior fair were also the primary users of our anatomy MOOC. Therefore, the course evaluations suggest the medical online course integrating with healthcare knowledge is an innovative and acceptable teaching model for multidisciplinary in the medical school and public environments.

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Cancer Biology

Functional roles of Phosphoglycerate dehydrogenase in Tumorigenesis

Muthukrishnan Chandrika, Chua Pei Jou, Bay Boon Huat
Department of Anatomy, Yong Loo Lin School of Medicine, National University of Singapore, Singapore

The enzyme Phosphoglycerate dehydrogenase acts as a pathbreaking oncogene in the pathogenesis of breast cancer. It supports cell growth through the serine production pathway and replenishes alpha ketoglutarate enhancing cancer cell proliferation. PHGDH poses as a biomarker as majority of Estrogen receptor negative breast tumours exhibited overexpression of this protein. The overall survival time and prognosis of certain breast cancers can be reckoned by assessing the enzyme. Studies show that Phosphoglycerate dehydrogenase knockdown by sh RNAs causes decline in serine levels leading to reduced cell proliferation. The identification of a Phosphoglycerate dehydrogenase inhibitor may be a harbinger to future anticancer therapy as it is so perspicuous that PHGDH suppression abridge the availability of glycine and glutamine for the cancer cells thereby causing tumour regression.

Keywords: Phosphoglycerate dehydrogenase, Serine, Breast Cancer

Poster Abstract
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Good practices in Teaching Gross Anatomy

Web - An easy accessible source for learning Anatomy

Muthukrishnan Chandrika, Krishnan Jayabharathi, Omid Iravani
Department of Anatomy, Yong Loo Lin School of Medicine, National University of Singapore, Singapore

Web has become the major source of information globally for the last decade. Students use the web to obtain their knowledge and work on research projects. Biomedical students widely use web as an accessible learning resource to study Anatomy.
Anatomy is the fundamental basis in medical education. Besides original textbooks, classes, and dissections, web has become a major source for medical students to obtain their knowledge. Web provides a variety of scientific information in form of e-books, lectures, animated videos, figures, courses etc.
During the academic Year 2015-16, over 97 medical, 47 dental and 20 pharmacy students from Yong Loo Lin School of Medicine, NUS were assessed to study their attitude towards usage and values of web as a scientific source for learning Anatomy. A LIKERT questionnaire was used for this assessment. Analysis of the data revealed that web is an easy accessible source of information as 70.1% of the cohort agreed it. Moreover 57.9% of them believe that web saved their time in learning anatomy. 53.3% of the students ascertained web as a reliable source of information. Approximately, 88.4% of the students mentioned that the web information augmented their knowledge in Anatomy.
In brief, the web, besides being accessible source to the students provides a valuable database to nurture their knowledge.

Keywords: Web; Anatomy; Medical Education

Poster Abstract
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Good practices in Teaching Gross Anatomy

Drawing Diagrams, A Method that Never Dies in Teaching Anatomy

Krishnan Jayabharathi, Muthukrishnan Chandrika, Omid Iravani
Department of Anatomy, Yong Loo Lin School of Medicine, National University of Singapore, Singapore

Anatomy is the cornerstone of medical education. A number of methods have been used for teaching Anatomy. Lectures, small group discussions, question-based learning (QBL), team-based learning (TBL), problem-based learning (PBL), dissections are all methodologies, which have been hired for teaching Anatomy. Drawing diagrams has been frequently used in majority of the biomedical disciplines to facilitate learning process. In fact, drawing diagrams uses to illustrate the structure and relations of the body structures. Since Anatomy is a visual science, simple drawings let students to touch, think and learn the structures deeply. A critical question is: How beneficial is the simple drawings? A research was carried out on first-year medical (n=82) and dental (n=42) students during academic year 2015-16 in Yong Loo Lin School of Medicine, National University of Singapore (NUS). A LIKERT questionnaire was designed to assess the student's attitude about the values and impact of simple drawings on their learning. In this study, majority of the students believe that they learnt Anatomy better when they were taught by drawings. More than 86% of the students agreed that simple drawings greatly helped them to realize normal Anatomical structures and complicated concepts. Additionally, 83.9% of the students mentioned that learning Anatomy by drawings greatly let them retain the information for a longer term. Majority of the medical students believed that simple drawings had a great impact on their examination performance (n=124, p=0.004). Together, besides several other innovative methodologies, it seems that simple drawings may hardly be substituted by other methodologies. Although students nowadays may seek for web-based materials for their own learning, many of them may still tend to learn Anatomy through drawing diagrams to consolidate their knowledge.

Keywords: Drawing Diagram, Anatomy, Teaching methodology

The assessment of expression of ectoderm, mesoderm and endoderm markers in embyoid body-like cell aggregates formed from Wharton's jelly mesenchymal stem cells using in regenerative medicine

Bahmanpour S, Rezaei F, Talaei-khozani T
Laboratory for Stem Cell Research, Department of Anatomical Sciences, School of Medicine, Shiraz University of Medical Sciences, Shiraz, Iran

Regenerative medicine is an area of medicine with the potential to heal damaged tissues and organs. Stem cells have the ability of differentiation into many different types of cells and are a key component of regenerative medicine, as a new clinical application. Mesenchymal stem cells (MSC) isolated from Human umbilical cord Wharton's jelly (HUCWJ) have been shown to be able to differentiate into various cell types. As they are readily available, do not raise any ethical issues and showed higher differentiation potential compared to adult stem cells. Therefore, HUCWJ is a potential source of material that can be used in regeneration medicine. The objective of this study was to find if these cells could form cell aggregates similar to that formed by ESCs (embroid body-like and form three germ layers.

The Umbilical Cords were cut into small pieces and the explants were cultured in the presence of α-MEM containing 10% fetal bovine serum (FBS), 1% L-glutamine, 100 g/mL penicillin/ streptomycin. At passage 3rd, 1000, 5000 and 10.000 cells/ 20μL were cultured in hanging drops for 3 days. Then, they were incubated for additional 3 days in non-adhesive dishes. The cell aggregates were fixed by 4% paraformaldehyde and were incubated with human three germ layer, 3 color antibodies and the flowcytometry was done.
The data showed that the embryoid-body-like aggregates had little expression for ectoderm and endoderm markers and much expression for mesoderm markers.

These aggregates stay at the mesenchymal cell mass manner and showed a poor differentiation potential toward the ectoderm and endoderm.

Poster Abstract
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Stem Cells Research

The effect of platelet rich plasma on the blood vessels volume of CCl4-induced hepatotoxic model

Zahra Vojdani, Afsaneh Mafi, Farzaneh Dehghani, Abbass Moghadam, Ali Noorafshan, Tahereh Talaei-Khozani
Anatomy Department, Shiraz Medical School, Shiraz University of Medical Sciences, Shiraz, Iran
vojdaniz@sums.ac.ir

Platelet rich plasma (PRP) is an autologous source of growth factors including those involved in angiogenesis. Activated PRP has been reported to increase the blood vessel and microvascular density. PRP also has healing impact on the fibrosis in cirrhosis model rats and has beneficial effects on regeneration of liver after hepatectomy. Morphological and structural deformity was reported in microvasculature of cirrhosis liver. The aim of this study was to investigate the effect of PRP administration on the volume of microvasculature and blood vessels of hepatotoxic-induced liver fibrosis.
Rats were fed with CCl4 in corn oil for 10 weeks. The total protein and albumin were checked in peripheral blood samples to confirm hepatotoxicicity. The rats were divided into 4 groups; animals without any intervention, hepatotoxic animal model, hepatotoxic animal model with PRP and hepatotoxic animal model with PRP vehicle. After 1 week, the liver removed, fixed, sectioned by IUR method and stained with H&E. The volume of the vessels was calculated by stereology method. The results were ANOVA.
CCL4 had no impact on the microvasculature volume compared with normal control. PRP led to a significant decrease in the microvasculature of hepatotoxic liver (P=0.003). CCl4 also led to a significant increase in central vein and portal vein volume compared with control (P=0.002, P=0.001). The central vein volume reduced significantly by PRP treatment (P=0.001) but it has no impact on portal vein volume. Hepatic artery volume did not change by treatment of both CCl4 and PRP.
PRP had adverse effects on microvasculature and macrovasculature of hepatotoxic liver. The decrease in blood vasculature volume may result on a reduction in blood flow.

Keywords: Microvasculature, Microvasculature, Hepatotocicity, Platelet rich plasma

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Cancer Biology

Role of A kinase anchoring protein 12 (AKAP12) as a potential tumour suppressor in breast cancer

Regina You Zhen Soh [1], Jia Pei Lim [1], Pei Jou Chua [1], Boon Huat Bay [1]
[1] Department of Anatomy, Yong Loo Lin School of Medicine, National University of Singapore, Singapore

A kinase anchoring protein 12 (AKAP12), also known as Gravin, is a protein kinase C substrate and a scaffolding protein for many key signalling proteins, including protein kinase C (PKC), PKA, cyclins and actin cytoskeleton. The expression of AKAP12 is suppressed in human malignancies such as prostate, ovarian and gastric cancers where it functions as a potential tumour suppressor. Nevertheless, not much is known of the exact role of AKAP12 in breast cancer progression and hence, we evaluated the role of AKAP12 in breast cancer in this study. The gene and protein expression of AKAP12 were firstly evaluated in a panel of seven different breast cancer cell lines (MCF10A, MCF7, T47D, ZR751, AU565, Hs578T and MDA-MB-231). At the gene level, both MCF10A normal breast cells and Hs578T triple negative breast cancer cells showed high expression of *AKAP12*. However, at the protein level, AKAP12 protein was highly expressed in the MCF10A normal breast cell line. Immunofluorescence staining followed by confocal microscopy revealed that AKAP12 is predominantly expressed in the cytoplasm. To evaluate the expression of AKAP12 in the clinical setting, *AKAP12* gene expression was subsequently determined in TissueScan breast cancer cDNA array panel I which consisted of 48 breast tissue cDNA samples. It was found that *AKAP12* gene expression was higher in normal breast tissues, similar to the protein expression in breast cell lines. Future functional analysis of the AKAP12 protein will be performed to elicit a better understanding of its role as a tumour suppressor particularly in breast cancer.

The Effect of Argan Oil with Electromagnetic Field Exposure on Open Wound in Rats

[1]Yanti Rosli, [1]Solehah Sarahanim Othman, [1]Hamid, [1]Sabarina Ismail,
[1] Kaswandi Mohd Ambia, [2] Normalawati Shamsudin, [1]H. L. Hing
[1] Biomedical Science Program, PPSDKG, Faculty of Health Science,
UKM Jalan Raja Muda Abdul Aziz, 50300 Kuala Lumpur, Malaysia
[2] Electron Microscope Unit, Faculty of Science and Technology,
43600 UKM Bangi, Selangor, Malaysia

Previous study that using electromagnetic field in wound healing can facilitate, improve and produced positive result in wound healing process. This study was conducted to study the effect of argan oil on open wound healing with the exposure of EMF in rats. Ninety male wistar rats (200-250g) were used and divided into three treatment group which are PBS negative control, *Solcoseryl* gel positive control and argan oil with the exposure of EMF treatment. Each treatment group then divided into five group according to the days the rats had being sacrificed. A total of six rats sacrified according appointed day. About 6mm open wound being induced at rats dorsal on day 0 and treatment is given once a day. Open wound being treated with argan oil will be exposed to EMF (8mT) about 30 minutes. Six rats sacrificed on the first day (after 24 hours wound being induced), third, sixth, tenth and fourteenth day. The parameter of this study are macroscopic observation (percentage wound healing), biochemical test (total protein, uronic acid and hydroxyproline) and histopathology analysis of wound tissue. Macroscopic observation showed that argan oil that being exposed to EMF treatment significantly increased ($p < 0.05$) during third (41.01 ± 5.597) %, sixth (62.86 ± 1.89) % and tenth day (94.39 ± 0.719) % compared both of control treatment. The total protein of argan oil that being exposed to EMF treatment significant ($p < 0.05$) lower on first day (24.07 ± 8.57) µg/ml and significantly ($p < 0.05$) increase on fourteenth day (23.49 ± 8.84) µg/ml compared PBS negative control group. The level of acid uronic of argan oil that being exposed to EMF treatment significantly lower ($p < 0.05$) on first day (25.62 ± 11.14)µg/ml and increase significantly on tenth (67.78 ± 10.84)µg/ml and fourteenth (83.28 ± 9.29)µg/ml compared both control group. Hydroxyproline level of argan oil that being exposed to EMF treatment also significantly lower ($p < 0.05$) on first day (2.04 ± 0.39)µg/ml and significantly increase ($p < 0.05$) on fourteenth day (6.53 ± 0.64)µg/ml. Histopatology analysis showed that wound that being treated with argan oil that being exposed to EMF formed granulation tissue at faster rate and more dense than both control group. Within in four day, between day sixth until day tenth, there was an increase about 15 percent of healing process of open wound for argan oil that being exposed with EMF group compared negative group. As a conclusion, argan oil that being exposed to EMF treatment give a better result and can accelerate the healing time of wound and its have a great potential to be develop as wound healing agent in future.

Keywords: electromagnetic field, EMF, wound healing, argan oil

Regulation on the gene expressions of ASPP family members

KK Chan, Oscar GW Wong, and Annie NY Cheung
Department of Pathology, Faculty of Medicine, The University of Hong Kong, Hong Kong

Introduction: ASPP (apoptosis-stimulating protein of p53) is a group of proteins which can act as either a stimulator or inhibitor to control p53-mediated apoptosis so that their expressions in cancer cells are essential for their survival. There are three members known as ASPP1, ASPP2 and iASPP so far. Dysr egulation of ASPP family members has been found in gestational trophoblastic disease (GTD). One of t he malignant forms of GTD, choriocarcinoma, exhibited downregulation of ASPP1/2 but elevated level of iASPP when compared to normal trophoblastic tissues. Nevertheless, the underlying mechanisms for t he dysregulation of their expressions in choriocarcinoma are unclear. Methylation and acetylation are tw o major epigenetic regulations while miRNA also plays a regulatory role on gene expression. Our *in sil co* analysis suggested that the 3'UTR of ASPP2 may has a potential binding site for miR-205. In this study, these regulations on ASPP family members in choriocarcinoma were investigated and these findin gs provide a better understanding on the pathogenesis of choriocarcinoma.

Objectives: To investigate the gene regulation on ASPP family members in choriocarcinoma.

Experimental setup: Choriocarcinoma cell lines, JEG-3 and JAR, were used as model and compared with normal trophoblastic cell lines, TEV-1 and HTR-8/SVneo cells on their ASPP expressions. 5-azacytidine (5-Aza) was used to study the effect of methylation while Trichostatin A (TSA) was used to study the effect of acetylation on ASPP expressions in trophoblastic cell lines. The CpG methylation of ASPPs' promoter regions were detected by bisulfite sequencing, whereas chromatin immunoprecipitation (CHIP) qPCR assay was used to examine the acetylation status. miR-205 inhibitor was used to manipulate ASPP2 expression. The changes in ASPP expressions were measured by both western blotting and real-time PCR.

Results and Outcome: The CpG sites on the promoter regions of ASPP1/2 were found to be unmethylated, implying that regulation on ASPP1/2 through methylation was unlikely. However, addition of 5-Aza increased both the mRNA and protein expressions of ASPP1 and ASPP2 in JEG-3 and JAR cells, suggesting that other genes affected by 5-Aza may act on ASPP1/2. On the other hand, TSA treatment increased the expression of ASPP1 in JEG-3 and JAR cells. CHIP qPCR assay also reflected histone hyperacetylation on its promoter region after TSA treatment, suggesting that acetylation may be responsible for regulating ASPP1 but not ASPP2 in choriocarcinoma. Neither methylation nor acetylation has effects on iASPP expression. On the other hand, choriocarcinoma cells had higher miR-205 expression than normal trophoblastic cells. Inhibition on miR-205 upregulated ASPP2 expression, indicating an regulatory role of miR-205 on ASPP2. Altogether, our results indicate that the gene expressions of ASPP family members are regulated by different mechanisms.

Poster Abstract
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Neuroscience

Electrical stimulation rescues dopaminergic neurodegeneration in the dorsal raphe nucleus of vulnerable depressive-like rats

Sharafuddin Khairuddin [1], Wei Ling Lim [1,2], William Fung Wai Lun [1],
Yasin Temel [3], Lee Wei Lim [1]
[1] School of Biomedical Sciences, Li Ka Shing Faculty of Medicine,
The University of Hong Kong, Hong Kong
[2] Department of Biological Sciences, Sunway University, Malaysia
[3] Departments of Neuroscience and Neurosurgery, Maastricht University, the Netherlands

Electrical stimulation is a potential treatment for patients with depression. In this study, we investigate the effects of high-frequency stimulation (HFS) on various depressive-like behaviors using the stress resilience and vulnerable rat depression models. Our results demonstrated that animals with 3 weeks chronic unpredictable stress (CUS) induced 51% of animals exhibiting reduced sucrose consumption, separating the resilience and vulnerable group of CUS-induced model. CUS vulnerable sham animals demonstrated anxiety-like behavior, decreased motivation and increased immobility compared to that of the resilience group, implicating high susceptibility of vulnerable individuals to the CUS procedure. Interestingly, ventromedial prefrontal cortex (vmPFC HFS) significantly reduced anxiety response, increased hedonia and motivation levels for food intake in the vulnerable group compared to the resilience group. HFS in the vmPFC and lateral habenula also showed reduced behavioral despair in both CUS vulnerable and resilience group. In histochemistry, our results demonstrate that vmPFC HFS rescued the stress-induced dopamine neuron degeneration in the dorsal raphe nucleus, as well as increased hippocampal neurogenesis of stressed-vulnerable animals. In conclusion, these results suggest that vmPFC HFS effectively restores depressive-like behaviors by mechanisms of dorsal raphe dopaminergic neurons restoration in the vulnerable CUS-induced model.

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Cancer Biology

The Anti-inflammatory Effect of Cysteamine in Experimental Autoimmune Uveitis through the Downregulation of Interleukin-22 and Its Receptor

Yejin Kim[1], Soyoung Cheon[1], Junmyung Lee[1], Tae Wan Kim[2], Hyeong Gon Yu[2], Wang Jae Lee[1], Jae Seung Kang[1,*]
[1] Laboratory of Vitamin C and Anti-oxidant Immunology, Department of Anatomy and Cell Biology, Seoul National University College of Medicine, Korea
[2] Department of Ophthalmology, Seoul National University College of Medicine, Korea

IL-22 has both a pro- and anti-inflammatory role and its levels are increased in patients with autoimmune noninfectious uveitis, but the mechanism of this upregulation is unclear. Cysteamine, a degradation product of cysteine, has demonstrated anti-inflammatory properties, but its regulatory role in the pathogenesis of uveitis has not been explored. In this study, we investigated whether cysteamine has therapeutic effects in experimental autoimmune uveitis (EAU) and if so, how it relates to the regulation of IL-22 and its receptor expression. Our study showed that serum levels of IL-22 in uveitis patients were increased compared to those in healthy donors. In addition, IL-22 stimulation increased the production of MCP-1 and the proliferation of human retinal pigment epithelial cell line, ARPE-19 via the activation of p38MAPK and NK-κB. In the EAU model, daily administration of cysteamine significantly delayed and decreased ocular inflammation compared to untreated mice. The amelioration of EAU in cysteamine-treated mice correlated with decreased number of IL-17-positive CD4+ T cells and reduced production of IL-22. Interestingly, we also confirmed that RORγt expression and the production of IL-22 were inhibited by cysteamine in treated PBMCs from uveitis patients. In addition, IL-22Rα expression in ARPE-19 was increased in a PI3K/Akt dependent manner, but this was reduced by cysteamine treatment. We demonstrated that IL-22 can play a key role in the development of EAU and cysteamine has an anti-inflammatory effect in EAU, which may be associated with the decreased expression of IL-17-positive CD4+ T cells and reduced IL-22 production.

Keywords: Experimental autoimmune uveitis, IRBP, Cysteamine, IL-22, Th17, IL-22 receptor

Regulation of Sodium/potassium-transporting ATPase subunit gamma by HNF-1β in ovarian clear cell carcinoma cells

Huiju Lim, Oscar Gee-Wan Wong, and Annie Nga-Yin Cheung
Department of Pathology, Faculty of Medicine, The University of Hong Kong, Hong Kong, China

Introduction: Ovarian clear cell carcinoma (OCCC) is a histological subtype of ovarian cancer which generally displays poorer prognosis than other subtypes due to its high chance of chemoresistance. In Asia, the relative proportion of OCCC among various subtypes of ovarian cancer is higher when compared with Caucasian women. HNF-1β is a transcription factor that is expressed in nearly all OCCC, making it useful for the diagnosis of OCCC. However, its roles in OCCC carcinogenesis are unclear, mainly because its transcription targets in cancer cells are poorly characterized. FXYD2 (encoding the γ-subunit of Na+, K+-ATPase) is a transcription target of HNF-1β in kidney cell. It is also one of the genes identified in the OCCC specific gene signature.

Hypothesis: FXYD2 is a transcription target of HNF-1β in OCCC cells.

Methodology: The binding of HNF-1β to the FXYD2 promoter was tested by chromatin immunoprecipitation (ChIP), and the effect of HNF-1β knockdown on FXYD2 expression was tested by reverse transcription quantitative PCR (RT-qPCR). We used two OCCC cell lines OVMANA and KK that express high levels of HNF-1β as models.

Results and Discussion: Our results showed that antibody against HNF-1β but not the negative control IgG could enrich DNA sequences containing the FXYD2 promoter, suggesting that HNF-1β indeed can bind to the FXYD2 promoter. Knocking down HNF-1β with siRNA approach also resulted in down-regulation of FXYD2 transcripts. Our results suggest that HNF-1β can directly control the expression of FXYD2 in OCCC cells.

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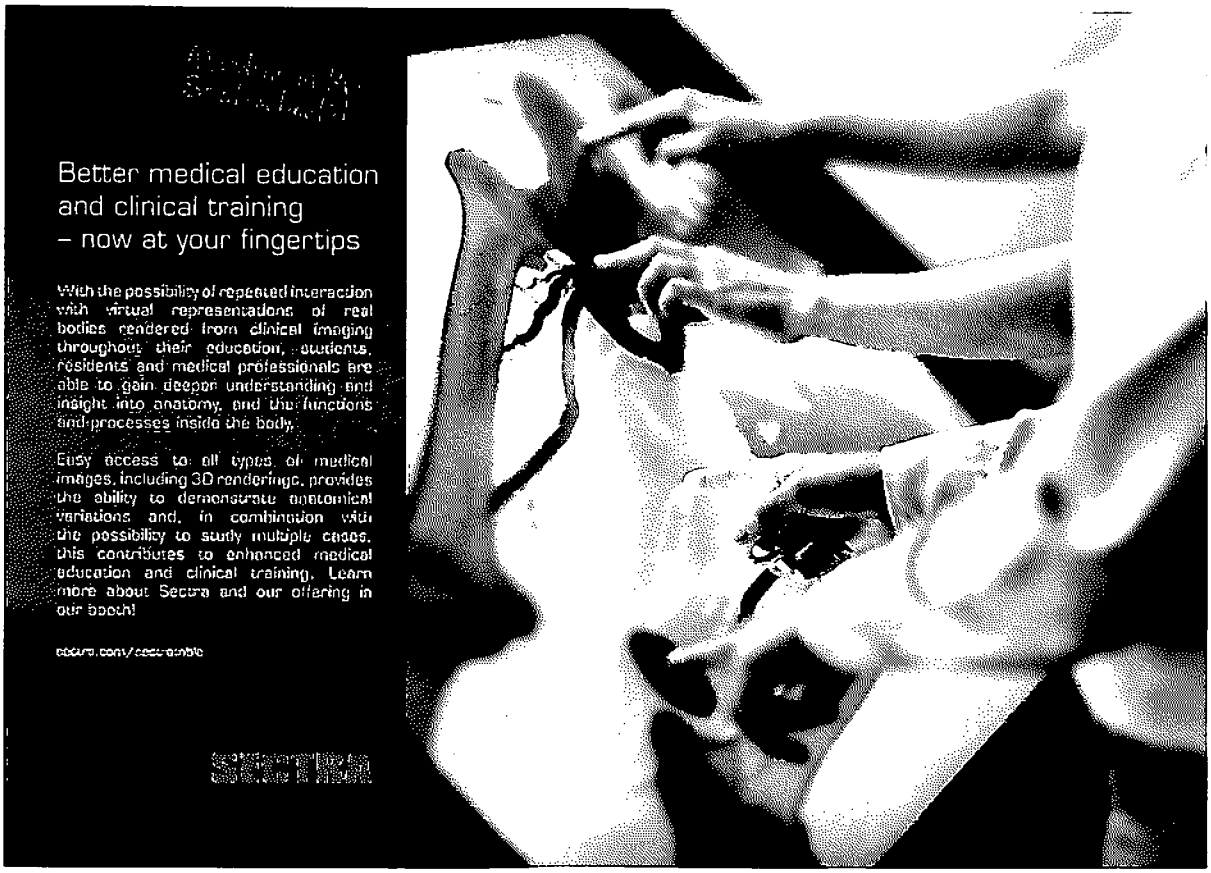

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"The visualization table allows us to modernize and increase the quality of our teaching," says Sandra Ceccatelli, Head of the Department of Neuroscience in charge of the anatomy teaching at KI. "The table opens up unique opportunities to convey basic knowledge about the body, offering a whole new way to teach anatomy to our students."

Interacting with 3D images of actual human bodies helps students at KI to gain an enhanced understanding of the human anatomy and also provides opportunities to see variations in the anatomy between individuals.

"To study a real body is the best way to learn anatomy. It has previously been the preserve of medical and dental students, but now all students studying anatomy will have similar possibilities," says Professor Björn Meister, Department of Neuroscience, KI.

Sharing competence and knowledge

To further enhance the benefits of the Sectra Table, KI has chosen to connect to Sectra's education portal, a unique concept for sharing patient cases with other seats of learning around the world that use Sectra's table. This allows KI to share in materials from other universities around the world that use the visualization table.

"We will use this chance to share our competence and knowledge with other teaching institutes around the world," says Björn Meister.

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Knowledge and passion

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Date	Service Hours
Sunday, 4 December 2016	08:30 – 17:00
Monday, 5 December 2016	08:30 – 17:30
Tuesday, 6 December 2016	08:30 – 14:00

Certificate of Attendance

Certificate of Attendance will be issued to delegates who have pre-registered during their registration. For onsite registrants, the Certificate of Attendance will be issued after the registration. No certificate will be issued after the Conference.

Inclement weather arrangement

In case the Tropical Cyclone Warning Signal No. 8 (or above) or Black Rainstorm Signal is hoisted, the following arrangements will apply:

(a) For Conference sessions not yet started

If either of the warnings is hoisted or in force at or after 6:00 a.m.	All sessions commencing before 2:00 p.m. will be cancelled automatically.
If either of the warnings is hoisted or in force at or after 11:00 a.m.	All afternoon sessions commencing at any time from 2:00 p.m. and before 6:00 p.m. will be cancelled automatically.

(b) For Conference sessions already started

When Tropical Cyclone Signal No. 8 or above is hoisted	All Conference sessions will be suspended immediately.

When Tropical Cyclone Warning Signal No. 3 or below or Red or Amber Rainstorm Signal is in force, it is assumed that all Conference sessions will be held as scheduled unless an announcement to the contrary has been made by the University.

Shops and restaurants near Conference Venue

It takes 5-minute drive/ taxi ride from HKU Medical Campus to the Arcade at Cyberport. Address: 100 Cyberport Road, Cyberport, Pokfulam

The Arcade, situated in the core zone of Cyberport, is a 27,000 m^2 retail and entertainment complex. It has a cinema, restaurants featuring fine cuisines, a variety of lifestyle shops and a supermarket. It is a one-of-a-kind, high-end shopping arcade in the Southern District of Hong Kong.

Shop directory:
http://www.cyberport.hk/en/arcade/shop_directory

Recreational information

Discover Hong Kong
www.discoverhongkong.com
It is a website of the Hong Kong Tourism Board, the official organisation overseeing tourism in Hong Kong. It contains highlights of attractions and tips for tourists.

The Peak and the Peak Tram
www.thepeak.com.hk

The Harbour view and the Star Ferry
www.starferry.com.hk

Contact us

Telephone: (852) 3917 9259 / 3917 9540
Fax: (852) 2817 0857
Email: iascbc16@hku.hk
Website: www.sbms.hku.hk/iascbc2016

For more details, please visit the website of IASCBC 2016:

http://www.sbms.hku.hk/iascbc2016/

Co-organised by:

The University of Hong Kong
Neuroscience Research Centre
Strategic Research Theme of Cancer
Strategic Research Theme of Neuroscience

Li Ka Shing Faculty of Medicine, HKU
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