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# Welcome Message

Dear Colleagues, Participants and Friends,

## **Welcome to the 19<sup>th</sup> Congress of Asian Pacific Society of Respirology (APSR)!**

APSR takes great pleasure in welcoming you to the upcoming 19th Congress of the Asian Pacific Society of Respirology (APSR 2014). It is a great privilege to host participants from the Asia Pacific region – a distinguished community of about 2,000 clinicians and researchers specializing in the areas of pulmonology, thoracic surgery, internal medicine, critical care, pediatrics, as well as graduate students in the field of respiratory medicine.

We are honored to have an esteemed faculty of international, regional and local experts who will be leading the postgraduate courses and workshops, plenary sessions and concurrent tracks of symposia. Together with the poster and oral presentations, our 4-day program is replete with scientific and educational sessions exploring the latest developments in the management of respiratory illnesses.

On behalf of the APSR 2014 Organizing Committee, we wish you a stimulating and rewarding APSR 2014 experience, and an enjoyable stay in Bali, Indonesia!

Your sincerely,



**Faisal Yunus**  
President of APSR 2014  
Local Congress Committee



**Arifin Nawas**  
Chairman of Indonesia Society of  
Respirology

# About the Asian Pacific Society of Respiriology

The Asian Pacific Society of Respiriology was established in 1986. The objectives of the Society are the advancement and promotion of knowledge of the respiratory system in health and disease. It strives to encourage research, improve clinical practice through teaching, increase awareness of health problems in the area and promote the exchange of knowledge among respirologists in the Asia Pacific region. The Society has since grown to approximately 15,000 members from over 40 countries/regions.

## Aims

- Promoting and coordinating activities in the field of respiratory medicine
- Fostering research activities in the field of respiratory medicine
- Organizing and coordinating regular congresses and occasional meetings
- Producing regular publications, including a Newsletter, the APSR Respiratory Updates and Respiriology, a journal of international repute

## Officers

President

Arth Nana

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Teresita de Guia

Editor-in-Chief

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Chunxue Bai

Chairperson, Education Committee

David C L Lam

Representative, Editorial Office

Peter Eastwood

Congress President, 18th Congress of the APSR

Toshihiro Nukiwa

Congress President, 19th Congress of the APSR

Faisal Yunus

## Heads of Assemblies

Asthma

Bronchoscopy and Interventional Techniques

Cell and Molecular Biology

COPD

Clinical Allergy & Immunology

Clinical Respiratory Medicine

Critical Care Medicine

Environmental & Occupational Health and Epidemiology

Interstitial Lung Disease

Lung Cancer

Pediatric Lung Disease

Pulmonary Circulation

Respiratory Infections (non-tuberculosis)

Respiratory Neurobiology and Sleep

Respiratory Structure and Function

Tuberculosis

Kittipong Maneechotesuwan

Takashi Ishida

Yasuhiro Yamauchi

Masaharu Nishimura

Shu Hashimoto

Philip Eng

Yoshiki Ishii

Soon-Hee Jung

Masahito Ebina

Kwun Fong

Albert Li

Hiroshi Kimura

Jun-Hee Woo

Kazuo Chin

Yasutaka Nakano

Charles Yu

# About the Indonesian Respiratory Society

The Indonesian Respiratory Society (ISR; Indonesian: Perhimpunan Dokter Paru Indonesia, PDPI) is a non-profit organization consisting of hundreds of pulmonology and respiratory physicians from across the country.

Founded in 1973, ISR is the paramount of holistic and state-of-the-art for asthma/COPD, thoracic oncology, lung infections and tuberculosis diagnosis and management; intervention, critical care and emergency medicine; sleep medicine; environmental lung disease and tobacco control; and translational medicine research in Indonesia.

## Mission

- To promote the best of quality of lung and respiratory health in Indonesia, by continuing, life-long improvement of the knowledge and skill of its members.
- To ensure and improve the welfare of its members as they are devoting themselves for the community
- To maintain and improve the collegial harmony between its members

## Officers and Board Members

### Advisors

Muhammad Amin  
Faisal Yunus  
Taufik  
Suradi  
Ida Bagus N. Rai  
Tamsil Syafiuddin

### Officers

Chairman	Arifin Nawas
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Co-chief	Susanti Djajalaksana
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Members	Amira Permatasari Tarigan Deddy Herman Dewa Made Artika

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# APSR 2014 Organizing Committee

## **Congress Advisors**

Arth Nana (President of APSR)  
Arifin Nawas  
Menaldi Rasmin  
Hadiarto Mangunnegoro  
Muhammad Amin  
Dianiati Kusumo Sutoyo

## **Congress President**

Faisal Yunus

## **Congress Vice-President**

Ida Bagus Ngurah Rai  
Wiwien Heru Wiyono  
Suradi  
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## **Executive Secretary**

Sita Laksmi Andarini  
Erlina Burhan

## **Treasurer**

Fathiyah Isbaniah  
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Wahju Aniwidyaningsih  
Sita Laksmi Andarini  
Jamal Zaini  
Triya Damayanti  
Wahyuningsih  
Susanthy Djajalaksana  
Helmia Hasan  
Oea Khairsyaf  
Resti Yudhawati  
Prasenohadi

**Fund Raisers**

Priyanti ZS  
Sutji Astuti Mariono  
Alex Ginting Kaliaga Suka  
Prajnaparamita  
Budhi Antariksa  
Rita Rogayah

**Social Programme**

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Ida Bagus Suta  
I Dewa Made Artika  
Putu Wardana

**Exhibition & Congress Facilities**

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Dicky Soehardiman  
Anang Isnin Marhana  
Temmasonge R. Pakki  
Nur Ahmad Tabri  
Amira Permatasari Tarigan  
Fahmi Alatas

**Publication**

Agus Dwi Susanto  
Isnu Pradjoko  
Heidy Agustin  
Frans Abednego Barus  
Diah Handayani

**APSR 2014 Secretariat  
Head Office**

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Pulmonology and  
Respiratory Medicine  
Faculty of Medicine,  
University of Indonesia  
Persahabatan Hospital,  
Jalan Persahabatan Raya  
No.1  
Rawamangun, Jakarta  
13230  
Indonesia

**APSR 2014 Congress Secretariat Office c/o MIMS Asia****Indonesia:**

Aquarius Building, 3rd Floor  
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No. 7  
Pondok Indah, Jakarta  
Selatan 12240  
Indonesia

**Overseas queries:**

6 Shenton Way,  
OUE Downtown 2 #15-08  
Singapore 068809  
Tel: + 65 6290 7400





# AWARDS

APSR MEDAL

APSR RESEARCH AWARDS

Michiyoshi Harasawa Research Award

Ann J. Woolcock Research Award

FUKUCHI AWARDS

for the Best Paper in Respiriology

YOUNG INVESTIGATOR

FROM APSR, ATS, ERS and TSANZ

APSR TRAVEL AWARDS



# AWARDS

## APSR Medal

*The APSR Medal is given by the APSR in appreciation for the awardee's commitment to the advancement of respirology in the Asian Pacific region, including major achievements in advocacy, teaching, clinical practice and leadership, enshrining excellence in respiratory research and research translation, and for the exceptional contribution made to the growth of the Asian Pacific Society of Respirology.*



### **Christine Jenkins**

*Senior Staff Specialist  
Department of Thoracic Medicine  
Concord Hospital  
Sydney, Australia*

Dr. Christine Jenkins is currently Senior Staff Specialist in Thoracic Medicine at the Concord Hospital and Clinical Professor at the Concord Clinical School in Sydney, Australia. She is also the Head of Respiratory Discipline at the University of Sydney and Head of the Respiratory Trials and Professorial Fellow at the George Institute for Global Health also in Sydney, Australia.

Dr. Jenkins is a founding member of the Global Guidelines for Obstructive Lung Disease (GOLD) which is now the key international group developing, guiding and implementing best practice in COPD, a chronic lung disease that is a leading global cause of death and disability, related to tobacco smoking, indoor and outdoor pollution. She is also currently a member of a 3-person international adjudication committee convened by the US Food and Drug Administration, to adjudicate asthma deaths in a series of asthma trials. These were mandated by the FDA to assess the safety of one class of asthma drugs, and involve 35,000 patients, the largest ever in asthma.

Dr. Jenkins has been actively involved in local and international clinical guidelines development in respiratory medicine for over 20 years. Apart from her teaching commitments and contributions in continuing education initiatives for General Practitioners, Dr. Jenkins also leads a clinical research group focusing predominantly in the management of COPD and asthma.



# AWARDS

## Michiyoshi Harasawa Research Award

*The Michiyoshi Harasawa Research Award is given in memory of the late Dr. Harasawa, who died September 2001. The award is given to a person who embodies the leadership and respect of his/her peers and who excels in the field of Pulmonary Medicine.*

*Dr. Harasawa is highly respected professor of Medicine from Japan, a clinician and a researcher in the field of Pulmonary and Geriatrics rolled into one. His leadership traits with so much dedication and commitment in the field of respiratory medicine, led him to pioneer and form the international organization of the Asian Pacific Society of Respirology (APSR) in 1986 that became a highly structured and leading society of pulmonologists in the Asian Pacific.*



### **Kazuhisa Takahashi**

*Professor and Chairman  
Division of Respiratory Medicine,  
Juntendo University  
Tokyo, Japan*

Prof. Kazuhisa Takahashi is presently Professor and Chairman of the Division of Respiratory Medicine at the Juntendo University Faculty of Medicine and Graduate School of Medicine. He is currently serves as the Assistant Director of the Juntendo University Hospital.

Prof. Takahashi completed his medical and PhD degrees from the Juntendo University and underwent a post-doctoral fellowship at the Massachusetts General Hospital Division of Surgical Oncology/Harvard Medical School in Boston, USA. He is a member of the American Thoracic Society and the American Society of Clinical Oncology. In 2013, Prof Takahashi was the Chair of Local Scientific Committee of the 18<sup>th</sup> Congress of the Asian Pacific Society of Respirology (APSR2013) held in Yokohama, Japan.

# AWARDS

## Ann Janet Woolcock Research Award

*The Ann Janet Woolcock Research Award is given in the memory of the late Dr. Woolcock who died February 2001. The award is given to a person who embodies the leadership and respect of his/her peers and who excels in the field of Pulmonary Medicine.*

*Dr. Woolcock is an eloquent professor of Medicine from Australia, a clinician and researcher in the field of pulmonary medicine with particular specialization on the physiology and epidemiology of Bronchial Asthma. Her dedication to the field of Pulmonary Medicine developed her analytical mind and enthusiasm to promote researchers. Her failing health did not prevent her in continuing her quest of unveiling the science of asthma. She helped Dr. Harasawa to form the international organization of the Asian Pacific Society of Respirology (APSR) in 1986.*



### **Greg King**

*Head, Respiratory Investigation Unit  
Department of Respiratory Medicine  
Royal North Shore Hospital;  
Conjoint Associate Professor  
Sydney Medical School, University of Sydney*

Gregory G. King is the Research Leader of the Airway Physiology and Imaging Group at the Woolcock Institute of Medical Research and staff specialist in the Department of Respiratory Medicine at Royal North Shore Hospital, where he directs the asthma service and heads the Respiratory Investigation Unit. He is a National Health and Medical Research Council Practitioner Fellow (2010) and Conjoint Associate Professor of the Northern Clinical School of the University of Sydney (2006). He currently supervises PhD and other postgraduate students of the University of Sydney.

Dr. King is a medical graduate of Otago University in New Zealand and a PhD of Sydney University on the topic of airway closure in asthma. He completed Postdoctoral Research Fellowships at Sydney University and in the Pulmonary Research Laboratory of the University of British Columbia in Vancouver. He is a member of the American Thoracic Society (ATS), the American Physiological Society (APS) and Thoracic Society of Australia and New Zealand (TSANZ) and serves on committees of the ATS Structure and Function Assembly and of the TSANZ.

His research interests focus on the use of advanced 3-dimensional (3D) imaging and complex lung function as research and clinical tools in sleep apnoea, pulmonary vascular disease, including pulmonary embolism, lung cancer, asthma, chronic obstructive pulmonary disease, and airway hyper-responsiveness. Dr. King and his group use 3D imaging and analysis (including SPECT, PET, CT and MRI), complex lung function tests (including forced oscillation technique and multiple breath nitrogen washout) and time-series analyses of respiratory function.

# AWARDS

## Fukuchi Award for the Best Original Paper in Respiriology

*Respirology and Wiley Publishing are delighted to present the 2014 'Fukuchi Award' for the Best Original Paper published in Respirology, the official journal of the APSR, in 2013.*

*This prestigious award has been named after Professor Fukuchi who was President of APSR between 2004 and 2006 and who was also the Editor of Respirology between 1996 and 1999. The decision on the best paper published in Respirology was made by a panel of world-renowned international researchers including Professors Michael Niederman, Antonio Anzueto and John Mastronarde from the USA, Professor Chunxue Bai from China, Professor Albert Li from Hong Kong and Dr Christina Spengler from Switzerland.*

*The 2014 winning paper is entitled "Alterations in inflammatory, antiviral and regulatory cytokine responses in peripheral blood mononuclear cells from pregnant women with asthma" and was submitted by Dr Rebecca Vanders from Australia.*



### **Rebecca Vanders**

*Post-Doctoral Researcher  
Department of Microbiology and Immunology  
School of Biomedical Sciences, Faculty of Health  
University of Newcastle, NSW, Australia*

Rebecca Vanders is an early career Post-Doctoral Researcher in the Department of Microbiology and Immunology, School of Biomedical Sciences, Faculty of Health, at the University of Newcastle, NSW, and Australia. Dr. Vanders is based at the Hunter Medical Research Institute in Newcastle where she works with the leaders of the Microbiology, Asthma and Airways Research Group, whose primary research areas include allergy, asthma, influenza, respiratory diseases, inflammation and host defence. Dr. Vanders received her Bachelor of Biomedical Science (Honours Class I) from the University of New England in 2008, followed by a PhD (Medicine) at the University of Newcastle in 2012. Her primary research focuses on maternal immunity and the host responses to respiratory virus infections during pregnancy. This also includes how the presence of asthma can further confound the immune response during pregnancy. In 2013, Dr. Vanders commenced her post-doctoral research, funded by the Astra Zeneca/Thoracic Society of Australia and New Zealand Early Career Research Fellowship. This fellowship is allowing her to continue her research into influenza infections during pregnancy and asthma, and the potential for the development of therapies that may improve the maternal immune response for pregnant women and their babies. Her research utilizes both human in vitro and animal in vivo models to make important observations of immune suppression that is induced by respiratory virus infections in pregnancy. Applying these two methods of study will allow for future translational research in order to develop new therapeutic strategies during pregnancy.

Dr. Vanders currently has 9 national/international conference presentations, 5 first-author refereed journal articles including in *Thorax*, *Journal of Infectious Diseases* and *Respirology* as well as another 2 currently under submission.



# AWARDS

## Young Investigator Awards

Outstanding works by abstract presenters at/under the age of 40 years at the time of abstract submission were considered for the Young Investigator Awards. These highly-coveted awards are offered based on the scientific merit of the submitted abstracts.

### APSR Young Investigator Awardees



**Jun-Pyo Myong**

*Assistant Professor  
Department of Occupational and Environmental Medicine  
Seoul St. Mary's Hospital  
Korea*

**Presentation:** The Association Between Occupational and Environmental Asbestos Exposure and Asbestosis in Korea - Asbestos Health Damage Relief System



**Yang Tian**

*Attending Physician  
Department of Respiratory Critical Care Medicine  
First Affiliated Hospital-Xi'an Jiaotong University  
Shaanxi, China*

**Presentation:** miR-15a Induces Cell Apoptosis and Inhibits Metastasis by Targeting BCL2L2 in Non Small Cell Lung Cancer

### ATS Young Investigator Awardees



**Katherine Rivera-Spoljaric**

*Assistant Professor in Pediatrics  
Division of Pediatric Allergy, Immunology and Pulmonary Medicine  
Washington University School of Medicine  
Missouri, USA*

**Presentation:** Identifying Early Signs and Symptoms of Impending Asthma Exacerbations in Children



**Rebecca E. Sell**

*Assistant Clinical Professor and  
Associate Program Director, Internal Medicine Residency  
University of California, San Diego  
California, USA*

**Presentation:** Difference in Outcomes between Senior Physician and Medicine Resident-led Code Blues

# AWARDS

## Young Investigator Awards

### ERS Young Investigator Awardees



**Jiaying Luo**

*Technician-in-charge  
State Key Laboratory of Respiratory Disease  
Guangzhou, China*

**Presentation:** The Relationship Between Environment and the Prevalence of Allergy Rhinitis in Pre-School Children in Guangzhou City



**So Young Yang**

*Clinical Assistant Professor  
Department of Anesthesiology and Pain Medicine  
Chung-Ang University Medical Center  
Seoul, Republic of Korea*

**Presentation:** Associations of sRAGE and S100A12 on Postoperative Respiratory Complications According to Different Two Ventilation Modes after Laparoscopic-Assisted Colorectal Surgery

### TSANZ Young Investigator Awardee



**Luke Garratt**

*Research Assistant  
Telethon Kids Institute  
Perth, Australia*

**Presentation:** Identification of MMP Dysregulation in Early CF – Evidence Based Rationale for Anti-protease Therapy

# AWARDS

## APSR Travel Awardees

*A limited number of travel awards are given based on the scientific merit of the submitted abstracts, and is aimed at partially supporting investigators from developing regions to attend the congress. Separately, presenting authors under age 35 who are members of the European Respiratory Society (ERS) are additionally considered for the APSR-ERS Travel Award.*

Vincent Yi-Fong Su

Ungky Agus Setyawan

Sumeet Nawani

Mohammad Hoseein Rahimirad

Yoshiko Ikeda-Maquiling

Md. Mamunur Rashid

Yasunori Ichimura

Sang Hoon Lee

Siti Kamariah Othman

Dhamith Nandadeva

Ariani Permatasari

Sawang Saenghirunvattana

Nguyen Thanh Thuy

Shaghayegh Rahimirad

Marvin Mendoza

Narongkorn Saiphoklang

Gusakov Andrey Andreevich

Devi Yolandha

Lv Xuejiao

Shuan-Shaun, Xie

Lalita Fernandes

Michael Trotter

Xu-Guang Guo

Supanee Siphurmsukskul

Esther Tan Qiao Li

Siti Nafsiah

Dmytro Butov

Dhamith Nandadeva





# SPONSORS & EXHIBITORS



# SPONSORS

## Gold Sponsor



## Silver Sponsor



## Bronze Sponsor



## Sponsored Symposia



## Sponsored Workshops



## APSR 2014 Trade Exhibitors





# EXHIBITORS

## Back of Exhibition Hall

### Tea Break Tables

NDD Medical Technologies	ERBE Elektromedizin GmbH	Carefusion Inc	FUJIFILM Asia Pacific Pte. Ltd	Boehringer Ingelheim	Dexa Medica	Medela AG
PT. SOHO Industri Pharmasi	Eli Lilly		AstraZeneca Oncology		PT Combiphar	
PARI GmbH	Qiagen Singapore Pte Ltd	Res Med Ltd	Omron	Olympus Singapore Pte Ltd	PT. Phapros	Beijing Continent Pharmaceutical Co. Ltd
	Vitalograph Ltd	PT Roche Indonesia			PT Otsuka Indonesia	
FAPSR lounge		Internet Café	Chiesi	PT Bayer Indonesia		
Trudell Medical International	Philips Electronics (Pte) Ltd	PT Zambon Indonesia				
				Covidien	PT Sandoz	Indonesian Asthma Foundation
					Boston Scientific Asia Pacific	American Thoracic Society (ATS)
Takeda Pharmaceuticals International GmbH	Vifor Pharma Asia Pacific Pte Ltd	Pfizer		Mundipharma Pte Ltd	GlaxoSmithKline	European Respiratory Society (ERS)
	PT Ferron Phar Pharmaceuticals					
						Asian Pacific Society of Respiriology (APSR)
		AstraZeneca		Daiichi Sankyo Co Ltd		Asian Pacific Society of Respiriology (APSR) Malaysia



Exhibition Hall Entrance







# APSR Meetings

APSR Business Meetings

APSR Assembly Meetings



# APSR MEETINGS

## APSR Business Meetings

Date	Time	Meeting	Venue
<b>Thurs</b> 13 <sup>th</sup> November	08:30 – 15:30	Executive Committee Meeting	Kintamani 4
	15:30 – 16:00	Annual General Meeting (AGM)	Uluwatu 6
<b>Fri</b> 14 <sup>th</sup> November	10:00 – 11:00	Council Meeting	Kintamani 4
	13:00 – 14:00	Leaders of en-bloc Membership Meeting (APSR, JRS, TSANZ, TSPCCM, PCCP, KATRD, ISR, SRS, MTS)	Kintamani 4
<b>Sat</b> 15 <sup>th</sup> November	08:00 – 09:30	APSR/ERS Leadership Meeting	Kintamani 4
	11:00 – 12:00	APSR/ATS Leadership Meeting	Kintamani 4
	14:00 – 15:00	Editorial Board Meeting	Kintamani 4
<b>Sun</b> 16 <sup>th</sup> November	09:00 – 10:00	Central Congress Committee Meeting	Kintamani 4
	10:30 – 11:00	APSR/KTS Leadership Meeting	Kintamani 4



## APSR Assembly Meetings

Date	Time	Meeting	Venue	
<b>Thurs</b> 13 <sup>th</sup> November	09:00 – 10:00	Clinical Allergy & Immunology	Taman Sari 2	
	09:00 – 10:00	Environmental & Occupational Health and Epidemiology	Kintamani 6	
	12:00 – 13:00	Clinical Respiratory Medicine	Kintamani 6	
	13:00 – 14:00	Tuberculosis	Taman Sari 2	
<b>Fri</b> 14 <sup>th</sup> November	09:00 – 10:00	Cell and Molecular Biology	Taman Sari 2	
	09:00 – 10:00	COPD	Kintamani 6	
	10:00 – 11:00	Asthma	Taman Sari 2	
	10:00 – 11:00	Respiratory Structure and Function	Kintamani 6	
	11:00 – 11:45	Respiratory Neurobiology and Sleep	Taman Sari 2	
	11:00 – 12:00	Bronchoscopy and Interventional Techniques	Kintamani 6	
	12:00 – 12:45	Lung Cancer	Taman Sari 2	
	12:00 – 13:00	Paediatric Lung Disease	Kintamani 6	
	15:00 – 16:00	Pulmonary Circulation	Taman Sari 2	
	15:00 – 16:00	Interstitial Lung Disease	Uluwatu 6	
	<b>Sun</b> 16 <sup>th</sup> November	10:00 – 11:00	Critical Care Medicine	Taman Sari 2







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# CONGRESS OVERVIEW

GENERAL INFORMATION

GUIDE FOR CHAIRS AND SPEAKERS

SOCIAL EVENTS

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# CONGRESS OVERVIEW

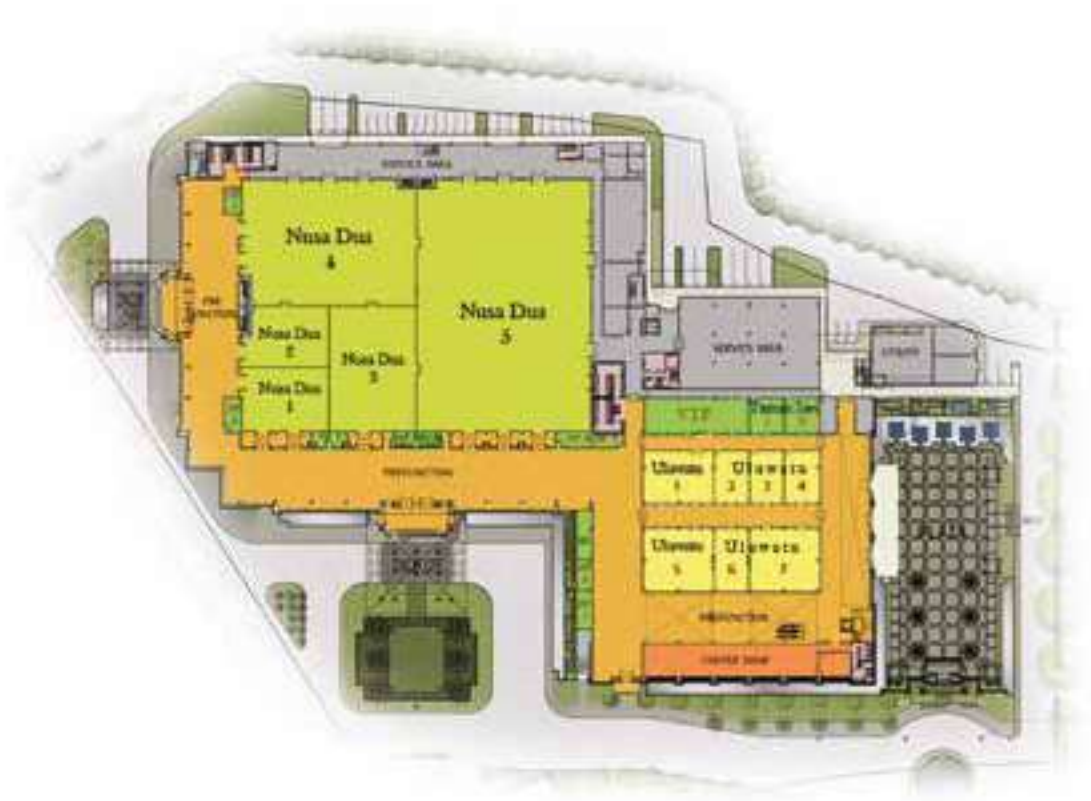
## General Information

### Congress Venue

Located in the Southern Bali, the Bali Nusa Dua Convention Center (BNDCC) is 20 minutes away by car from the International Airport and has an easy access to transport by a taxi. Taxi is recommended for up to 3 people travelling together, as long as the luggage fits into the boot/trunk. If you have a lot of luggage or are carrying a large item, such as a golf set, a surf board or a bicycle, it is advisable to get the hotel or your travel agent to pick

you up from the airport or rent a car (self drive or chauffeur driven). The rates are fixed and there is no surcharge for a midnight transfer as the drivers have a different working shift. En route to the center, you may get a glimpse of some areas of interest including the By Pass Ngurah Rai and Pratama Raya Street. The BNDCC is in the vicinity of restaurants, ATM, medical centers, beach, art market, museum, Pura Sampuh, or Bali Golf & Country Club.

### Floor Plan



### Internet Café

Free use of computers is available at the Internet Café located in the exhibition hall. The Internet Café is sponsored by Takeda Pharmaceuticals International GmbH.

### Certificate of Attendance

Attendees may collect their certificate of attendance on 15<sup>th</sup> November at the congress bag collection desk using the coupon from the congress badges.

### Accreditation

This congress is accredited by the Indonesian Medical Association for Indonesian physicians. The list of accreditation will be listed at the back of the congress certificate.

### Exhibition

Exhibition takes place in Nusa Dua 5. Only attendees holding a Delegate or Exhibitor badge may access the exhibition. Accompanying persons and children are not permitted to enter in the exhibition area.

Opening hours of the exhibition:

Fri 14 <sup>th</sup> November	07:30 – 18:00
Sat 15 <sup>th</sup> November	07:30 – 18:00
Sun 16 <sup>th</sup> November	08:00 – 12:00

### Hospitality Desk

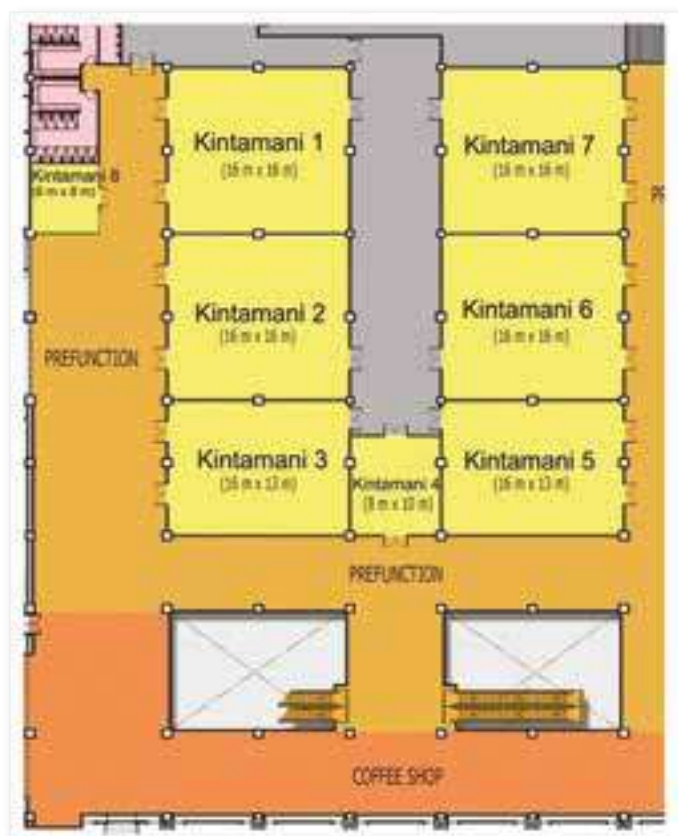
There is a hospitality desk located next to the registration desk in the South Lobby. You may seek advice regarding hotel, travel, shuttle bus schedule and cultural activities in Bali.

### Lost and Found

Please contact the hospitality desk.

### Prayer Room

Prayer room is located at the Kintamani 1.



# CONGRESS OVERVIEW

## Guide for Chairs and Speakers

Internet terminals will be available for use of the congress participants. The internet corner is located inside the exhibition hall. This service is supported by Takeda.

### Oral Sessions

**Chairs:** Please come to the session room 10 minutes before your session begins and take the next-Chair seat as at the front of the hall. Each presenter is allowed 3 minutes for questions after their presentation. Please do take strict note of the scheduled time.

**Speakers:** For all speakers of oral presentations, please load your presentations at the Speaker Room (Uluwatu 4):

The opening times for the speaker rooms are as follows:

- 12<sup>th</sup> November, 2pm-6pm
- 13<sup>th</sup> November, 7am-5pm

If you are unable to upload your presentation during the above times please go to the room where your presentation will be held **1 hour prior to your scheduled presentation** and hand your presentation at the AV table located at the back of the hall.

Do arrive 10 minutes prior to your session and be seated on the seats allocated for you at the front of the hall, marked as "Next Speaker Seats". Please do strictly keep within your presentation time.



## Poster sessions

**Chairs:** Please arrive at the poster reception desk located in the **Jimbaran Lobby** and **Uluwatu Lobby** to sign in **15 minutes prior to the start of the session.** We would like to ask you kindly to stand in front of the first poster ready for the session to start.

Each poster presentation should last for 7 minutes and 3 minutes for Q&A. Please do keep within the schedule time.

Once the session has been completed do return to the poster reception desk to collect your Thank You Letter.

**Presenters:** Please take note of your poster's scheduled date, poster group and assigned #, as well as timing for poster pin-up, discussion and poster removal, to be observed on the presentation day.

## Important Note About Timing

Timing for Your Poster on the Presentation Day

08:00 – 12:00	Poster Pin-up*
13:00 – 15:00	Discussion**
15:15 – 17:15	Poster Removal***

\*Posters boards are located in the Jimbaran Lobby and Uluwatu Lobby. Your poster number will be indicated on the boards. To check your poster's Assigned #, please refer to the *Respirology* abstract book.

\*\*During the Discussion period, please be stationed at your poster ready to present to the chair.

\*\*\*Kindly remove your posters by 17:15 on the same day. Any posters that have not been collected will be moved to the secretariat room and discarded at 10:00 on Sun 16<sup>th</sup> November 2014. The APSR 2014 would not be held responsible for any lost or damaged posters.

# CONGRESS OVERVIEW

## Social Events

All attendees are cordially invited to participate in the following social events:

### **Opening Ceremony**

Date: Thursday 13<sup>th</sup> November 2014

Time: 16:30 - 17:15

Venue: BNDCC, Nusa Dua 4

### **Welcome Reception:**

Date: Thursday 13<sup>th</sup> November 2014

Time: 17:15 - 18:30

Venue: BNDCC, Tama Jepun

### **Closing Ceremony**

Date: Sunday 16<sup>th</sup> November 2014

Time: 12:00 – 12:30

Venue: BNDCC, Nusa Dua 4

### **Closing Lunch Reception**

Date: Sunday 16<sup>th</sup> November 2014

Time: 12:00 – 12:30

Venue: BNDCC, Singaraja

### **Gala Dinner**

For participation at the Gala Dinner, please register your attendance at the registration desk. Entrance to the Gala Dinner will require a coupon which will be provided at the registration desk upon badge collection.

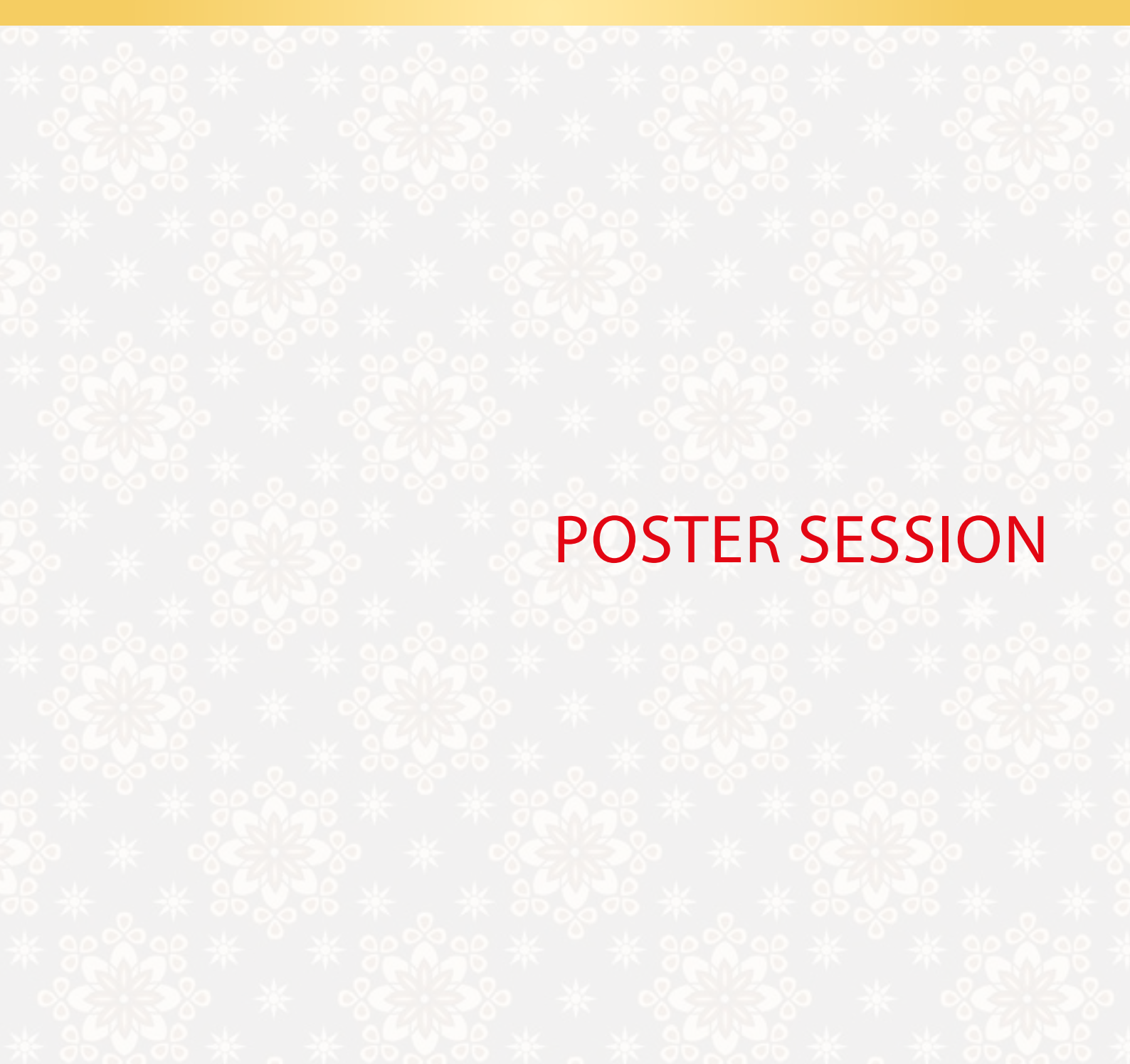
Please join us at the Gala Dinner to congratulate award winners and experience traditional Balinese performances and food.

Date: Sat 15<sup>th</sup> November 2014

Time: 18:30 – 21:30

Venue: BNDCC, Singaraja





# POSTER SESSION





# POSTER SESSION

## Poster Presentation Schedule

For poster presentations, please take note of your poster's scheduled date, poster group and assigned #, as well as timing for poster pin-up, discussion and poster removal, to be observed on the presentation day.

### Important Note About Timing

#### Timing for Your Poster on the Presentation Day

08:00 – 12:00	Poster Pin-up*
13:00 – 15:00	Discussion**
15:15 – 17:15	Poster Removal***

\*Posters boards are located in the Jimbaran Lobby and Uluwatu Lobby. Your poster number will be indicated on the boards. To check your poster's Assigned #, please refer to the *Respirology* abstract book.

\*\*During the Discussion period, please be stationed at your poster ready to present to the chair.

\*\*\*Kindly remove your posters by 17:15 on the same day. Any posters that have not been collected will be moved to the secretariat room and discarded at 10:00 on Sun 16<sup>th</sup> November 2014. The APSR 2014 would not be held responsible for any lost or damaged posters.

### Scheduled Date

14 Nov Poster Groups	Chairs	Assigned #
Shortlisted for Best Poster Judging - Please Pin-up at Best Posters Group	Jamal Zaini Triya Damayanti	P-K-006, P-K-008, P-K-084, P-N-010, P-N-039, P-Q-002, P-C-001, P-D-003, P-F-009, P-F-022  <i>*Please note you are all invited to the Gala Dinner which the Best Poster Award will be announced, please collect your invite card from the registration desk</i>

14 Nov Poster Groups	Chairs	Assigned #
Asthma 1	Puti Alexander K Ginting	P-A-001, P-A-002, P-A-003, P-A-004, P-A-005, P-A-006, P-A-007, P-A-008, P-A-009, P-A-010, P-A-011
Asthma 2	Erdal Karaoz Fahmi Alatas	P-A-012, P-A-013, P-A-014, P-A-016, P-A-017, P-A-018, P-A-019, P-A-020, P-A-021, P-A-022, P-A-023
Bronchoscopy and interventional 1	Wahju Aniwidyaningsih Anang Isnin	P-B-001, P-B-002, P-B-003, P-B-004, P-B-005, P-B-006, P-B-007, P-B-008, P-B-009
Clinical Allergy & Immunology 1	Woo Kyung Kim Triwahyu Astutik	P-D-002, P-D-004, P-D-005, P-D-006, P-D-007, P-D-008
Clinical Respiratory Medicine 1	Caroline Armas Pompini Agustina	P-E-001, P-E-002, P-E-003, P-E-004, P-E-005, P-E-006, P-E-007
COPD 1	Nihal Arzu Suradi	P-F-001, P-F-003, P-F-004, P-F-005, P-F-006, P-F-007, P-F-008, P-F-010, P-F-011, P-F-013
COPD 2	Takanobu Shioya Yani Jane Sugiri	P-F-014, P-F-015, P-F-016, P-F-017, P-F-018, P-F-019, P-F-020, P-F-021, P-F-023, P-F-024
COPD 3	Seung Jun Lee Amira Permatasari Tarigan	P-F-025, P-F-026, P-F-027, P-F-028, P-F-029, P-F-031, P-F-032, P-F-033, P-F-034, P-F-035

COPD 4	Woo Jin Kim Helmia Hassan	P-F-036, P-F-037, P-F-038, P-F-039, P-F-040, P-F-041, P-F-042, P-F-044, P-F-045, P-F-046
Critical Care Medicine 1	Hyun-Kyung Lee Prasenhadi	P-G-002, P-G-003, P-G-004, P-G-005, P-G-007, P-G-008, P-G-009, P-G-010, P-G-011, P-G-012
Environment & Occupational Health and Epidemiology 1	Jun Pyo Myong Agus Dwi Susanto	P-H-001, P-H-003, P-H-004, P-H-005, P-H-007, P-H-008, P-H-010, P-H-011, P-H-012
Interstitial Lung Disease 1	Onda Naomi Dianiati K Sutoyo	P-I-001, P-I-002, P-I-003, P-I-004, P-I-005, P-I-006, P-I-007, P-I-008, P-I-009, P-I-010, P-I-011
Lung Cancer 1	Wang Ke Antonius Sianturi	P-J-001, P-J-002, P-J-003, P-J-004, P-J-005, P-J-006, P-J-007, P-J-008, P-J-009, P-J-010, P-J-011
Lung Cancer 2	Laksmi Wulandari Nunuk Sri Muktiati	P-J-012, P-J-013, P-J-014, P-J-015, P-J-016, P-J-017, P-J-018, P-J-019, P-J-020, P-J-021, P-J-022
Lung Cancer 3	Yusuf Yilmaz Mulawarman Jayusman	P-J-023, P-J-024, P-J-025, P-J-026, P-J-027, P-J-028, P-J-029, P-J-030, P-J-031, P-J-032, P-J-034
Other 1	TBC Andika Putra	P-K-002, P-K-001, P-K-003, P-K-004, P-K-005, P-K-007, P-K-009, P-K-010, P-K-011, P-K-012, P-K-013
Other 2	MH Rahimirad Jamal Zaini	P-K-014, P-K-015, P-K-016, P-K-017, P-K-018, P-K-019, P-K-020, P-K-021, P-K-022, P-K-023
Other 3	Oea Khairsyaf Sardikin Giriputro	P-K-024, P-K-025, P-K-026, P-K-027, P-K-028, P-K-029, P-K-030, P-K-031, P-K-032, P-K-033
Other 4	Anarima Teguh Widjaja	P-K-034, P-K-035, P-K-036, P-K-037, P-K-038, P-K-039, P-K-040, P-K-041, P-K-042, P-K-043
Pulmonary Circulation 1	Menaldi Rasmin Prasenhadi	P-M-001, P-M-002, P-M-003, P-M-004, P-M-005, P-M-006, P-M-007, P-M-008
Respiratory Infections (Non- Tuberculosis) 1	Myung-Goo Lee Resti Y	P-N-001, P-N-003, P-N-005, P-N-006, P-N-007, P-N-008, P-N-009, P-N-011, P-N-012
Respiratory Infections (Non- Tuberculosis) 2	Priyanti Soepandi Reviono	P-N-013, P-N-014, P-N-015, P-N-016, P-N-017, P-N-018, P-N-020, P-N-021, P-N-022, P-N-023, P-N-024
Respiratory Infections (Non- Tuberculosis) 3	Rita Rogayah Yessy Sabri	P-N-025, P-N-026, P-N-027, P-N-028, P-N-029, P-N-030, P-N-031, P-N-032, P-N-033, P-N-034, P-N-035
Respiratory Neurobiology and Sleep 1	Oguz Kokturk Ratnawati	P-O-001, P-O-002, P-O-003, P-O-004, P-O-005, P-O-006, P-O-007, P-O-008, P-O-009
Respiratory Structure and Function 1	Triya Damayanti Retno Wihastuti	P-P-001, P-P-002, P-P-003, P-P-004, P-P-005, P-P-006, P-P-007, P-P-008
Tuberculosis 1	Adria Rusli Fathiyah	P-Q-001, P-Q-003, P-Q-004, P-Q-007, P-Q-008, P-Q-009, P-Q-010, P-Q-011, P-Q-012, P-Q-013
Tuberculosis 2	Diah Handayani IB Sila Wiweka	P-Q-015, P-Q-016, P-Q-017, P-Q-018, P-Q-019, P-Q-020, P-Q-021, P-Q-022, P-Q-024, P-Q-025

# POSTER SESSION

15 Nov Poster Groups	Chairs	Assigned #
Asthma 3	Kehnya Kohyama Suradi	P-A-024, P-A-025, P-A-026, P-A-027, P-A-028, P-A-029, P-A-031, P-A-032, P-A-033, P-A-034
Asthma 4	Kazuhiro Yatera Pradnaparamita	P-A-035, P-A-036, P-A-037, P-A-038, P-A-039, P-A-040, P-A-041, P-A-042, P-A-043, P-A-044, P-A-045
Bronchoscopy and interventional 2	Young Min-Li Yessy Sabri	P-B-010, P-B-011, P-B-012, P-B-013, P-B-014, P-B-015, P-B-016, P-B-017, P-B-018, P-B-019
Bronchoscopy and interventional 3	Sawang Saenghirunvattana Isnu Pradjoko	P-B-020, P-B-021, P-B-023, P-B-024, P-B-025, P-B-026 P-B-027, P-B-028, P-B-029
Clinical Respiratory Medicine 2	Kang Hong-Mo Wahyuningsih	P-E-008, P-E-009, P-E-010, P-E-011, P-E-012, P-E-013, P-E-014, P-E-015
COPD 5	Susanthy Djajalaksana Naoki Ijiri	P-F-047, P-F-048, P-F-049, P-F-050, P-F-051, P-F-052, P-F-053, P-F-054, P-F-055, P-F-056, P-F-057
COPD 6	Hiroyuki Ohbashi Sutji Mariono	P-F-058, P-F-059, P-F-060, P-F-061, P-F-062, P-F-063, P-F-064, P-F-065, P-F-066, P-F-067, P-F-068
COPD 7	Paul Leong Triwahyu Astutik	P-F-069, P-F-070, P-F-071, P-F-072, P-F-073, P-F-074, P-F-075, P-F-076, P-F-077, P-F-078
COPD 8	Deog Kyom Kim Frans Abednego Barus	P-F-079, P-F-080, P-F-081, P-F-082, P-F-083, P-F-084, P-F-085, P-F-086, P-F-087, P-F-088, P-F-089, P-F-090
Critical care Medicine 2	Jae Hwa Cho IB Sila Wiweka	P-G-013, P-G-014, P-G-015, P-G-016, P-G-017, P-G-018, P-G-019, P-G-020, P-G-021, P-G-022
Critical care Medicine 3	Hong Mo Kan Arif Hanafi	P-G-023, P-G-024, P-G-025, P-G-026, P-G-027, P-G-028, P-G-029, P-G-030, P-G-031, P-G-032
Environment & Occupational Health and Epidemiology 2	Winariani Erlang Samoedro	P-H-013, P-H-014, P-H-015, P-H-017, P-H-018, P-H-019, P-H-020, P-H-021, P-H-023
Interstitial Lung Disease 2	Dianiati K Sutoyo Helmia Hassan	P-I-012, P-I-013, P-I-014, P-I-015, P-I-016, P-I-017, P-I-018, P-I-019, P-I-020, P-I-021, P-I-022, P-I-023
Lung Cancer 4	Bulent Karagoz Elisna Syahrudin	P-J-035, P-J-036, P-J-037, P-J-038, P-J-039, P-J-040, P-J-041, P-J-042, P-J-043, P-J-044, P-J-045, P-J-046
Lung Cancer 5	Sezai Cubuk Noni Soeroso	P-J-047, P-J-048, P-J-049, P-J-050, P-J-051, P-J-052, P-J-053, P-J-054, P-J-055, P-J-056, P-J-057
Lung Cancer 6	Mohammad Al-Ghobain Andika Putra	P-J-058, P-J-059, P-J-060, P-J-061, P-J-062, P-J-063, P-J-064, P-J-065, P-J-066, P-J-067, P-J-068
Other 5	Anarima Alex Ginting	P-K-044, P-K-045, P-K-046, P-K-047, P-K-048, P-K-049, P-K-050, P-K-051, P-K-052, P-K-053
Other 6	Mulawarman Jayusman M. Tabri	P-K-054, P-K-055, P-K-056, P-K-057, P-K-058, P-K-059, P-K-060, P-K-061, P-K-062, P-K-063



Other 7	Nunuk Sri Muktiati Feni Fitriani	P-K-064, P-K-065, P-K-068, P-K-069, P-K-070, P-K-071, P-K-072, P-K-073, P-K-074
Other 8	Orhan Yucel TBC	P-K-075, P-K-076, P-K-077, P-K-078, P-K-079, P-K-080, P-K-081, P-K-082, P-K-083, P-K-085
Other 9	Turgut Isitmangil Bintang Sinaga	P-K-086, P-K-087, P-K-088, P-K-089, P-K-090, P-K-091, P-K-092, P-K-093, P-K-094
Pediatric Lung Disease 1	Luke Garratt Darmawan Budi Setyanto	P-L-001, P-L-002, P-L-003, P-L-004, P-L-005, P-L-006, P-L-007, P-L-008, P-L-009
Respiratory Infections (Non- Tuberculosis) 4	Fathiyah Isbaniah Masrul Basyar	P-N-036, P-N-037, P-N-038, P-N-040, P-N-041, P-N-042, P-N-043, P-N-044, P-N-045, P-N-046
Respiratory Infections (Non- Tuberculosis) 5	Yani Jane Sugiri Oea Khairsyaf	P-N-047, P-N-048, P-N-049, P-N-050, P-N-051, P-N-052, P-N-053, P-N-054, P-N-055, P-N-056
Respiratory Neurobiology and Sleep 2	Hiroyuki Yoshimine Budhi Antariksa	P-O-010, P-O-011, P-O-012, P-O-013, P-O-016, P-O-017, P-O-018, P-O-019
Tuberculosis 3	TBC Ida Bagus Suta	P-Q-026, P-Q-027, P-Q-028, P-Q-029, P-Q-030, P-Q-031, P-Q-032, P-Q-033, P-Q-034, P-Q-035
Tuberculosis 4	Jae-Woo Jung Ratnawati	P-Q-036, P-Q-037, P-Q-038, P-Q-039, P-Q-040, P-Q-041, P-Q-042, P-Q-043, P-Q-044, P-Q-045
Tuberculosis 5	Erlina Burhan Pompini Agustina	P-Q-046, P-Q-047, P-Q-048, P-Q-049, P-Q-050, P-Q-051, P-Q-052, P-Q-053, P-Q-054, P-Q-055, P-Q-056



## DAY 1 THURSDAY, 13<sup>TH</sup> NOVEMBER

	Nusa Dua 4	Nusa Dua 3	Nusa Dua 1	Nusa Dua 2	Uluwatu 1	Uluwatu 2	Uluwatu 3	Uluwatu 5	Uluwatu 7	Uluwatu 6
8:00 - 9:00 am	<b>Registration</b>									
<b>Morning</b> (9:00 - 12:00)		Postgraduate Course in Lung Cancer (an ESAP Seminar): Updates in Lung Cancer	Workshop on Bronchoscopy: Peripheral Lung Nodules and Interventional Bronchoscopy for Asthma	Workshop on Bronchoscopy: Managing Airway Stenosis and Corpus Alienum  Sponsored by Olympus	Workshop on Pleural Disease and Thoracoscopy: Updates on Pleural Disease and Thoracoscopy	Postgraduate Course: Respiratory Neurobiology and Sleep	Workshop on Lung Mechanical Ventilation	Postgraduate Course: MDR Tuberculosis	Workshop on Basic and Advances of Lung Function: Patient-oriented PFTs to assist clinical decision-making	Postgraduate Course on COPD: Pathogenesis and Disease Phenotype of COPD
<b>Afternoon</b> (13:00 - 16:00)								Postgraduate Course: Pneumonia Lung Infection		
<b>Evening</b> (16:30 - 19:00)	<b>Opening Ceremony</b>									
	<b>Welcome Reception</b>									

Workshop

Postgraduate Course

## DAY 2 FRIDAY, 14<sup>TH</sup> NOVEMBER

	Nusa Dua 2	Nusa Dua 3	Nusa Dua 4	Uluwatu 1	Uluwatu 2	Uluwatu 3	Uluwatu 5	Uluwatu 6	Uluwatu 7	Jimbaran Lobby	Uluwatu Lobby	Nusa Dua 5
<b>Morning</b> (8:00 - 9:30)	CS3: Respiratory Neurobiology and Sleep  OSA - The Burden of Disease	CS2: Lung Cancer  Mediastinal Tumor and Mesothelioma	CS1: Asthma  Small Airways Diseases in Asthma and Severe Asthma	CS4: COPD  Current Issues in COPD in Asia Pacific	OS2: Pulmonary Circulation	OS3: Respiratory Infections (Nontuberculosis)	CS5: Respiratory Structure and Function  Lung Function and Various Disease		OS1: Interstitial Lung Disease			
<b>Pre-lunch</b> (9:45 - 10:45)	Zambon Pre-lunch Symposia	Mundipharma Pte Ltd Pre-lunch Symposia	Pfizer Anti-infectives Pre-lunch Symposia							<b>Poster Pin-up and Viewing</b>		
<b>Lunch</b> (11:00 - 12:00)	Boston Scientific Asia Pacific Lunch Symposia	Daiichi Sankyo Lunch Symposia	Boehringer Ingelheim Lunch Symposia									
<b>Afternoon</b> (13:00 - 17:00)	AS3: Respiratory Neurobiology and Sleep  Advances in OSA Pathophysiology, Comorbidity and Treatment	AS2: Lung Cancer  Targeted Strategies and Beyond in NSCLC	AS1: Clinical Allergy & Immunology  Current Topics on Asthma and COPD Pathogenesis	AS4: Respiratory Structure and Function  Phenotyping of Lung Disease	OS5: Asthma  OS7: Bronchoscopy and Interventional Techniques	OS6: COPD  OS8: COPD	AS5: Environmental & Occupational Health and Epidemiology  Trends in Occupational Lung Diseases in Developing and Developed Nations	OS4: Respiratory Neurobiology and Sleep	CS6: The evolving role of real-life research in respiratory medicine (Respiratory Effectiveness Group)	<b>Poster Discussion</b>		
	AS8: Tuberculosis  Achieving Zero TB: Make it happen	AS7: Cell and Molecular Biology  Hot Topics in Respiratory Cellular and Molecular Biology	AS6: Asthma  Asthma-COPD Overlap Syndrome	AS9: Pediatric Lung Disease  OSAS in Children	OS9: Lung Cancer  OS10: Clinical Respiratory Medicine	OS11: Tuberculosis	CS7: Environmental & Occupational Health and Epidemiology  Nature and Respiratory Management	AS10: Clinical Respiratory Medicine  Interesting clinical cases from Hong Kong	<b>Poster Viewing and Removal</b>			

Congress Symposia

Assembly Symposia

Industry-Sponsored

Oral Sessions



## DAY 3 SATURDAY, 15<sup>TH</sup> NOVEMBER

	Nusa Dua 2	Nusa Dua 3	Nusa Dua 4	Uluwatu 1	Uluwatu 2	Uluwatu 3	Uluwatu 5	Uluwatu 7	Jimbaran Lobby	Uluwatu Lobby	Nusa Dua 5
<b>Morning</b> (8:30 - 10:00)	AS12: Bronchoscopy and Interventional Techniques Novel Bronchoscopy and Interventional Technique	CS8: Interstitial Lung Disease Comorbidity in IPF	AS11: COPD Redefining COPD	Best Oral Presentation Selection Session	OS12: Asthma	OS13: Tuberculosis	CS9: Lung Cancer Lung Cancer in Asia Pacific	AS13: Clinical Respiratory Medicine Interesting Clinical Cases			
					CS10: Medical Ethics and Patient Safety	OS14: Tuberculosis					
				Presidential Lecture							
<b>Pre-lunch</b> (10:45 - 11:45)	Boehringer Ingelheim Pre-lunch Symposia	Covidien Pvt Ltd Pre-lunch Symposia	Takeda Pharmaceuticals International GmbH Pre-Lunch Medical Forum						<b>Poster Pin-up and Viewing</b>		
<b>Lunch</b> (12:00 - 13:00)	Chiesi Lunch Symposia	Astra Zeneca Lunch Symposia	Pfizer Vaccine Lunch Symposia								
			Memorial Lecture 1 Ann Janet Woolcock Research Award  Memorial Lecture 2 Michiyoshi Harasawa Research Award								
<b>Afternoon</b> (13:00 - 18:00)	AS16: Pulmonary Circulation What's New in Pulmonary Hypertension?	AS15: Interstitial Lung Disease Interstitial Lung Disease Diagnosis and Treatment	AS14: Respiratory Infections (Non-tuberculosis) Community-Acquired Pneumonia in Asia Pacific	CS11: Bronchoscopy and Interventional Techniques Updates in Pleural Disease	OS15: Lung Cancer	OS16: COPD	AS17: Clinical Respiratory Medicine Smoking cessation	Young Investigator Awardees	<b>Poster Discussion</b>		
					OS17: Cell and Molecular Biology	OS18: Critical Care Medicine					
	CS12: Tuberculosis TB Risk Factors and Intervention	AS19: Interstitial Lung Disease IPF: The Next Target	AS18: COPD COPD: Progress on the Management	CS13: Lung Infection (Non-tuberculosis) Difficult Respiratory Infections	OS19: Asthma	OS20: Environmental & Occupational Health and Epidemiology	AS20: Lung Cancer What's New in Small Cell Lung Cancer and Neuroendocrine Tumor of the Lung	Young Investigator Awardees			

Congress Symposia

Assembly Symposia

Industry-Sponsored

Oral Sessions

## DAY 4 SUNDAY, 16<sup>TH</sup> NOVEMBER

	Nusa Dua 2	Nusa Dua 3	Nusa Dua 4	Uluwatu 1	Uluwatu 5	Uluwatu 7	Nusa Dua 5
<b>Morning</b> (8:00 - 12:00)	Respiratory Organization Workshop	AS22: Critical Care Medicine Hot Issues in ARDS	AS21: Tuberculosis ISTC to Ensure Quality of Care	OS21: Lung Cancer	OS22: Others	OS23: Respiratory Infections (Non-Tuberculosis)	<b>Exhibition Area</b>
						OS24: Respiratory Structure and Function	
		AS24: Asthma What is in the Pipelines in Asthma Therapy?	AS23: Respiratory Infections (Non-tuberculosis) Viral Pneumonia	OS26: Interstitial Lung Disease	OS27: Critical Care Medicine		
<b>Lunch</b>			Closing Ceremony	OS28: Clinical Allergy and Immunology			

Congress Symposia

Assembly Symposia

Oral Sessions



# DETAILED PROGRAM

DAY 1 - THURSDAY, 13<sup>TH</sup> NOVEMBER

DAY 2 - FRIDAY, 14<sup>TH</sup> NOVEMBER

DAY 3 - SATURDAY, 15<sup>TH</sup> NOVEMBER

DAY 4 - SUNDAY, 16<sup>TH</sup> NOVEMBER



# DAY 1 THURSDAY, 13<sup>TH</sup> NOVEMBER

**Room:** Nusa Dua 1

**Session:** Workshop Bronchoscopy: Peripheral Lung Nodules and Interventional Bronchoscopy for Asthma

**Session Chairs:** Isnu Pradjoko, Nirwan Arief

**Sponsored by:** Olympus

**Target Audience:** Pulmonologists and respiratory residents

9:00 - 9:20	Course Introduction, Objectives and Anatomy for Bronchoscopy	Dicky Soehardiman
9:20 - 9:40	CT Assessment for Pulmonary Nodules	Kazuma Kishi
9:40 - 10:00	Principles of EBUS	Noriaki Kurimoto
10:00 - 10:20	EBUS in Staging Mediastinal Nodes	Noriaki Kurimoto
10:30 - 11:00	BREAK	
11:00 - 11:20	EBUS for TBLB	Teruomi Miyazawa
11:20 - 11:40	Lung Mapping with CT and Biopsy for Peripheral Lung Lesion	Takashi Ishida
11:40 - 12:00	Interventional Bronchoscopy for Asthma Management: Bronchial Thermoplasty	Pyng Lee
12:00 - 13:00	LUNCH	
	Hands-on Workshops	
13:00 - 15:00	1. Bronchoscopy + Lung Mapping	Takashi Ishida
	2. EBUS - TBNA	Noriaki Kurimoto
	3. EBUS TBLB	Pyng Lee



**Room:** Nusa Dua 2

**Session:** Workshop Bronchoscopy: Managing Airway Stenosis and Corpus Alienum

**Session Chairs:** Boedi Swidarmoko, Oea Khairsyaf

**Sponsored by:** FujiFilm

**Target Audience:** Pulmonologists and respiratory residents

9:00 - 9:15	Course Introduction	Boedi Swidarmoko, Oea Khairsyaf
9:15 - 9:30	Rigid Bronchoscopy: Its Role in Recent Airway Stenosis and Malacia	TBC
9:30 - 9:45	Airway Stenting	TBC
9:45 - 10:00	Useful Techniques in Airway Stenosis	Teruomi Miyazawa
10:00 - 10:15	CT Mapping for EBUS	Takashi Ishida
10:15 - 10:30	What To Do for Corpus Alienum	Boedi Swidarmoko
10:30 - 11:00	BREAK	
11:00 - 11:15	EBUS in Airway Stenosis	Noriaki Kurimoto
11:15 - 11:30	Endobronchial Tuberculosis	Wahju Aniwidyaningsih
11:30 - 11:45	EBUS TBNA for Diagnosing Lung Cancer	Masahide Oki
12:00 - 13:00	LUNCH	
	Hands-on Workshops	
	1. Bronchoscopy & Corpus alienum	Boedi Swidarmoko
13:00 - 15:40	2. EBUS TBNA	Masahide Oki/Dicky Soehardiman
	3. Rigid Bronchoscopy and Airway Stenting	TBC

# DAY 1 THURSDAY, 13<sup>TH</sup> NOVEMBER

**Room:** Nusa Dua 3

**Session:** Postgraduate Course in Lung Cancer (an ESAP Seminar): Updates in Lung Cancer

**Session Chairs:** Kwun Fong, Achmad Hudoyo

**Target Audience:** Pulmonologists and respiratory physicians, healthcare professionals and researchers

9:00 - 9:10	Course introduction	Kwun Fong
9:10 - 9:35	Molecular Diagnosis of Lung Cancer	Eiso Hiyama
9:35 - 10:00	Lung Cancer Stem Cell	Toshiaki Kikuchi
10:00 - 10:25	Thoracic CT pattern in Lung Cancer: Correlation of CT and Pathological Diagnosis	Kazuma Kishi
10:30 - 11:00	BREAK	
11:00 - 11:25	Genetic Heterogeneity in Lung Cancer	Koichi Hagiwara
11:25 - 11:50	The TNM Staging System for Lung Cancer	Kwun Fong
12:00 - 13:00	LUNCH	
13:00 - 13:10	Welcome Back from Lunch and Summarize Morning	Kwun Fong
13:10 - 13:35	Targeted Agent in Lung Cancer: EGFR TKI and Beyond	David C Lam
13:35 - 14:00	Lung Tumour Immunology	Bruce Robinson
14:00 - 14:25	Neuroendocrine Tumors of The Lung	Chong-Kin Liam
14:30 - 15:00	BREAK	
15:00 - 15:25	SCLC: What We Have Now	Chunxue Bai
15:25 - 15:50	Lung Cancer: The Future Ahead	Koichi Hagiwara
15:50 - 16:00	Closing	Kwun Fong

**Room:** Uluwatu 1

**Session:** Workshop on Pleural Disease and Thoracoscopy: Updates on Pleural Disease and Thoracoscopy

**Session Chairs:** Gary Lee, Menaldi Rasmin

**Target Audience:** Pulmonologists and respiratory residents

	Course Introduction	Menaldi Rasmin
9:00 - 9:15	Pleural Disease: What Should We Know?	Richard Light
9:15 - 9:30	Thoracic Ultrasound: Guidance to Thoracic Procedures	Gary Lee
9:30 - 9:45	Closed Pleural Biopsy: Does It Still Have Value?	Menaldi Rasmin
9:45 - 10:00	Percutaneous Pleural Biopsy: Fine Needle or Core Biopsy	Menaldi Rasmin
10:00 - 10:15	Diagnostic and Therapeutic Thoracoscopy	Pyng Lee
10:15 - 10:30	Flex-rigid and Rigid Thoracoscopy: When to Choose and the Drawback	Pyng Lee
10:30 - 11:00	BREAK	
11:00 - 11:15	Indwelling Pleural Catheter in MPE	Gary Lee
11:15 - 11:30	New Therapy of Pleural Infection (tPA and DNase)	Gary Lee
11:30 - 11:45	Case Discussion	Wahju Aniwidyaningsih
12:00 - 13:00	LUNCH	
13:00 - 15:40	Hands-on Workshops	
	1. Indwelling Pleural Catheter & Percutaneous Pleural Biopsy	Gary Lee
	2. Rigid Pleuroscopy	Menaldi Rasmin
	3. Flex-rigid Pleuroscopy	Wahju Aniwidyaningsih



# DAY 1 THURSDAY, 13<sup>TH</sup> NOVEMBER

**Room:** Uluwatu 2

**Session:** Postgraduate Course on Respiratory Neurobiology and Sleep: Respiratory Neurobiology and Sleep

**Session Chairs:** Mary S-M Ip, Kazuo Chin

**Target Audience:** Pulmonologists, sleep medicine, cardiologists, all physicians, trainee, nurses, respiratory therapists, sleep technologists, medical students, allied health professionals

9:00 - 9:05	Course Introduction	Kazuo Chin
9:05 - 9:30	Overview: Sleep-Related Breathing Disorders and Their Treatment	Kazuo Chin
9:30 - 10:00	Sleep Apnea in Children: Diagnosis and Management	Carole L Marcus
10:00 - 10:30	Clinical Characteristics Including Cephalogram in SDB	Mary S-M Ip
10:30 - 11:00	BREAK	
11:00 - 11:30	Methods of Sleep Testing: Polysomnography and Portable Monitoring	Michelle Cheong
11:30 - 12:00	OSA and Pathophysiology Including Sustained and Intermittent Hypoxia	Geraldo Lorenzi-Filho
12:00 - 13:00	LUNCH	
13:00 - 13:30	OSA and Cardiovascular, Metabolic Complications and the Effects of Treatment	Himender Makker
13:30 - 14:00	Sleep Apnea and Heart Failure	Takatoshi Kasai
14:00 - 14:30	Sleep-related Breathing Disorders in Indonesia	Agus D Susanto
14:30 - 15:00	Maintaining Compliance in the Treatment of OSA	Geraldo Lorenzi-Filho
15:00 - 15:30	Discussion	

**Room:** Uluwatu 3

**Session:** Workshop on Lung Mechanical Ventilation: Lung Mechanical Ventilation

**Session Chair/s:** Gerald Chua, Prasenohadi Pradono

**Target Audience:** Pulmonologists, anesthesiologists, intensivists, trainees, fellows, and those interested in critical care medicine

9:00 - 9:10	Course Introduction	Prasenohadi Pradono
9:10 - 9:40	Overview of Mechanical Ventilation: Modes of Mechanical Ventilation	Philia Setiawan
9:40 - 10:10	Monitoring of Mechanical Ventilation	Albert Oluwasegun
10:30 - 11:00	Mechanical Ventilation Approach in ALI and ARDS	Chian Min Loo
11:00 - 11:30	Mechanical Ventilation Approach in Viral Infection-Related ALI and ARDS	Sang-Bum Hong
11:30 - 12:00	Auto PEEP: Mechanical Ventilation in COPD and Chronic Lung Disease	Chua Gerald
12:00 - 13:00	LUNCH	
13:00 - 13:30	Weaning: How to Overcome Difficulty in Weaning	Gerald Chua
13:30 - 14:00	Noninvasive Ventilation for Chronic Lung Disease	Nicholas Hill
14:00 - 14:30	Case Presentation	Prasenohadi Pradono
14:40 - 15:00	BREAK	
15:00 - 15:45	Dry Workshop	Albert Oluwasegun, Prasenohadi Pradono

# DAY 1 THURSDAY, 13<sup>TH</sup> NOVEMBER

**Room:** Uluwatu 5

**Session:** Postgraduate Course on MDR TB: MDR Tuberculosis

**Session Chairs:** Giovanni Battista Migliori, Erlina Burhan

**Target Audience:** Pulmonologists, microbiologists, all physicians, trainees, nurses, fellows and trainees, medical students, allied health professionals

9:00	Course Introduction	Giovanni Battista Magliori/Erlina Burhan
9:00 - 9:15	Challenge of Drug Resistance TB in Asia and Western Pacific	Chiang Chen-Yuan
9:15 - 9:30	Implementing ISTC to Prevent MDR	Philip Hopewell
9:30 - 9:45	How MDR Developed	Francis Drobniewski
10:00 - 10:15	Treatment of MDR and XDR TB	Giovanni Battista Migliori
10:15 - 10:30	Second Line Anti-TB Drugs: Mechanism of Action and Rational for Use	Chiang Chen-Yuan
10:30 - 11:00	BREAK	
11:00 - 11:15	TB MDR Among HIV Patients	Charles Yu
11:15 - 11:30	Adverse Event of Second-line Drug	Erlina Burhan
11:30 - 12:00	Countries' Experiences: Indonesia, Philippines, Thailand, Hongkong, Taiwan	Charles Yu, Reviono
12:00	Closing	Session Chairs



**Room:** Uluwatu 5

**Session:** Postgraduate Course on Pneumonia: Pneumonia

**Session Chairs:** Jun-Hee Woo, Priyanti Soepandi

**Target Audience:** Pulmonologists, microbiologists, all physicians, fellows and trainees, nurses, medical students, allied health professionals

13:00 - 13:25	Burden of CAP in the Asia Pacific Region	Jun-Hee Woo
13:25 - 13:50	CAP in TB Endemic Countries	VK Vijayan
13:50 - 14:15	Treatment Approach in CAP, HAP/HCAP	Jun-Hee Woo
14:15 - 14:40	Pulmonary Mycosis: What We Have Learned	Jennifer Mendoza
14:40 - 15:00	BREAK	
15:00 - 15:25	Hajj Pilgrim and Viral Pneumonia	Abdullah Alshimemeri
15:25 - 15:50	Viral Pneumonia: Diagnosis and Management	Jasper Chan
15:50 - 16:00	Closing	Session Chairs

# DAY 1 THURSDAY, 13<sup>TH</sup> NOVEMBER

**Room:** Uluwatu 6

**Session:** Postgraduate Course on COPD: Pathogenesis of COPD and Disease  
Phenotype of COPD

**Session Chairs:** Sang-Do Lee, Muhammad Amin

**Target Audience:** Pulmonologists, all physicians, trainee, medical students, and allied health professionals

9:00 - 9:30	Pathogenesis of COPD	Tomoko Betsuyaku
9:30 - 10:00	Phenotyping of COPD from Asian Perspective	Sang-Do Lee
10:00 - 10:30	Asthma COPD Overlap Syndrome	Peter Gibson
10:30 - 11:00	BREAK	
11:00 - 11:30	GOLD Documents: Current Issue?	Jorgen Vestbo
11:30 - 12:00	Assessment of Exacerbation	Paul Jones

**Room:** Uluwatu 7

**Session:** Workshop on Lung Function: Patient-oriented PFTs to Assist Clinical Decision-making

**Session Chairs:** Paul Enright, Ratnawati

**Target Audience:** Physicians, respiratory therapists, trainees, fellows, registered nurses, advance practice nurses and other interested health care providers

9:00 - 9:20	Course Introduction, Anatomy of the Lung and Basic Lung Function	Triya Damayanti
9:20 - 9:40	Clinical Application of Pulmonary Testing	Yasutaka Nakano
9:40 - 10:00	Evaluation of Lung Function in Chronic Disease	Le Thi Tuyet Lan
10:00 - 10:20	Case Presentation on Adult Smokers	Paul Enright
10:30 - 11:00	BREAK	
11:00 - 11:20	Hands-on Pocket Spirometry and Diagnostic Spirometry	Paul Enright
11:20 - 11:40	Case Presentation on Patient with Episodic Wheezing	Paul Enright
11:40 - 12:00	Hands-on Spirometry, PEF Meter and eNO Testing	Paul Enright
12:00 - 13:00	LUNCH	
13:00 - 13:20	Case Presentation of Slow Onset Dyspnea	Paul Enright
13:20 - 13:40	DLCO Demonstration	Paul Enright
13:40 - 14:00	Bodyplethysmograph: Principles and Technique	Paul Enright
14:00 - 14:20	Changes in Lung Volume in Various Disease	Paul Enright
14:20 - 14:40	Case Presentation - Interactive Discussion	Paul Enright
14:40 - 15:00	BREAK	
15:00 - 15:45	Evaluation	Triya Damayanti, Paul Enright



# DAY 2

## FRIDAY, 14<sup>TH</sup> NOVEMBER

### SESSION TIME: 08:00 - 09:30

**Room:** Nusa Dua 2

**Session:** Congress Symposia 3: OSA - The Burden of Disease

**Session Chairs:** Budhi Antariksa, Mary Ip

**Target Audience:** Pulmonologists and sleep physicians and dentists, clinicians, researchers, registered nurses, respiratory therapists, GPs, fellows and residents in training interested in sleep apnea

8:00 - 8:30	OSA in Asia Pacific: Next Burden Disease?	Koji Narui
8:30 - 9:00	OSA: Cardiovascular and Metabolic Complications: Next Threat?	Himender Makker
9:00 - 9:30	OSA: Organisation of Care to Reduce Disease Burden	Atul Malhotra

**Room:** Nusa Dua 3

**Session:** Congress Symposia 2: Mediastinal Tumor and Mesothelioma

**Session Chairs:** Achmad Hudoyo, Achmad Mulawarman

**Target Audience:** Pulmonologists, oncologists, radiologists, thoracic surgeons, radiotherapy specialists, clinicians, researchers, registered nurses, respiratory therapists, GPs, fellows and residents in training interested in thoracic malignancy

8:00 - 8:30	Staging System and Treatment of Thymoma	Achmad Hudoyo
8:30 - 9:00	Mesothelioma and Malignant Pleural Effusion	Gary Lee
9:00 - 9:30	Discussions	Achmad Hudoyo, Gary Lee

**Room:** Nusa Dua 4

**Session:** Congress Symposia 1: Small Airways Diseases in Asthma and Severe Asthma

**Session Chairs:** Greg King, Susanty Djajalaksana

**Target Audience:** Pulmonologists, clinicians, researchers, registered nurses, respiratory therapists, GPs, fellows and residents in training, medical students

8:00 - 8:30	Small Airways in Asthma: Pathogenesis	Masakazu Ichinose
8:30 - 9:00	Clinical Phenotype of Severe Asthma	Greg King
9:00 - 9:30	Does the Treatment of the Small Airways Matter?	Omar Usmani

**Room:** Uluwatu 1

**Session:** Congress Symposia 4: Current Issues of COPD in Asia Pacific

**Session Chairs:** Muhammad Isa, Sang-Do Lee

**Target Audience:** Pulmonologists, clinicians, researchers, registered nurses, respiratory therapists, GPs, fellows and residents in training, medical students

8:00 - 8:20	The Role Biomass in COPD	Faisal Yunus
8:20 - 8:40	Phenotyping of COPD from Asian Population	Sang-Do Lee
8:40 - 9:00	Air Pollution and Burden of COPD: Next Threat?	D. Behera
9:00 - 9:20	Community-based COPD Management	Ngo Quy Chau
9:20 - 9:30	Discussion	

**Room:** Uluwatu 2

**Session:** Oral Session 2: Pulmonary Circulation

**Session Chairs:** Mohmmadhossein Rahimirad, Hiroshi Kimura

8:00 - 8:10	Reference Values for Normal Pulmonary Artery Diameter, Ratio of Pulmonary Artery to Aorta by CT Scan in Korean Population (O-M-001)	Sang Hoon Lee
8:10 - 8:20	Comparison serum CA-125 Level in Patients with Chronic Obstructive Pulmonary Disease with and Without Pulmonary Hypertension (O-M-002)	Mohmmadhossein Rahimirad
8:20 - 8:30	Effect of Thrombomodulin Alpha for Disseminated Intravascular Coagulation Treatment in Patients with Lung Cancer (O-M-003)	Kentaro Nakano
8:30 - 8:40	Clinical Conundrum in Pulmonary Embolism Diagnosis: Management of Imaging Discordance (Negative CT Pulmonary Angiography and High probability Ventilation-Perfusion Scan); A case series (O-M-004)	Vicky Chang
8:40 - 8:50	The Correlation Between CD4 Level and Left Ventricular Systolic Function in HIV/AIDS Patients (O-M-005)	Amir Muzakkir

# DAY 2

## FRIDAY, 14<sup>TH</sup> NOVEMBER

### SESSION TIME: 08:00 - 09:30

**Room:** Uluwatu 3

**Session:** Oral Session 3: Respiratory Infections (Non-tuberculosis)

**Session Chairs:** Jennifer Mendoza, Alex K Ginting

8:00 - 8:10	The Pathophysiological Comparison of Secondary Pneumococcal Pneumonia After H1N1 Pandemic 2009 or H1N1 New Caledonia Influenza (O-N-002)	Masafumi Seki
8:10 - 8:20	Investigation of the Antibacterial Mechanism of Eugenol and Cinnamaldehyde on <i>Legionella pneumophila</i> (O-N-003)	Jiangwei Ma
8:20 - 8:30	Long-term Macrolide Antibiotic Therapy May Prevent the Development of Pneumonia in the Elderly (O-N-004)	Hiroki Yoshikawa
8:30 - 8:40	Are There Any Differences on Clinical Manifestations of Community Acquired Pneumonia According to Presence of Non-Tuberculous Mycobacterium? (O-N-007)	Byoung-Hoon Lee
8:40 - 8:50	Incremental Prognostic Predict Ability of Chest Computed Tomography in Patients with Community Onset Pneumonia (O-N-009)	Nemoto Masahiro
8:50 - 9:00	Common Presentation of Uncommon Infection: A case of Pulmonary Actinomycosis (O-N-010)	Dushantha Madegedara
9:00 - 9:10	Prognostic Factors of Mortality in Patients with Drug-resistant <i>Acinetobacter baumannii</i> Ventilator- Associated Pneumonia (O-N-011)	Atikun Limsukon



**Room:** Uluwatu 5

**Session:** Congress Symposia 5: Lung Function and Various Diseases

**Session Chairs:** Kenneth Tsang, Muhammad Amin

**Target Audience:** Pulmonologists, clinicians, researchers, registered nurses, respiratory therapists, GPs, fellows and residents in training, medical students

8:00 - 8:30	Role of Spirometric Full Flow-volume Curve in Searching for Causes of Dyspnea	Le Thi Tuyet Lan
8:30 - 9:00	Aspiration Pneumonia	Kenneth Tsang
9:00 - 9:30	Lung Function in Various Disease	Paul Enright

**Room:** Uluwatu 7

**Session:** Oral Session 1: Interstitial Lung Disease

**Session Chairs:** Masahito Ebina, Dianiati

8:00 - 8:10	Efficacy and Safety of Nintedanib in Patients with Idiopathic Pulmonary Fibrosis: Results of Two 52-Week, Phase III, Randomized, Placebo-Controlled Trials (INPULSIS™) (O-I-001)	Dong Soon Kim
8:10 - 8:20	Clinical Characteristics of Secondary Pulmonary Alveolar Proteinosis (sPAP): The Reason for the Difficulty of Diagnosis as sPAP (JSPS KAKENHI Grant Number 26305028) (O-I-002)	Haruyuki Ishii
8:20 - 8:30	Long-term Follow-Up of Serum Autoantibody Against GM-CSF Levels in Patients with Autoimmune Pulmonary Alveolar Proteinosis (O-I-003)	Akiko Matsumuro
8:30 - 8:40	A Pre-Clinical Study for Development of a New GM-CSF Inhalation Drug as a Treatment of Pulmonary Alveolar Proteinosis (O-I-005)	Ryushi Tazawa
8:40 - 8:50	Transbronchial Lung Biopsy for the Diagnosis of Lymphangioleiomyomatosis (O-I-006)	Taro Koba
8:50 - 9:00	Subgroup Analysis of Asian patients in the INPULSIS™ Trials of Nintedanib in Idiopathic Pulmonary Fibrosis (O-I-007)	Hiroyuki Taniguchi
9:00 - 9:10	Cytokine Profile of Lung Tissues and Serum in Patients with Interstitial Lung Diseases (O-I-008)	Xiaohong Chen
9:10 - 9:20	Predictive Factors for the Effect of Pirfenidone in Idiopathic Pulmonary Fibrosis (O-I-009)	Yasunori Ichimura
9:20 - 9:30	Experimental Study of Hypersensitivity Pneumonitis in Guinea Pig Induced by Inhalation of Hair Dye Ingredients (O-I-010)	Jieyu Luo

# DAY 2

## FRIDAY, 14<sup>TH</sup> NOVEMBER

### SESSION TIME: 09:45 - 10:45

**Room:** Nusa Dua 2

**Session:** Pre-Luncheon Symposia 3: Looking Beyond FEV1: New Mechanisms and Clinical Outcomes with NAC at High Doses in COPD

**Session Chairs:** Wiwien Heru Wiyono

**Sponsored by:** Zambon

09.45 - 10.05	Twice Daily N-Acetylcysteine 600 mg for Exacerbations of Chronic Obstructive Pulmonary Disease (PANTHEON): A Randomized, Double-Blind Placebo-Controlled Trial	Jin Ping-Zheng
10.05 - 10.25	High Dose N-Acetylcysteine in COPD: Focus on Small Airways Function	Fu Qiang-Wen
10.25 - 10.45	Discussion	

**Room:** Nusa Dua 3

**Session:** Pre-Luncheon Symposia 2: Beyond Asthma Control

**Session Chairs:** Sang-Heon Cho

**Sponsored by:** Mundipharma Pte Ltd

9.45 - 10.15	Optimizing Proven Therapies to Achieve Asthma Control	TBC
10.15 - 10.45	Factors beyond Asthma Control in the Real World	David Price

**Room:** Nusa Dua 4

**Session:** Pre-Luncheon Symposia 1: Optimizing Management of Respiratory Tract Infections: Are Bugs in Asia Getting Smarter?

**Session Chairs:** Faisal Yunus

**Sponsored by:** Pfizer Anti-infectives

9:45 - 9.50	Chairman Introduction	Faisal Yunus
9.50 - 10.05	Changing Trends in Bacterial Resistance in Asia: Truth or Myth?	Camilla Rodrigues
10.05 - 10.35	Respiratory Tract Infections: From Community to the Hospital. Has Anything Changed?	Thomas M. File
10.35 - 10.40	Q&A	
10.40 - 10.45	Closing Remarks	Faisal Yunus

# DAY 2

FRIDAY, 14<sup>TH</sup> NOVEMBER  
SESSION TIME: 11:00 - 12:00

**Room:** Nusa Dua 2

**Session:** Luncheon Symposia 3

**Session Chair:** TBC

**Sponsored by:** Boston Scientific Asia Pacific

11.00 - 12:00	Bronchial Thermoplasty for the Management of Asthma	Michael E. Wechsler
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**Room:** Nusa Dua 3

**Session:** Luncheon Symposia 2

**Session Chair:** Arifin Nawas

**Sponsored by:** Daiichi Sankyo

11.00 - 11.25	Optimal strategy treatment for Community acquired Pneumonia	Lionel A. Mandell
11.25 - 11.50	Practice Guideline on Community Acquired Pneumonia in Indonesia	Priyanti Soepandi
11.50 - 12.00	Discussion	All

**Room:** Nusa Dua 4

**Session:** Luncheon Symposia 1

**Session Chair:** Faisal Yunus

**Sponsored by:** Boehringer Ingelheim

11.00 - 12:00	Optimizing Bronchodilation and Improving Outcomes in COPD	Antonio Anzueto
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# DAY 2

## FRIDAY, 14<sup>TH</sup> NOVEMBER

### SESSION TIME: 13:00 - 15:00

**Room:** Nusa Dua 2

**Session:** AS3, Assembly of Respiratory Neurobiology and Sleep: Advances in OSA Pathophysiology, Comorbidity and Treatment

**Session Chairs:** Geraldo Lorenzi-Filho, Kazuo Chin

**Target Audience:** Pulmonologists and sleep physicians and dentists, clinicians, researchers, registered nurses, respiratory therapists, GPs, fellows and residents in training interested in sleep apnea

13:00 - 13:30	Recent Advances in Cardiovascular Comorbidity in OSA	Geraldo Lorenzi-Filho
13:30 - 14:00	Metabolic Disorders and Comorbidity in OSA	Kazuo Chin
14:00 - 14:30	Fluid shift in Patients with Sleep Disordered Breathing	Takatoshi Kasai
14:30 - 15:00	CPAP and non-CPAP Treatment for OSA	Mary S-M Ip

**Room:** Nusa Dua 3

**Session:** AS2: Assembly of Lung Cancer: Targeted Strategies and Beyond in NSCLC

**Session Chairs:** Kwun Fong, Elisna Syahrudin

**Target Audience:** Pulmonologists, clinicians, researchers, fellows and residents in training interested in thoracic oncology and molecular biology of lung cancer

13:00 - 13:30	Current Molecular Targets in NSCLC	Koichi Hagiwara
13:30 - 14:00	Recent Advances of Molecular Diagnosis for Cancer	Eiso Hiyama
14:00 - 14:30	Second-line Treatment and Beyond of Targeted Therapy in NSCLC	Kazuhisa Takahashi
14:30 - 15:00	Gene Sequencing and Immunotherapy in Lung Malignancies	Bruce Robinson

**Room:** Nusa Dua 4

**Session:** AS1, Assembly of Clinical Allergy: Current Topics on Asthma and COPD Pathogenesis

**Session Chairs:** Shu Hashimoto, Eric Bateman

**Target Audience:** Pulmonologists, clinicians, basic and clinical researchers, registered nurses, fellows and residents in training

13:00 - 13:30	Molecular Targeting Therapy	Klaus Rabe
13:30 - 14:00	Tuberculosis-associated chronic obstructive pulmonary disease (TOPD): structure/function relationship	Eric Bateman
14:00 - 14:30	Role of Airway Mucus Hypersecretion in COPD	Jun Tamaoki
14:30 - 15:00	Airway Epithelial Cells In Asthma Pathogenesis	Shu Hashimoto

**Room:** Uluwatu 1

**Session:** AS4, Assembly of Lung Structure and Function: Phenotyping of Lung Disease

**Session Chairs:** Yasutaka Nakano, Fachrial Harahap

**Target Audience:** Pulmonologists, clinicians, researchers, registered nurses, respiratory therapists, GPs, fellows and residents in training, medical students

13:00 - 13:30	Phenotyping Lung Disease Using Computed Tomography (CT)	Yasutaka Nakano
13:30 - 14:00	Phenotyping Lung Disease Using Magnetic Resonance Imaging (MRI)	Yoshiharu Ohno
14:00 - 14:30	Phenotyping Lung Disease Using Optical Coherence Tomography (OCT)	Harvey O. Coxson

**Room:** Uluwatu 2

**Session:** Oral Session 5: Asthma

**Session Chairs:** Sang Heon Cho, Susanthi Djajalaksana

13:00 - 13:10	Plume Temperature and Force of Fluticasone Propionate/Formoterol pMDI Compared with Fluticasone Propionate/Salmeterol pMDI ( O-A-001)	Jonathan Marshall
13:10 - 13:20	Therapeutic Effects of Histone Deacetylase Enzyme 6 Inhibitors (HDAC6i) in a Murine Asthma Model ( O-A-002)	Yuan Ren
13:20 - 13:30	Tracheobronchial Wall Vessel Remodeling and Endothelial Cell Activation in a Murine Asthma Model (O-A-003)	Xinming Su
13:30 - 13:40	Omalizumab Improves Quality of Life and Asthma Control in Chinese Patients with Moderate-To-Severe Asthma: A Randomised Phase III Study (O-A-004)	Jing Li
13:40 - 13:50	A Novel Thromboxane A2 Receptor Inhibitor, Seratrodast Shows Greater Improvement in Peak Expiratory Flow, Expectoration Score, Sputum Eosinophil Cationic Protein and Albumin Levels as Compared to Montelukast in a Double Blind Comparative Clinical Trial (O-A-005)	Bhupesh Dewan
13:50 - 14:00	Median Mass Aerodynamic Diameter (MMAD) and Fine Particle Fraction (FPF): Influence on Lung Deposition? (O-A-006)	Jonathan Marshall

# DAY 2

## FRIDAY, 14<sup>TH</sup> NOVEMBER

### SESSION TIME: 13:00 - 15:00

**Room:** Uluwatu 2

**Session:** Oral Session 7: Bronchoscopy and Interventional Techniques

**Session Chairs:** Terence S Ting, Eddy Soeratman

14:00 - 14:10	Bronchoscopic Lipiodol-Marking Utilizing Diagnostic Guided-Bronchoscopy to Facilitate Minimal Surgery for Small Pulmonary Nodule (O-B-001)	Masafumi Misawa
14:10 - 14:20	Functional Bronchoscopy in the Management of Airway Stenosis Due to Tracheobronchial Tuberculosis (O-B-003)	Seiichi Nobuyama
14:20 - 14:30	Endobronchial Ultrasound Elastography in the Diagnosis of Mediastinal and Hilar Lymph Nodes (O-B-005)	Takehiro Izumo
14:30 - 14:40	Talc Poudrage Using Catheter Through Flexi-Rigid Thoracoscope Under Local Anesthesia for Malignant Pleural Effusion (O-B-006)	Yuko Ise

**Room:** Uluwatu 3

**Session:** Oral Session 6: COPD

**Session Chairs:** Mohammed Al-Ghobain, Amira Permatasari Tarigan

13:00 - 13:10	Long Acting Beta 2 Agonist Inhibits Cigarette Smoke-Induced Airway Inflammation and Remodeling Through Camp Pathway (O-F-001)	Yi-Han Hsiao
13:10 - 13:20	Methylation Status of Perforin Gene Promoter in Cd4+T Lymphocytes of Patients with Chronic Obstructive Pulmonary Disease (O-F-002)	Lin Liu
13:20 - 13:30	Vitamin D Inhibits Expression and Activity of Matrix Metalloproteinase (MMP) in Human Lung Fibroblasts (HFL-1) Cells (O-F-004)	Hui Jung Kim
13:30 - 13:40	Impact of Extrapulmonary Comorbidities on Quality of Life in Male Patient with Stable COPD (O-F-005)	Bu Xiaoning
13:40 - 13:50	miR-146a Plays Pathogenic Role in Abnormal Inflammatory Response of a Murine COPD Model (O-F-007)	Hario Baskoro
13:50 - 14:00	Evaluating Drug Utilisation Pattern and Direct Cost of Critical Care Management in Patients with Chronic Obstructive Pulmonary Disease in Mangalore, India (O-F-008)	Aiswarya Aravind

**Room:** Uluwatu 3

**Session:** Oral Session 8: COPD

**Session Chairs:** Tomoko Betsuyaku, Budhi Antariksa

14:00 - 14:10	Eosinopenia as a Marker of Outcome in Acute Exacerbations of Chronic Obstructive Pulmonary Disease (O-F-009)	Shagayegh Rahimirad
14:10 - 14:20	Decrease of Interleukin 6 Serum Level, Improvement of SGRQ and Depression Comorbid in COPD Patient Population with which are Treated with Medical Rehabilitation (O-F-010)	Ungky Agus Setyawan
14:20 - 14:30	A summary of the Difficulties in Diagnosis and Treatment of Elderly Patients with Chronic Obstructive Pulmonary Disease (O-F-012)	Qiao Yu
14:30 - 14:40	Clinical Impact of Combined Viral and Bacterial Infection in COPD Patients with Community-Onset Pneumonia (O-F-013)	Kei Nakashima
14:40 - 14:50	Higher Circulating IL-13 in Biomass-Related COPD Compared to Smokers with or without COPD (O-F-014)	Sumeet Nawani
14:50 - 15:00	TNF- $\alpha$ Relationship on Air Flow Resistance Mechanism of COPD (O-F-016)	Nia Marna Premesti

**Room:** Uluwatu 5

**Session:** AS5, Assembly of Environmental and Occupational Health and Epidemiology: Trends in Occupational Lung Diseases in Developing and Developed Nations

**Session Chairs:** Soon-Hee Jung, Mukhtar Ikhsan

**Target Audience:** Pulmonologists, epidemiologists, oncologists, thoracic surgeons, basic scientist of mesothelioma, carcinogenesis, occupational and environmental lung diseases

13:00 - 13:30	Pathology of Asbestos-Related Diseases	Andrew Churg
13:30 - 14:00	Mechanisms for Asbestos-Related Diseases	Richard Lake
14:00 - 14:30	Mesothelioma: Diagnosis and Management	Takashi Nakano
14:30 - 15:00	Compensation for Asbestos-Related Diseases in Korea: Current Situation	Soon-Hee Jung



# DAY 2

## FRIDAY, 14<sup>TH</sup> NOVEMBER

### SESSION TIME: 13:00 - 15:00

**Room:** Uluwatu 6

**Session:** Oral Session 4: Pediatric Lung Disease

**Session Chairs:** Himender Makker, Darmawan Budi Setyanto

13:00 - 13:10	Familial Aggregation of Obstructive Sleep Apnoea Using Children Probands – Obesity Makes The Difference	Albert Martin Li
13:10 - 13:20	Familial Aggregation of Obstructive Sleep Apnoea in Children without Tonsillar Hypertrophy	Albert Martin Li
13:20 - 13:30	5-HT <sub>1A</sub> Receptor in Raphe Magnus Nucleus Modulates Ventilatory Long-Term Facilitation Induced by Chronic Intermittent Hypoxia	Jiao Su
13:30 - 13:40	5-HT <sub>1A</sub> Receptor in Raphe Dorsal Nucleus Modulates Genioglossus Corticomotor Activity During Acute Intermittent Hypoxia	Wei Wang
13:40 - 13:50	The Obstructive Sleep Apnea and Ischemic Heart Disease Study	D J Christopher

**Room:** Uluwatu 7

**Session:** Congress Symposia 6: Respiratory Effectiveness Group (REG) Collaboration Symposium: The Evolving Role of Real-life Research in Respiratory Medicine (Respiratory Effectiveness Group)

**Session Chairs:** TBC

**Target Audience:** Pulmonologists, clinicians, basic and clinical researchers, registered nurses, fellows and residents in training

13:00 - 13:40	Real Life Studies - A Poor Relation or Important Partner to the Respiratory RCT?	David Price
13:40 - 14:20	The Implications of Real-life (Comorbid Conditions, Inhaler Technique, Age and Lifestyle Factors) on Asthma Management: Is There Any Evidence Available?	Omar Usmani
14:20 - 15:00	Leveraging Datasets and Insisting on Quality	Rupert Jones

# DAY 2

## FRIDAY, 14<sup>TH</sup> NOVEMBER

### SESSION TIME: 15:00 - 17:45

**Room:** Nusa Dua 2

**Session:** AS8, Assembly of Tuberculosis: Achieving Zero TB: Make it Happen

**Session Chairs:** Giovanni Battista Migliori, Arifin Nawas

**Target Audience:** All physicians, pulmonologists, nurses, healthcare personels related with TB

15:15 - 15:40	Preventing TB Disease: Improving the Diagnosis and Treatment of TB Infection	Charles Daley
15:40 - 16:05	Latent TB: To Treat or Not to Treat	Dean Schraufnagel
16:05 - 16:30	Moving Towards TB elimination: Experience and Lesson Learned Through ERS Engagement in Europe	Giovanni Battista Migliori
16:30 - 16:55	Public-Private Mix in TB Management	Erlina Burhan
16:55 - 17:20	Current Situation in Asia Pacific: What We Have Learned	Chi Chiu Leung

**Room:** Nusa Dua 3

**Session:** AS7, Assembly of Cellular and Molecular Biology: Hot Topics in Respiratory Cellular and Molecular Biology

**Session Chairs:** Yasuhiro Yamauchi, Kenneth Tsang

**Target Audience:** Pulmonologists, clinicians, basic and clinical researchers, registered nurses, fellows and residents in training interested in respiratory cellular and molecular biology

15:15 - 15:45	Bronchiectasis – Current Understanding of Pathogenesis and Clinical Management	Kenneth Tsang
15:45 - 16:15	Hot Topics in Experimental Animal Model	Takahide Nagase
16:15 - 16:45	Pulmonary Stem Cell: Role in Lung Disease Pathogenesis and Therapy	Toshiaki Kikuchi
16:45 - 17:15	Epithelial Messenchymal Transtition in Airway Epithelial Cells	Yasuhiro Yamauchi

# DAY 2

## FRIDAY, 14<sup>TH</sup> NOVEMBER

### SESSION TIME: 15:00 - 17:45

**Room:** Nusa Dua 4

**Session:** AS6, Assembly of Asthma: Asthma-COPD Overlap Syndrome

**Session Chairs:** Maneechotesuwan Kittipong, Ida Bagus Ngurah Rai

**Target Audience:** Pulmonologists, clinicians, basic and clinical researchers, registered nurses, fellows and residents in training

15:15 - 15:45	Cellular Mechanism of Asthma	Gary Anderson
15:45 - 16:15	Clinical Application of iPSC cell in the respiratory field including COPD and Asthma	Michiaki Mishima
16:15 - 16:45	Management Recommendations	Maneechotesuwan Kittipong
16:45 - 17:15	Future Therapies for Asthma	Peter J Barnes

**Room:** Uluwatu 1

**Session:** AS9, Assembly of Pediatric Lung Disease: OSAS in Children

**Session Chairs:** Albert Li, Darmawan Budi Setyanto

**Target Audience:** Pediatricians, pulmonologists and sleep physicians and dentists, clinicians, researchers, registered nurses, respiratory therapists, GPs, fellows and residents in training interested in sleep apnea

15:15 - 15:45	Burden of OSAS in Children in Asia Pacific Region	Albert Li
15:45 - 16:15	Can We Apply Personalized Medicine to the Management of Childhood Obstructive Sleep Apnea: Newer Treatment Modalities	Carole L. Marcus
16:15 - 16:45	What Happens to a Child with Untreated Obstructive Sleep Apnea?	Rosemary S. Horne
16:45 - 17:15	PSG Should Be Carried Out Before Adenotonsillectomy for Childhood OSA – A Debate	Bambang Supriyatno

**Room:** Uluwatu 2

**Session:** Oral Session 9: Lung Cancer

**Session Chairs:** Koichi Hagiwara, Anarima

15:15 - 15:25	Integrative Analysis of DNA Copy Number in Metastatic NSCLC Identifies Drug Sensitivity to Afatinib (O-J-001)	Mian Xie
15:25 - 15:35	Simple Latex Balloon Reservoir System for Exhaled Breath Condensate as Noninvasive Molecular Diagnostic Method in Lung Cancer: Preliminary Study (O-J-004)	Achmad Hudoyo
15:35 - 15:45	Epigenetic Alterations of IGF1 Regulate TAZ Expression in Lung Cancer (O-J-005)	Mian Xie
15:45 - 15:55	Construction of Expression Vector, Establishment of Stably Transfected A549 Cell Line and Preliminary Research on the Function of RBM5 Gene (O-J-007)	Bixiu He
15:55 - 16:05	Cisplatin-Induced Downregulation of RBM5 Increases Drug Resistance by Activating Autophagy in Non-small Cell Lung Cancer Cells (O-J-008)	Wang Ke

**Room:** Uluwatu 2

**Session:** Oral Session 10: Clinical Respiratory Medicine

**Session Chairs:** M. Al-Ghobain, Yani Jane Sugiri

16:15 - 16:25	Factors Associated with Radiologic Progression of Non-Cystic Fibrosis Bronchiectasis During Long-Term Follow Up (O-E-001)	Jae Ho Lee
16:25 - 16:35	Thromboembolic Events in Malignant Pleural Mesothelioma (O-E-002)	Deniz Koksal
16:35 - 16:45	Impact of Ancillary Findings in Patients with Suspected Pulmonary Embolism (O-E-003)	Marika Bajc
16:45 - 16:55	Preparation and Characterization of Controlled Release Microspheres for Management of Pulmonary Hypertension (O-E-004)	Aparna Saigal
16:55 - 17:05	Pregnancy Doesn't Always Cause Deterioration of Lymphangiomyomatosis (O-E-005)	Katsutoshi Ando
17:05 - 17:15	Frequency of Pulmonary Medicine Consultation in Hyperbaric Medicine Clinic (O-E-006)	Erdinc Ercan
17:15 - 17:25	A Multi-Disciplinary Survey on Attitudes and Perceptions on Adult Outpatient Tracheostomy Care in the Philippine General Hospital (O-E-007)	Johann Paolo Augusto D. Almazar



# DAY 2

## FRIDAY, 14<sup>TH</sup> NOVEMBER

### SESSION TIME: 15:00 - 17:45

**Room:** Uluwatu 3

**Session:** Oral Session 11: Tuberculosis

**Session Chairs:** Behera, VK Vijayan

16:15 - 16:25	Clinical Benefit of Delamanid (OPC-67683) in the Treatment of Multidrug-Resistant Tuberculosis Patients in China (O-Q-001)	Qing Zhang
16:25 - 16:35	Xpert MTB/RIF Testing of Pooled Induced Sputum (O-Q-003)	T.K. Lim
16:35 - 16:45	Analysis of Medical Burden of TB Patients in DOTs of Taiwan Health Insurance (O-Q-006)	Tsung Hsien Yang
16:45 - 16:55	Comparison of Characteristics of Patients with Tuberculosis Diagnosed and Not Diagnosed by Fiberoptic Bronchoscopy (O-Q-007)	Tsugitoshi Onuki
16:55 - 17:05	Clinical and Bronchoscopic Characteristics of Patients Diagnosed with Endobronchial Tuberculosis (EBTB) at Selayang Hospital, Selangor Malaysia (O-Q-008)	Siti Kamariah Othman
17:05 - 17:15	Audit of Tuberculosis Treatment Interrupters in Kandy District, Sri Lanka (O-Q-009)	Dhamith Nandadeva
17:15 - 17:25	Prospective Study: IL-10 And IL-17 Plasma Levels During Oral Antituberculosis Treatment with Sputum Conversion and Successful Treatment in Active Lung Tuberculosis Patient (O-Q-010)	Fitri Emizola
17:25 - 17:35	The Profile of Matrix Metalloproteinase-9 (MMP-9) Serum Levels and its Correlation with Disease Severity in Patients with Pulmonary Tuberculosis (O-Q-011)	Eliana Muis
17:35 - 17:45	The Effect of Ethanol Extract Propolis (EEP) on the Level of IFN- $\gamma$ and Superoxide Dismutase (SOD) Activities in Patients with MDR Tuberculosis (O-Q-012)	Ariani Permatasari

**Room:** Uluwatu 5

**Session:** Congress Symposia 7: Nature and Respiratory Management

**Session Chairs:** Winariani Koesoemoprodjo, Bruce Robinson

**Target Audience:** Pulmonologists, epidemiologists, oncologists, thoracic surgeons, basic scientists of mesothelioma, carcinogenesis, occupational and environmental lung diseases

15:15 - 15:45	Potential Carcinogenicity of Man-Made Fibers in Lung Cancer	Faisal Yunus
15:45 - 16:15	Respiratory Management in Natural Disaster	Bruce Robinson
16:15 - 16:45	Air Pollution and Respiratory Diseases in the Asia-Pacific Region	Mukhtar Ikhsan
16:45 - 17:15	Smoking and Lung-Related Disease: Current Data	Talant Sooronbaev

**Room:** Uluwatu 7

**Session:** AS10, Assembly of Clinical Respiratory Medicine: Interesting Clinical Cases

**Session Chairs:** Philip Eng, Yusuf Subagio

**Target Audience:** Pulmonologists, clinicians, basic and clinical researchers, registered nurses, fellows and residents in training

15:15 - 16:00	Interesting Clinical Cases 2	Philip Eng
16:00 - 16:45	Interesting Clinical Cases 2	David Lam

# DAY 3

## SATURDAY, 15<sup>TH</sup> NOVEMBER

### SESSION TIME: 08:00 - 10:00

**Room:** Nusa Dua 2

**Session:** AS12, Assembly of Bronchoscopy and Interventional Techniques Assembly: Novel Bronchoscopy and Interventional Technique

**Session Chairs:** Takashi Ishida, Oea Khairsyaf

**Target Audience:** Pulmonologists, interventional and bronchoscopist, clinicians, basic and clinical researchers, fellows and residents in training

8:00 - 8:20	Latest Update on Interventional Bronchoscopy	Teruomi Miyazawa
8:20 - 8:40	Navigational Bronchoscopy: The Advances	Takashi Ishida
8:40 - 9:00	Bronchial Thermoplasty in the Management of Airway Obstruction	Pyng Lee
9:00 - 9:20	Management of Endobronchial Tuberculosis	Wahju Aniwidyaningsih
9:20 - 9:40	Ariway Stenting: The Future and Drawbacks	TBC

**Room:** Nusa Dua 3

**Session:** Congress Symposia 8: Comorbidity in IPF

**Session Chairs:** Yasuhiro Kondo, Dianati Sutoyo

**Target Audience:** Pulmonologists, clinicians, basic and clinical researchers, fellows and residents in training interested in interstitial lung disease

8:00 - 8:30	Infection in Interstitial Lung Disease	Sita Andarini
8:30 - 9:00	Pathogenesis of IPF, Emphysema, and the Combined	Masahito Ebina
9:00 - 9:30	Acute Exacerbation of IPF	Yasuhiro Kondo
9:30 - 10:00	Comorbidity in IPF	Arata Azuma

**Room:** Nusa Dua 4

**Session:** AS11, Assembly of COPD: Redefining COPD

**Session Chairs:** Masaharu Nishimura, Muhammad Amin

**Target Audience:** Pulmonologists, clinicians, basic and clinical researchers, registered nurses, fellows and residents in training interested in COPD

8:00 - 8:30	COPD Phenotyping	Jorgen Vestbo
8:30 - 9:00	Biomarkers Assessment in COPD	Diahn-Warng Perng
9:00 - 9:30	3D CT Airway Analysis	Masaharu Nishimura
9:30 - 10:00	Assessment of Quality of Life in COPD	Paul Jones

**Room:** Uluwatu 1

**Session:** Best Oral Presentation Selection Session

**Judges:** Dean Schraufnagel, Faisal Yunus, Harvey Coxson

8:00 - 8:10	Flexible Fibre-optic Bronchoscopy, Obesity and Sleep Disordered Breathing: Patients Characteristics and Complications in 570 Cases (O-B-002)	Terence S T Ting
8:10 - 8:20	Dynamic Change of House Dust Mites and Its Components- sIgE & sIgG4 in Specific Immunotherapy (O-D-001)	Baoqing Sun
8:20 - 8:30	Mild to Moderate Airflow Obstruction is Not Directly Associated with Coronary Arteriosclerosis But Is Associated with the Elevation of Serum Creatinine Levels (O-F-006)	Takehiko Kobayashi
8:30 - 8:40	MEF50/MEF25 and RV/TLC% Correlate with HRCT Phenotypes of COPD (O-F-011)	Xian Wen Sun
8:40 - 8:50	Simple Shipping Procedure Using Filter Paper for Detection of EGFR and KRAS Mutation in Malignant Pleural Fluid from Remote Area in Indonesia: Preliminary Study (O-J-003)	Asep Ridwanulloh
8:50 - 9:00	Translational Evidence of Zinc-finger E-box Binding Homeobox 1 Involvement in Acquired Resistance to Gefitinib in Non-small Cell Lung Cancer (O-J-006)	Fariz Nurwidya
9:00 - 9:10	BREAK	
9:10 - 9:20	Determinants of Annual Change in Peak Expiratory Flow Rate (PEFR) and Peak Inspiratory Flow Rate (PIFR) Values in a Cohort of Indian Children Aged 4-10 Yrs (O-L-003)	Rahul Kodgule
9:20 - 9:30	Correlation Between Endotracheal Aspirate Gram Stain and Results of Bacterial Culture among ICU Patients Admitted in a Tertiary Hospital in Cebu from September 2009 to August 2010: A Retrospective Study (O-N-001)	Beatriz C. Tan
9:30 - 9:40	Dynamic Chest Imaging Evaluation in Severe Avian Influenza H7N9 Virus Infected Patients: A Retrospective Small-scale Study (O-N-008)	Cheng Chen
9:40 - 9:50	Evaluation Oof Brainstem Auditory Evoked Potentials in Male Patients with Moderate and Severe Obstructive Sleep Apnea Syndrome (O-O-003)	Jiao Su
9:50 - 10:00	A Case-Control Comparison of the Mantoux Test and a Commercial Interferon Gamma Release Assay (IGRA) in Adults: First Report in a Sri Lankan Setting (O-Q-004)	Champa Neelakanthi Ratnatunga



# DAY 3 SATURDAY, 15<sup>TH</sup> NOVEMBER

## SESSION TIME: 08:00 - 10:00

**Room:** Uluwatu 2

**Session:** Oral Session 12: Asthma

**Session Chairs:** Maneechotesuwan Kittipong, Wiwien Heru Wiyono

8:00 - 8:10	Asthma Severity and Phenotypes Associated with Vocal Cord Dysfunction (O-A-008)	Philip Bardin
8:10 - 8:20	STAT1/STAT6 Phosphorylation Balancing of CD4+ Memory T Cells in Asthma and COPD (O-A-009)	Zhihong Chen
8:20 - 8:30	Add-on Effect of Tiotropium Bromide to ICS/LABA Combination Inhaler for Peripheral Airways among Patients with Severe Uncontrolled Asthma (O-A-010)	Masayuki Hojo
8:30 - 8:40	Identification of Attitudinal Clusters in Patients with Asthma: Analysis from REALISE Asia (O-A-011)	Sang Heon Cho
8:40 - 8:50	Early Effect of Maternal Allergic Asthma on T-Regulatory Cells Immune Response in Cord Blood of Offsprings (O-A-012)	Jing Liu
8:50 - 9:00	Discrepancy Between Patient-Perception and Guideline-Defined Asthma Control in Asia: A Survey of Over 2400 Patients (O-A-013)	Sang Heon Cho

**Room:** Uluwatu 2

**Session:** Congress Symposia 10: Medical Ethics and Patient Safety

**Session Chairs:** Arifin Nawas, Menaldi Rasmin

**Target Audience:** Pulmonologists, clinicians, basic and clinical researchers, registered nurses and respiratory technicians, fellows and residents in training and medical students

9:10 - 9:30	Medical Ethics	Prijo Sidipratomo
9:30-10:00	Patient Safety: Indicator and Improvement	Adib Yahya

**Room:** Uluwatu 3

**Session:** Oral Session 13: Tuberculosis

**Session Chairs:** T.K. Lim, Asif M Mahmud

8:00 - 8:10	Tuberculosis in Indonesian Hemodialysis Patients: A Single Centre Experience (O-Q-013)	Riri Andri Muzasti
8:10 - 8:20	Treatment Outcomes Among Pulmonary and Extra Pulmonary Tuberculosis Patients in Malaysia (O-Q-014)	Abdul Razak Muttalif
8:20 - 8:30	Characteristics and Problems of Foreign Tuberculosis Treated in Our Hospital (O-Q-015)	Jin Takasaki
8:30 - 8:40	Two-Year Follow-Up Study of TB Patients Put On DOTS from South India (O-Q-016)	K.Venugopal
8:40 - 8:50	The Profile of Interleukin (IL)-12p70 and IL-12p40 Serum Level Among Active Tuberculosis Patients, Latent with Positive Interferon Gamma Release Assay (IGRA), Contact with Negative IGRA (O-Q-017)	Nur Ahmad Tabri
8:50 - 9:00	A Study on Inoculum Density and Reproducibility of Drug Susceptibility Testing by DAC (Disc Agarose Channel) (O-Q-018)	Hyejin Kim

**Room:** Uluwatu 3

**Session:** Oral Session 14: Tuberculosis

**Session Chairs:** Mostafizur Rahman, Nur Ahmad Tabri

9:00 - 9:10	Comparative Analysis of DU Region in the <i>Mycobacterium bovis</i> BCG-Korea Strain (O-Q-019)	Eun-Hee Lee
9:10 - 9:20	Correlation Between Positivity of Acid Fast Bacilli (AFB) and Level of Interferon- $\gamma$ (IFN- $\gamma$ ) Among Tuberculosis Patients in Makassar (O-Q-020)	Erwin Arief
9:20 - 9:30	A Cross Sectional Study of Pengawas Minum Obat (PMO) in Going Hand in Hand with Tuberculosis Patients in Jakarta Respiratory Center (JRC) – The Indonesian Association Against Tuberculosis (O-Q-021)	Fabianto Santoso
9:30 - 9:40	Clinical Experience with Immunotherapy Regimens in TB Patients (O-Q-022)	Dmytro Butov
9:40 - 9:50	Smoking and Alcohol Consumption as Risk Factors of Pulmonary Tuberculosis Development in Medan, Indonesia (O-Q-023)	Bintang Yinke Magdalena Sinaga
9:50 - 10:00	Magnetic Bead Protocol Denoted Cogent Results for Culturing <i>Mycobacterium tuberculosis</i> from Sputum Specimens (O-Q-024)	Sungweon Ryoo

# DAY 3

SATURDAY, 15<sup>TH</sup> NOVEMBER  
SESSION TIME: 08:00 - 10:00

**Room:** Uluwatu 5

**Session:** Congress Symposia 9: Lung Cancer in Asia Pacific

**Session Chairs:** Bruce Robinson, Anarima

**Target Audience:** Pulmonologists, clinicians, basic and clinical researchers, fellows and residents in training interested in lung cancer

8:00 - 8:30	Lung Cancer in Indonesia and AP region: shifting disease paradigm	Elisna Syahrudin
8:30 - 9:00	Lung Cancer Screening program: any good news	Kazuma Kishi
9:00 - 9:30	Genetic Heterogeneity in Lung Cancer: Diversity in Asia-Pacific Region	Koichi Hagiwara
9:30 - 10:00	Challenges to Lung Cancer Guidelines Development in Asia Pacific	Kwun Fong

**Room:** Uluwatu 7

**Session:** AS13, Assembly of Clinical Respiratory Medicine: Interesting Clinical Cases

**Session Chairs:** Ratnawati, Sutji Mariono, D Behera

**Target Audience:** Pulmonologists, clinicians, basic and clinical researchers, fellows and residents in training, and medical students

8:00 - 8:50	Interesting Clinical Cases 1	Philip Eng
8:50 - 9:40	Interesting Clinical Cases 1	Low Su Ying

# DAY 3

SATURDAY, 15<sup>TH</sup> NOVEMBER  
SESSION TIME: 10:00 - 10:30

**Room:** Nusa Dua 4

**Session:** Presidential Lecture

**Session Chair:** Faisal Yunus, Arth Nana

**Target Audience:** Pulmonologists, clinicians, basic and clinical researchers, registered nurses and respiratory technicians, fellows and residents in training, and medical students

10:00 – 10:30	Diversity and Heterogeneity of Respiratory Disease in Asia Pacific: The Step Forward	Norbert Berend
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# DAY 3 SATURDAY, 15<sup>TH</sup> NOVEMBER

## SESSION TIME: 10:45 - 11:45

**Room:** Nusa Dua 2

**Session:** Pre-Luncheon Symposia 6: Unmet Needs in Asthma; Future Treatment Options

**Session Chair:** Hadiarto Mangunegoro

**Sponsored by:** Boehringer Ingelheim

10:45- 11:45	Unmet Needs in Asthma; Future Treatment Options	Mario Castro
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**Room:** Nusa Dua 3

**Session:** Pre-Luncheon Symposia 5: Lung Health Redefined, Discover Electromagnetic Navigation Bronchoscopy

**Session Chair:** Ashutosh Sachdeva

**Sponsored by:** Covidien Private Limited

10:45- 11:45	Discover Electromagnetic Navigation Bronchoscopy Ashutosh Sachdeva	Ashutosh Sachdeva
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**Room:** Nusa Dua 4

**Session:** Pre-Luncheon Medical Forum 5: New Insight into Inflammation in Obstructive Respiratory Diseases - Spotlight on COPD and ACOS

**Session Chair:** Hadiarto Mangunegoro

**Sponsored by:** Takeda Pharmaceuticals International GmbH

10:45 - 10.50	Welcome and Introduction	Hadiarto Mangunegoro
10.50 - 11.05	COPD Management: The Present and The Future	Faisal Yunus
11:05 - 11.25	Inhaled Steroids or PDE4 Inhibitors: Which Patient for Which Medication?	Klaus F. Rabe
11.25 - 11.45	Asthma-COPD Overlap Syndrome: Where are We Now?	Eric D. Bateman

# DAY 3

## SATURDAY, 15<sup>TH</sup> NOVEMBER

### SESSION TIME: 12:00 - 13:00

**Room:** Nusa Dua 2

**Session:** Luncheon Symposia 6: Small Airways in Asthma: Time to Rethink

**Session Chair:** Budhi Antariksa

**Sponsored by:** Chiesi

12.00 – 12.20	Update Asthma Therapy in Asia Pasific and Indonesia	Faisal Yunus
12.20 – 12.40	Combination Therapy in Asthma: A Focus on Small Airways	Alberto Papi
12.40 – 13.00	Discussion	All

**Room:** Nusa Dua 3

**Session:** Luncheon Symposia 5: A to Z Management in Lung Disease “Inhalation Therapy on Asthma and COPD”

**Session Chair:** Muhammad Amin

**Sponsored by:** AstraZeneca

12.00 - 12.20	New Paradigm : Combination Therapy as Maintenance and Reliever in Asthma	Peter J Barnes
12.20 - 12.40	Do All ICS/LABA Provide Equal Exacerbation Protection and Safety Benefits to COPD Patients?	Christine R Jenkins
12.40 - 13.00	Discussion	

**Room:** Nusa Dua 4

**Session:** Luncheon Symposia 4: Adult Pneumococcal Disease Prevention

**Session Chair:** Dianiati Kusumo Sutoyo

**Sponsored by:** Pfizer Vaccine

12.00 - 12.20	Addressing The Significant Burden of Pneumococcal Disease in Elderly - Investigating The Impact of Co-morbidities and Risk Factors	Sita Andarini
12. 20 - 12.45	Meeting the Adult Challenge: Prevention of Pneumococcal Disease with Conjugate Vaccines	Charles Feldman
12.45- 13.00	Q&A	All

# DAY 3

SATURDAY, 15<sup>TH</sup> NOVEMBER  
SESSION TIME: 13:00 - 14:00

**Room:** Nusa Dua 4

**Session:** Memorial Lecture 1: Ann Janet Woolcock Research Award

**Session Chairs:** Michiaki Mishima

13:00 -13:30	Why Physiologic Investigation in Asthma is More Relevant Now Than Ever	Greg King
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**Room:** Nusa Dua 4

**Session:** Memorial Lecture 2: Michiyoshi Harasawa Research Award

**Session Chairs:** Yoshinosuke Fukuchi

13:30 -14:00	A Challenge to Overcome of Drug Resistance in Lung Cancer	Kazuhisa Takahashi
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# DAY 3

## SATURDAY, 15<sup>TH</sup> NOVEMBER

### SESSION TIME: 14:00 - 16:00

**Room:** Nusa Dua 2

**Session:** AS16, Assembly of Pulmonary Circulation: What's New in Pulmonary Hypertension?

**Session Chairs:** Hiroshi Kimura, Triya Damayanti

**Target Audience:** Pulmonologists, clinicians, basic and clinical researchers, fellows and residents in training, and medical students

14:00 - 14:30	What's New in Pulmonary Hypertension	Hiroshi Kimura
14:30 - 15:00	Pulmonary Hypertension Associated with Pulmonary Disease/Hypoxia	Masayuki Hanaoka
15:00 - 15:30	Recent Strategies for Chronic Thromboembolic Pulmonary Hypertension	Nobuhiro Tanabe
15:30 - 16:00	Pulmonary Hypertension - Are We Making Progress?	Nicholas Hill

**Room:** Nusa Dua 3

**Session:** AS15, Assembly of Interstitial Lung Disease: Interstitial Lung Disease Diagnosis and Treatment

**Session Chairs:** Masahito Ebina, Ryushi Tazawa

**Target Audience:** Pulmonologists, clinicians, basic and clinical researchers, registered nurses and respiratory technicians, fellows and residents in training, and medical students interested in interstitial lung disease

14:00 - 14:25	Current Guidelines of ILD	Yoshikazu Inoue
14:25 - 14:45	Rare Lung Diseases	Ryushi Tazawa
14:45 - 15:10	Potential Pitfalls of IPF Diagnosis	Ganesh Raghu
15:10 - 15:35	Treatment of Idiopathic Pulmonary Fibrosis and Acute Exacerbations: Lessons from Japan	Arata Azuma
15:35 - 16:00	IPF in Korea: Diagnosis and treatment approach	Dong Soon Kim

**Room:** Nusa Dua 4

**Session:** AS14, Assembly of Respiratory Infection (non-TB): Community-Acquired Pneumonia in Asia Pacific

**Session Chairs:** Jun-Hee Woo, M. Sardikin

**Target Audience:** Pulmonologists, clinicians, basic and clinical researchers, registered nurses and respiratory technicians, fellows and residents in training, and medical students

14:00 - 14:40	CAP, Asia Pacific Guideline	Jun-Hee Woo
14:40 - 15:20	How Do We Treat CAP and Its Complications	Hadiarto Mangunegoro
15:20 - 16:00	Improving Outcomes of CAP	Ronald Grossman



# DAY 3

## SATURDAY, 15<sup>TH</sup> NOVEMBER

### SESSION TIME: 14:00 - 16:00

**Room:** Uluwatu 1

**Session:** Congress Symposia 11: Updates on Pleural Disease

**Session Chairs:** Lee Pyng, Boedi Swidarmoko

**Target Audience:** Pulmonologists, interventional and bronchoscopists, clinicians, basic and clinical researchers, registered nurses and respiratory technicians, fellows and residents in training, and medical students

14:00 - 14:30	Update in Pleural Disease	Richard Light
14:30 - 15:00	Managing Tuberculosis Pleural Disease	Menaldi Rasmin
15:00 - 15:30	Advances in Pneumothorax Management: COPD vs Tuberculosis Pneumothorax	Lee Pyng
15:30 - 16:00	New Therapy for Pleural Infection	Gary Lee

**Room:** Uluwatu 2

**Session:** Oral Session 15: Lung Cancer

**Session Chairs:** D. Behera, Achmad Hudoyo

14:00 - 14:10	A Specific Serum Chemokine Network Correlates with Tumorigenesis and Prognosis of Patients with Non-Small Cell Lung Cancer (O-J-009)	Dawei Yang
14:10 - 14:20	Kruppel-Like Factor 2 Improves Postoperative Prognosis of Lung Adenocarcinoma Patients (O-J-010)	Meiji Itakura
14:20 - 14:30	Improving the Quality of Life for Ambulatory Patients with Advance Lung Cancer through Phone Triaging (O-J-011)	Abdulaziz Aljohani
14:30 - 14:40	Differentially Expressed Glycosylated Patterns of Alpha-1-Antitrypsin as Serum Biomarkers for the Diagnosis of Lung Cancer (O-J-012)	Yiqian Liang
14:40 - 14:50	Epidermal Growth Factor Receptor Mutations in Lung Squamous Cell Carcinoma in Smokers and Never Smokers (O-J-013)	Tan Jiunn Liang
14:50 - 15:00	Study of Mechanism of Reverse of COX-2 Inhibitor Celecoxib on Acquired Resistance to Gefitinib in Non-Small Cell Lung Cancer (O-J-014)	Qin-Fang Deng

**Room:** Uluwatu 2

**Session:** Oral Session 17: Cellular and Molecular Biology

**Session Chairs:** Yasuhiro Yamauchi, Wang Ke

15:00 - 15:10	Activation of Lymphocytes Induced by RSV Persistently Infected Bronchial Epithelial Cells (O-C-001)	Ling Qin
15:10 - 15:20	Mesenchymal Stem Cell: Does it Work in an Experimental Model with Acute Respiratory Distress Syndrome? (O-C-002)	Erdal Karaoz
15:20 - 15:30	An In Vitro Model of Sick Building Syndrome Using Human Bronchial Epithelial Cells (O-C-003)	Kumi Matsushita
15:30 - 15:40	The Role of the Receptor for Advanced Glycation End Products in LPS-Induced Lung Injury in Mice (O-C-004)	Mei He
15:40 - 15:50	Role of Interferon Regulatory Factor 1 Based on Next Generation Sequencing in Mouse Model of Acute Respiratory Distress Syndrome (O-C-005)	Yang Se-Ran

**Room:** Uluwatu 3

**Session:** Oral Session 16: COPD

**Session Chairs:** Soo-Taek Uh, Mohammed Al Ghobain

14:00 - 14:10	The Efficacy and Safety of Inhaled Umeclidinium Bromide/Vilanterol in Asian Patients with Chronic Obstructive Pulmonary Disease (O-F-017)	Jinping Zheng
14:10 - 14:20	Clinical Significance of Interleukin (IL)-33 in Chronic Obstructive Pulmonary Disease (O-F-018)	Chin Kook Rhee
14:20 - 14:30	Number of Pack Year Smoked Decreases The Distance Travelled in 6MWT (O-F-019)	Mohit Bhatia
14:30 - 14:40	The Diagnostic Value of Macao Predictive Values in Chronic Obstructive Lung Disease of Advanced Age by Impulse Oscillometry (O-F-020)	Zhang Xiao Zhan
14:40 - 14:50	Hydrogen Sulfide Attenuates Airway Inflammation in Rats with Chronic Obstructive Pulmonary Disease (O-F-021)	Bai-Mei He
14:50 - 15:00	Differences Between CAT and mMRC in Korean COPD Patients (O-F-022)	Chin Kook Rhee
15:00 - 15:10	Risk Factors for Discontinuation of Roflumilast in COPD Patients (O-F-023)	Chin Kook Rhee

# DAY 3

## SATURDAY, 15<sup>TH</sup> NOVEMBER

### SESSION TIME: 14:00 - 16:00

**Room:** Uluwatu 3

**Session:** Oral Session 18: Critical Care

**Session Chairs:** Sang-Bum Hong, Abdelhaleem Bella

15:20 - 15:30	Evaluation of Impact of Protective Over-shoes on Floor Contamination of ICUs. Is the Practice a Paradigm? (O-G-002)	Mohammad Hosein Rahimirad
15:30 - 15:40	Evaluation of Thrombocytopenia and Its Trend as a Marker of Mortality in Medical Intensive Care Unit Patients (O-G-003)	Mohammadhossein Rahimirad
15:40 - 15:50	Endotracheal Tube Cuff (ETT cuff) Pressure of Patients Admitted at the Intensive Care Units (ICU) of the Philippine General Hospital (The PGH ETT Cuff Study) (O-G-004)	Joann Kathleen B. Ginete
15:50- 16:00	Nutritional Status of Critically-Ill Patients Admitted at the Intensive Care Units (ICUs) of the Philippine General Hospital (PGH) (O-G-008)	Joann Kathleen B. Ginete

**Room:** Uluwatu 5

**Session:** AS 17, Assembly of Clinical Respiratory Medicine: Smoking Cessation

**Session Chairs:** Jennifer Mendoza, Jamal Zaini

**Target Audience:** Pulmonologists, interventional and bronchoscopists, clinicians, basic and clinical researchers, registered nurse and respiratory technicians, fellows and residents in training, and medical students

14:00 - 14:30	Smoking-related disease in Asia-Pacific: Current Data	Ngo Quy Chau
14:30 - 15:00	Radiological Changes in Smokers: Early Detection for Lung Function Abnormalities	Yoshiharu Ohno
15:00 - 15:30	Smoking and Lung Disease in Children	Jennifer Mendoza
15:30 - 16:00	e-cigarettes	Dean Schraufnagel

**Room:** Uluwatu 7

**Session:** Young Investigator Awardees Session

**Session Chairs:** Chunxue Bai, Giovanni Batteista Migliori

14:00- 14:30	ERS Young Investigator: The Relationship Between Environment and the Prevalence of Allergy Rhinitis in Pre-school Children in Guangzhou City	Jiaying Luo
14:30- 15:00	ERS Young Investigator: Associations of sRAGE and S100A12 on Postoperative Respiratory Complications According to Different Two Ventilation Modes after Laparoscopic-assisted Colorectal Surgery	So Young Yang
15:00- 15:30	ATS Young Investigator: Identifying early signs and symptoms of impending asthma exacerbations in children	Katherine Rivera-Spoljaric
15:30- 16:00	ATS Young Investigator: Difference in Outcomes between Senior Physician and Medicine Resident -led Code Blues	Rebecca E. Sell



# DAY 3

## SATURDAY, 15<sup>TH</sup> NOVEMBER

### SESSION TIME: 16:15 - 17:55

**Room:** Nusa Dua 2

**Session:** Congress Symposia 12: TB Risk Factors and Intervention

**Session Chairs:** Francis Drobniowski, Laksmi Wulandari

**Target Audience:** Pulmonologists, clinicians, basic and clinical researchers, registered nurse and respiratory technicians, fellows and residents in training, and medical students, responsible for TB

16:15 - 16:40	Tuberculosis, tobacco and smoking cessation	Asif M Mahmud
16:40 - 17:05	TB and Diabetes control	Roslina Abdul Manap
17:05 - 17:30	TB-HIV	Charles Yu
17:30 - 17:55	TB Control and MDR-XDR Prevention in India	V K Vijayan

**Room:** Nusa Dua 3

**Session:** AS19, Assembly of Interstitial Lung Disease: IPF - The Next Target

**Session Chairs:** Masahito Ebina, Arata Azuma

**Target Audience:** Pulmonologists, clinicians, basic and clinical researchers, registered nurse and respiratory technicians, fellows and residents in training, and medical students

16:15 - 16:40	Smoking-Related Interstitial Lung Disease	Andrew Churg
16:40 - 17:05	IPF: What We Have Learned	Toshihiro Nukiwa
17:05 - 17:30	Update on the Treatment of IPF	Ganesh Raghu
17:30 - 17:55	IPF: The Future Ahead	Masahito Ebina

**Room:** Nusa Dua 4

**Session:** AS18, Assembly of COPD: COPD, Progress on the Management

**Session Chairs:** Wisia Wedzicha, Muljadi

**Target Audience:** Pulmonologists, clinicians, basic and clinical researchers, registered nurse and respiratory technicians, fellows and residents in training, and medical students

16:15 - 16:40	Should We Treat Earlier Stage of COPD?	Wisia Wedzicha
16:40 - 17:05	Exacerbation in COPD: The New Concept	Alejandro Casas Herrera
17:05 - 17:30	Comorbidities in COPD	Suzanne Hurd
17:30 - 17:55	Moving COPD Beyond FEV1: Role of Pulmonary Imaging	Harvey Coxson

**Room:** Uluwatu 1

**Session:** Congress Symposia 13: Difficult Respiratory Infections

**Session Chairs:** Isnun Pradjoko, Teguh Sartono

**Target Audience:** Pulmonologists, clinicians, basic and clinical researchers, registered nurse and respiratory technicians, fellows and residents in training, and medical students

16:15 - 16:45	Managing Respiratory Infection in ICU	Curtis Sessler
16:45 - 17:15	Preventing Chronic Lung Infections: Lessons from Cystic Fibrosis	Thomas Ferkol
17:15 - 17:45	Pulmonary Infection in Chronic Respiratory Disease	Ronald Grossman

**Room:** Uluwatu 2

**Session:** Oral Session 19: Asthma

**Session Chairs:** Philip Bardin, Helmia Hasan

16:15 - 16:25	Patterns of Hospitalization and Healthcare Utilization Across Attitudinal Clusters: Analysis from Patient Survey in Asia (O-A-014)	Diahn-Warnig Perng
16:25 - 16:35	Increasing Level of Interleukin-10, Interleukin-17 and ACT Scoring in Asthma Bronchial Patient with Vitamin D Deficiency after 2 Months Supplementation of 800 IU Vitamin D (O-A-015)	Dwi Yulianti
16:35 - 16:45	Patient Handling Study of Fluticasone Propionate/Formoterol Fumarate Pressurized Metered-Dose Inhaler (O-A-016)	Sanjeeva Dissanayake
16:45 - 16:55	Asthma Self Monitoring, Behaviour Modification and Medication Compliance in Comparison to Patients with Hypertension / High Cholesterol and Link to Asthma Hospitalisation Profile (O-A-017)	Amy E L Stebbings
16:55 - 17:05	Associations of Asthma Control with Systemic Inflammations (O-A-018)	Harun Iskandar
17:05 - 17:15	Long-term Efficacy of Omalizumab in Patients with Severe Asthma (O-A-019)	Junko Saji

# DAY 3 SATURDAY, 15<sup>TH</sup> NOVEMBER

## SESSION TIME: 16:15 - 17:55

**Room:** Uluwatu 3

**Session:** Oral Session 20: Environmental & Occupational Health and Epidemiology Assembly

**Session Chairs:** Akira Umeda, Richard Lake

16:15 - 16:25	Adverse Respiratory Health Effects of Tear Gas (O-H-001)	Benjamas Chuaychoo
16:25 - 16:35	Effect of Incomplete Smoking Cessation with Varenicline or Nicotine Patch on Vascular Endothelial Function as Assessed by Flow-Mediated Vasodilation (O-H-002)	Akira Umeda
16:35 - 16:45	Newly Established ELISA for N-ERC/Mesothelin Improves Diagnostic Accuracy in Patients with Suspected Pleural Mesothelioma (O-H-003)	Tadashi Sato
16:45 - 16:55	Welders' Siderosis: a Retrospective Cohort Study on Welder's Pneumoconiosis Patients with Small Round Opacities on Chest Radiograph (O-H-004)	Ling Mao
16:55 - 17:05	Indoor PM2.5 and CO Levels While Burning Mosquito Coils and Its Associated Respiratory Morbidity (O-H-005)	Rahul Kodgule

**Room:** Uluwatu 5

**Session:** AS20, Assembly of Lung Cancer: What's New in Small Cell Lung Cancer and Neuroendocrine Tumor of the Lung

**Session Chairs:** Kazuhisa Takahashi, Sita Andarini

**Target Audience:** Pulmonologists, clinicians, basic and clinical researchers, registered nurse and respiratory technicians, fellows and residents in training, and medical students interested in lung cancer

16:15 - 16:45	Molecular Signature of SCLC: The Difference with NSCLC	Kwun Fong
16:45 - 17:15	Neuroendocrine Tumor of the Lung	Chong-Kin Liam
17:15 - 17:45	Any Good News for SCLC?	Chunxue Bai

**Room:** Uluwatu 7

**Session:** Young Investigator Awardees Session

**Session Chairs:** Soon-Hee Jung, Shu Hashimoto

16:15 - 16:45	APSR Young Investigator - The Association Between Occupational and Environmental Asbestos Exposure and Asbestosis in Korea-Asbestos Health Damage Relief System	Jun-Pyo Myong
16:45 - 17:15	APSR Young Investigator - miR-15a Induces Cell Apoptosis and Inhibits Metastasis by Targeting BCL2L2 in Non Small Cell Lung Cancer	Yang Tian
17:15 - 17:45	TSANZ Young Investigator - Identification of MMP Dysregulation in Early CF - Evidence Based Rationale For Anti-Protease Therapy	Luke Garratt



# DAY 4 SUNDAY, 16<sup>TH</sup> NOVEMBER

## SESSION TIME: 08:00 - 10:00

**Room:** Nusa Dua 2

**Session:** Respiratory Organization Workshop

**Session Chairs:** Alexander Ginting, Erlina Burhan, Menaldi Rasmin

**Target Audience:** Pulmonologists, clinicians, basic and clinical researchers, registered nurses and respiratory technicians, fellows and residents in training, medical students, patients, caregivers, patient organizations, and persons involved in decision making

8:00 - 8:30	Anti-tobacco Movement: Where are We	Wanita Indonesia Tanpa Tembakau (WITT - Indonesian Woman Against Tobacco)
8:30 - 9:00	Patient Organization and Society Collaboration in Philippines	Teresita de Guia
9:00 - 9:30	Community Involvement in Chronic Respiratory Disease Care	D. Behera
9:30 - 10:00	Collaboration of Organizational and Professional Society for Better Respiratory Care: Australian Experiences	Christine Jenkins
10:00 - 10:30	Discussion	All
10:30 - 11:00	Building Partnership in Asthma Management: Experiences from Indonesian Asthma Foundation	Indonesian Asthma Foundation
11:00 - 11:30	Public-Private Mix in TB Management: Indonesian Experiences	Erlina Burhan
11:30 - 12:00	Emerging and Sustainable Collaboration between Patients/Professional Organizations in Japan	Yoshinosuke Fukuchi

**Room:** Nusa Dua 3

**Session:** AS22, Assembly of Critical Care: Hot Issues in ARDS

**Session Chairs:** Yoshiki Ishii, Prasenhadi Prasenhadi

**Target Audience:** Pulmonologists, clinicians, basic and clinical researchers, registered nurse and respiratory technicians, fellows and residents in training, and medical students

8:00 - 8:30	Subphenotype in ARDS and Biomarkers	Chian Min Loo
8:30 - 9:00	Stem-cell for ARDS: A Dream or Reality?	Sang-Bum Hong
9:00 - 9:30	Is Acute Exacerbation of Pulmonary Fibrosis ARDS	Yoshiki Ishii
9:30 - 10:00	Noninvasive Ventilation: The Horizon is Expanding	Nicholas Hill

**Room:** Nusa Dua 4

**Session:** AS21, Assembly of Tuberculosis: ISTC to Ensure Quality of Care

**Session Chairs:** Charles Yu, Reviono

**Target Audience:** Pulmonologists, clinicians, basic and clinical researchers, registered nurse and respiratory technicians, fellows and residents in training, and medical students

8:00 - 8:30	International Standard of TB Care	Philip Hopewell
8:30 - 9:00	TB in Asia Pacific: The Challenges	Charles Yu
9:00 - 9:30	Second line Anti TB drugs: Mechanism of Action and Rational for Use	Chen Yuan Chiang
9:30 - 10:00	Evolution and transmission of drug-resistant TB	Francis Drobniowski

**Room:** Uluwatu 1

**Session:** Oral Session 21: Lung Cancer

**Session Chairs:** Behera, Abdulaziz Aljohani

8:00 - 8:10	Diagnostic Time for Lung Cancer Patients and the Role of a Serum Based Biomarker Panel in the Early Diagnosis for a Cohort of High-Risk Patients (O-J-015)	Dawei Yang
8:10 - 8:20	Palladium (II)-Saccharinate Complex of Terpyridine Seems To Be a Promising Novel Compound for the Treatment of Lung Cancer In Vitro (O-J-016)	Engin Ulukaya
8:20 - 8:30	High-dose Cisplatin Exposure Increased Autophagy and Caused Cisplatin Resistance in Human Lung Cancer A549 Cells (O-J-018)	Ke Wang
8:30 - 8:40	Genetic Analysis of Hypoxia Inducible Factor-2 Alpha in Lung Cancer (O-J-019)	Andika Chandra Putra
8:40 - 8:50	Oncological Emergencies Arising at Diagnosis of Lung Cancer in Our Hospital (O-J-020)	Masaomi Marukawa
8:50 - 9:00	Afatinib in Management of Non-Small Cell Lung Cancer (NSCLC): What is Evidence So Far? (O-J-021)	Luke Lin
9:00 - 9:10	Frequency of Epidermal Growth Factor Receptor Mutations in Squamous Cell Carcinoma of the Lung in Smokers and Never Smokers (O-J-022)	Chong-Kin Liam

# DAY 4 SUNDAY, 16<sup>TH</sup> NOVEMBER

## SESSION TIME: 08:00 - 10:00

**Room:** Uluwatu 5

**Session:** Oral Session 22: Others

**Session Chairs:** Jennifer Mendoza, Winariani

8:00 - 8:10	The Association of Oxygen Desaturation Index and Lipoprotein Phospholipase A2 Towards Coronary Artery Disease in Obstructive Sleep Apnea Male Subjects (O-K-002)	Allen Widysanto
8:10 - 8:20	The Correlation Analysis Between Smoking and the Distal Lower Limb Arterial Abnormality as Detected by IRTI and CPUS in a Double Blind Controlled Clinical Study (O-K-003)	Fangge Deng
8:20 - 8:30	Long-term Results of Smoking Cessation and Factors Influencing the Success of Smoking Cessation (O-K-004)	Nurgül Bozkurt
8:30 - 8:40	Syringo-Pleural Shunt After Fifteen Years- A Rare Cause of Recurrent Pleural Effusion (O-K-005)	Prakash K Ashish
8:40 - 8:50	Assessment Of Knowledge, Accessibility and Utilization of Palliative Care Services Among Adult Cancer Patients at Tikur Anbesa Specialized Hospital, Addis Ababa, Ethiopia, 2014 (O-K-007)	Serawit Lakew Chillo

**Room:** Uluwatu 5

**Session:** Oral Session 24: Respiratory Structure and Function

**Session Chairs:** Yasutaka Nakano, Fachrial Harahap

9.00 - 9.10	The Role of Nitric Oxide in Tracheobronchial Ciliary Motility (O-P-001)	Takashi Kido
9.10 - 9.20	Age as the Main Predictor in Correlating Blood Pressure and Lung Function in Malaysian Population (O-P-002)	Nurul Yaqeen Mohd Esa
9.20 - 9.30	The Relationship Between Collagen Fiber Orientation and Anisotropy in Mechanical Property on the Human Lung (O-P-004)	Koichi Tomoda
9.30 - 9:40	Acute Effects of Hyperbaric Oxygen Therapy on Respiratory Rate and Basic Vital Functions (O-P-005)	Erdinc Ercan
9.40 - 9.50	Lung Function Among Early Adolescents Delivered Term with Low Birth Weight (O-L-001)	Joje B. Undar

**Room:** Uluwatu 7

**Session:** Oral Session 23: Respiratory Infections (Non-tuberculosis)

**Session Chairs:** Kwang Ha Yoo, Sardikin Giriputro

8:00 - 8:10	Antibiotic Use for Respiratory Tract Infections in Children (O-N-012)	Pramil Tiwari
8:10 - 8:20	Heart Rate Variability as a Tool to Assess the Severity of Community-Acquired Pneumonia in Hospitalized Patients: Preliminary Results (O-N-013)	Hean Ooi
8:20 - 8:30	A control of Respiratory Tract Infection in Adult Patients After Cardiac Surgery in Post Surgical ICU: A Study of 300 Patient Randomly and Co Relate the Patient Respiratory Culture with Environment and OT Culture. (O-N-014)	Rajeshkumar M Thosani
8:30 - 8:40	Nontuberculous Mycobacteria Lung Infection in Bronchiectasis in China: Prevalence and Clinical Characteristics (O-N-015)	Xu Jin-Fu



# DAY 4

SUNDAY, 16<sup>TH</sup> NOVEMBER  
SESSION TIME: 08:00 - 10:00

**Room:** Uluwatu 7

**Session:** Oral Session 25: Respiratory Infections (Non-tuberculosis) Assembly

**Session Chairs:** Xu Jin-Fu, Erlina Burhan

9.00 - 9.10	Association Between Interleukin-6 Serum Level with Treatment Failure of Community Acquired Pneumonia Patients with Comorbid COPD and Heart Failure (O-N-016)	Catur Elvi Purnamawati
9.10 - 9.20	Investigation of the Molecular Regulating Effect of Promoter-Associated Spacer Sequences on CTX-M Expression (O-N-017)	Lin Liu
9.20 - 9.30	Community-Acquired Pneumonias in Different Age Groups on Data of the Far East of Russia (O-N-018)	Martynova Alina V
9.30 - 9:40	Characterisation of Human Metapneumovirus Infection of Primary Human Airway Cells (O-N-019)	Philip Bardin

# DAY 4 SUNDAY, 16<sup>TH</sup> NOVEMBER

## SESSION TIME: 10:00 - 12:00

**Room:** Nusa Dua 3

**Session:** AS24, Assembly of Asthma: What is in the Pipelines in Asthma Therapy?

**Session Chairs:** Christopher KW Lai, Suradi

**Target Audience:** Pulmonologists, clinicians, basic and clinical researchers, registered nurses and respiratory technicians, fellows and residents in training, medical students and pharmacists

10:00 - 10:30	Asthma in Asia Pacific: The Burden	Christopher KW Lai
10:30 - 11:00	LABA, LAMA and Others Bronchodilators in Asthma Treatments	Teresita de Guia
11:00 - 11:30	Place of Biological Therapies	Gary Anderson
11:30 - 12:00	Whats is in the Pipeline in Asthma Therapy?	Christine Jenkins

**Room:** Nusa Dua 4

**Session:** AS23, Assembly of Respiratory Infection (Non-Tuberculosis): Viral Pneumonia

**Session Chairs:** Jasper Chan, Priyanti Soepandi

**Target Audience:** Pulmonologists, clinicians, basic and clinical researchers, registered nurse and respiratory technicians, fellows and residents in training, and medical students

10:00 - 10:30	SARS outbreak: What We Have Learned	Jasper Chan
10:30 - 11:00	H5N1 Pneumonia in Indonesia: Current Position and Strategies	Tjandra Yoga Aditama
11:00 - 11:30	MERS in Saudi Arabia	Abdullah Abdul Rahman Al Shimemeri
11:30 - 12:00	Respiratory Viral Outbreak: Future Ahead	Jasper Chan

# DAY 4 SUNDAY, 16<sup>TH</sup> NOVEMBER

## SESSION TIME: 10:00 - 12:00

**Room:** Uluwatu 1

**Session:** Oral Session 28: Clinical Allergy and Immunology

**Session Chairs:** Gary Anderson, Dianiaty Sutoyo

11:00 - 11:10	The Dynamic Change of Inflammatory Cytokines Treated by Specific Immunity and the Analysis of the Dynamic Change of House Dust Mites' Components-IgE&IgG4 (O-D-002)	Guangqiao Zeng
11:10 - 11:20	Tobacco Smoke Exposure Alters Immune Functions Mediated with DNA Damage in Alveolar Macrophage (AM) (O-D-003)	Minoru Takeuchi
11:20 - 11:30	FeNO Could Be an Effective Predictor for Eosinophilic Inflammation in Young Adult Patients with Allergic Rhinitis (O-D-004)	Baojuan Liu
11:30 - 11:40	Induced Pluripotent Stem Cells Reduces Neutrophils Chemotaxis via Activating GRK2 in Endotoxin-Induced Acute Lung Injury (O-D-005)	Vincent Yi-Fong Su
11:40 - 11:50	Effects of Familial Inheritance on Regulation of Th1/Th2 Cytokine Expression (O-D-006)	Jiaying Luo

**Room:** Uluwatu 5

**Session:** Oral Session 26: Interstitial Lung Disease

**Session Chairs:** Dianiaty K Sutoyo, Ryushi Tazawa

10:00 - 10:10	Case Analysis of Interstitial Lung Diseases Induced By Breast Cancer Chemotherapy (O-I-011)	Hiroshi Nakaoka
10:10 - 10:20	Fluorofenidone Attenuates BLM-Induced Pulmonary Inflammation and Fibrosis In Mice via Inhibiting the Activation of NALP3 Inflammasome and IL-1 $\beta$ /IL-1R1/MyD88 Signaling Pathway (O-I-012)	Meng Jie
10:20 - 10:30	Preparation of Lung-Targeting, Emodin-Loaded PLGA Microspheres and their Properties (O-I-013)	Xiaohong Chen
10:30 - 10:40	Improvement of Autoimmune Pulmonary Alveolar Proteinosis After Infectious Episodes (O-I-014)	Takehiko Kobayashi
10:40 - 10:50	Sarcoidosis- Indian Perspective (O-I-015)	Mahavir Modi
10:50 - 11:00	Impact of Pulse I.V. Cyclophosphamide on Scleroderma Related Interstitial Lung Disease: An Indian Experience (O-I-016)	Suman Paul

**Room:** Uluwatu 7

**Session:** Oral Session 27: Critical Care

**Session Chair:** Yoshiki Ishii, Wahjuani

10:00 - 10:10	Outcomes of Early Sedation versus No Sedation among Mechanically Ventilated Critically Ill Patients: A Prospective Comparative Study (O-G-006)	Yoshiko Ikeda-Maquiling
10:10 - 10:20	Accuracy of capillary versus venous blood glucose estimation in critically ill adult patients: an observational study (O-G-007)	Rahul Kumar Sharma
10:20 - 10:30	Efficacy of Bi-level Positive Pressure Ventilation (BiPAP) over invasive ventilation in Acute Exacerbation of COPD with Type 2 Respiratory Failure patients in a critical care setting (O-G-009)	Mamunur Rashid
10:30 - 10:40	Initial End-Tidal Carbon Dioxide as a Prognostic Indicator for Inpatient PEA Arrest (O-G-005)	Rebecca E Sell







# ABSTRACTS

DAY 1 - THURSDAY, 13<sup>TH</sup> NOVEMBER

DAY 2 - FRIDAY, 14<sup>TH</sup> NOVEMBER

DAY 3 - SATURDAY, 15<sup>TH</sup> NOVEMBER

DAY 4 - SUNDAY, 16<sup>TH</sup> NOVEMBER



# DAY 1 THURSDAY, 13<sup>TH</sup> NOVEMBER

## Sleep-Related Breathing Disorders in Indonesia

**Agus Dwi Susanto**

Indonesia

Sleep related breathing disorders (SRBD) data in Indonesia is still limited. Obstructive sleep apnea is the most SRBD that studied in Indonesia. From several study showed that OSA will be important health problems in Indonesia population for the future. OSA prevalence from several study were varied from 19.8% until 25% using Berlin questionnaire and 7.35% until 55.1% using polysomnography test. Sihombing (2008) reported that 55.1% taxi drivers with snoring proven OSA using portable polysomnography test. Wiadnyana and Susanto (2010) study in taxi drivers at Jakarta using Berlin questionnaire found 25% taxi drivers had high risk for OSA. Study in Children by Supriyatno (2010) found OSAS prevalence in children 10-12 y.o with obesity was 38.2%. Susanto et.al (2013) found 52.5% taxi drivers with overweight and obesity proven OSA base on clinical symptoms and polysomnography test. Susanto et.al (2014) found 21.7% police officers at Tangerang and East Jakarta have suspected OSA. Study in Department of Pulmonology and Respiratory Medicine Faculty of Medicine University of Indonesia by Astuti et.al in asthma patients found OSA prevalence was 19.8% using Berlin questionnaire and 9.8% with polysomnography test. Another study in COPD patients by Ratih et.al found 25% had high risk for OSA and 7.35% proven OSA by polysomnography test. From that all studies, the most OSA risk factors in Indonesia population were increased of body mass index, neck circumference and snoring history in the family.



## Sleep Apnea in Children: Diagnosis and Management

Carole Marcus

USA

The obstructive sleep apnea syndrome (OSAS) is common, affecting 2-4% of otherwise healthy children, and a much higher percentage of children with risk factors such as prematurity, Down syndrome, craniofacial anomalies or neuromuscular disease. OSAS result from a combination of anatomic and neuromotor factors. The American Academy of Pediatrics issued revised clinical practice guidelines for the diagnosis and management of childhood OSAS in 2012. These evidence-based guidelines were based on analysis of more than 3,000 articles. The following recommendations were made: (1) All children/adolescents should be screened for snoring; (2) Polysomnography should be performed in children/adolescents with snoring and symptoms or signs of OSAS; if polysomnography is not available, then alternative diagnostic tests or referral to a specialist for more extensive evaluation may be considered; (3) Adenotonsillectomy is recommended as the first-line treatment for patients with adenotonsillar hypertrophy; (4) High-risk patients should be monitored as inpatients postoperatively; (5) Patients should be reevaluated postoperatively to determine whether further treatment is required. Objective testing should be performed in patients who are high risk or have persistent symptoms/signs of OSAS following therapy; (6) Continuous positive airway pressure (CPAP) is recommended as treatment if adenotonsillectomy is not performed or if OSAS persists postoperatively; (7) Weight loss is recommended in addition to other therapy in patients who are overweight or obese; (8) Intranasal corticosteroids are an option for children with mild OSAS in whom adenotonsillectomy is contraindicated or for mild postoperative OSAS. Areas for further research will also be discussed.



# DAY 1 THURSDAY, 13<sup>TH</sup> NOVEMBER

## Second Line Anti-TB Drugs: Mechanism of Action and Rational Use

Chen-Yuan Chiang

Taiwan

Based on findings of an individual patient data meta-analysis of 9153 multidrug-resistant tuberculosis (MDR-TB) patients, World Health Organization recommends that in the treatment of patients with MDR-TB, four second-line anti-tuberculosis drugs likely to be effective, as well as pyrazinamide, should be included in the intensive phase; regimens should include at least pyrazinamide, a fluoroquinolone (FQ), a parenteral agent, ethionamide (or prothionamide), and either cycloserine or PAS (p-aminosalicylic acid) if cycloserine cannot be used. The core drug in the treatment of MDR-TB is FQ. The newer FQs, sparfloxacin, gatifloxacin, and moxifloxacin, have lower minimum inhibitory concentrations (MICs) against *M. tuberculosis* than levofloxacin, ciprofloxacin, and ofloxacin, and their activity is concentration dependent. An animal study showed that moxifloxacin 400mg and levofloxacin 1000mg have comparable activities during the initial 2 months of treatment, and that moxifloxacin had greater activity during the continuation phase of treatment. The binding target of FQs in *M. tuberculosis* is DNA gyrase, consisting of two A and two B subunits encoded by the *gyrA* and *gyrB* genes, respectively. Missense mutations within the quinolone resistance-determining region (QRDR) have been identified as the primary mechanism conferring fluoroquinolone resistance. Studies reported that high dose (800mg) moxifloxacin achieved excellent *M. tuberculosis* microbial kill and suppressed drug resistance. An observational study using high dose gatifloxacin, clofazimine, ethambutol pyrazinamide, prothionamide, kanamycin and high-dose isoniazid for at least 4 months till sputum conversion followed by high dose gatifloxacin, clofazimine, ethambutol and pyrazinamide for 5 months achieved >85% relapse-free cure rate in Bangladesh.

## Challenge of Drug Resistant Tuberculosis in Asia and Western Pacific

Chen-Yuan Chiang

Taiwan

World Health Organization estimated that globally, 3.6% (95% CI: 2.1–5.1%) of new TB cases and 20.2% (95%CI: 13.3–27.2%) of previously treated cases have multidrug-resistant tuberculosis (MDR-TB). On average, an estimated 9.6% (95% CI: 8.1%–11%) of MDR-TB cases have extensively drug-resistant tuberculosis (XDR-TB). The estimated number of incident MDR-TB cases globally in 2012 was 450 000 (range: 300 000–600 000), and that among patients with pulmonary TB notified in 2012 was 300 000 (range: 220 000–380 000). The burden of MDR-TB is high in South East Asia and Western Pacific Regions, especially in India, China, the Philippines, Indonesia, Myanmar, and Bangladesh. The proportion of TB cases with drug susceptibility testing (DST) for first line anti-tuberculosis drugs was low in South East Asia and Western Pacific Regions. In 2012, the proportion of retreatment TB cases with DST result was 7% in Bangladesh, 12% in China, 10% in Indonesia, 9% in the Philippines. Globally, 83 715 cases of MDR-TB were notified to WHO in 2012, represented 28% of the 300 000 pulmonary TB patients estimated to have MDR-TB in 2012. Outcome of MDR-TB was disappointingly low in both South East Asia and Western Pacific Regions. In both regions, the proportion of MDR-TB patients in the 2010 cohort who successfully completed treatment was <50%, while about 30% of cases were reported as lost to follow-up or had no outcome information. Intensified regional and national efforts to detect cases of MDR-TB and to improve treatment outcomes are urgently required.

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## Neuroendocrine Tumours of the Lung

**Chong-Kin Liam**

Malaysia

Neuroendocrine tumours (NETs) of the lung include a spectrum from low-grade typical carcinoid (TC) and intermediate-grade atypical carcinoid (AC) to high-grade large cell neuroendocrine carcinoma (LCNEC) and small cell lung carcinoma (SCLC). LCNEC and SCLC are found in heavy-smoking, older patients, whereas smoking is not strongly associated with carcinoid tumours. It is difficult to diagnose AC and LCNEC in small biopsies or cytology and a definitive diagnosis usually requires a surgical specimen. SCLC, characterised by a rapid progression of symptoms and a bulky central and/or mediastinal tumour, can usually be reliably diagnosed by limited biopsy.

Surgery is the primary treatment for TC and AC. Up to 64% of patients with AC present with lymph node metastases, and 5-year survival ranges from 61% to 88%. In contrast, lymph node metastases are present in fewer than 15% of cases of TC, and 5-year survival exceeds 90%. The role of targeted therapy for TC and AT remains incompletely defined, with data from relatively few clinical trials to help guide clinical decision making. In patients with LCNEC, locally advanced or metastatic stages are usual at presentation and surgery is possible in less than a third of patients. Because of the rarity of LCNEC, treatment recommendations are not based on clinical trials, but are extrapolated from the approach to patients with NSCLC and SCLC as well as literature for LCNEC which is primarily retrospective in nature. Treatment of SCLC is usually chemotherapy.

Lung NETs have been underrepresented in clinical trials of NET treatments. In recent years, re-sults specific to lung NETs have been reported only in a phase 2 retrospective study of the dacarbazine derivative temozolomide and the phase 3 RAD001 in Advanced Neuroendocrine Tumors Trial 2 (RADIANT-2) which evaluated the impact of combination therapy with the oral mammalian target of rapamycin (mTOR) inhibitor everolimus and the somatostatin analogue octreotide LAR in patients with advanced NET and carcinoid symptoms. The first large phase 2 prospective, randomised 3-arm trial to evaluate the efficacy and safety of pasireotide LAR alone or everolimus alone or in combination in patients with lung or thymus neuroendocrine carcinoma (LUNA Trial) is ongoing.

## Community Acquired Pneumonia (CAP) in TB Endemic Countries

Dr. V. K. Vijayan

India

Community acquired pneumonias (CAP) are common infectious diseases occurring worldwide with considerable morbidity and mortality. Last century witnessed considerable improvements in public health infrastructure, introduction of novel diagnostic methods, discovery of antibiotics and development of vaccines against influenza and *Streptococcus pneumoniae* and all these developments had contributed to better diagnosis and treatment of community acquired pneumonias especially in industrially advanced countries. However, the emergence of human immunodeficiency virus and other immunodeficiency states have complicated the diagnosis and management of community acquired pneumonias. The diagnosis and treatment are further complicated in countries where tuberculosis is endemic. The increasing incidence of fungal pneumonias which are difficult to diagnose and treat is another challenge. The common causative agents of pneumonia in the community are *Streptococcus pneumoniae*, *Haemophilus influenzae*, *Mycoplasma pneumoniae*, *Legionella spp.*, *Chlamydia pneumoniae* and respiratory viruses (influenza A and B, adenovirus, respiratory syncytial virus and parainfluenza). The bacteriological profile of CAP is not the same across various countries and within the same country. This is due to differences in the frequency of use of antibiotics, environmental pollution, awareness of the disease and life expectancy. The most common causative pathogen reported from Europe, the United States and the United Kingdom is *S. pneumoniae*. However in Singapore, it is *K. pneumoniae*. In a prospective study from Mumbai, *S. pneumoniae* was the leading cause of CAP in 22% and atypical organisms were identified in 19% of patients. Importantly, *Mycobacterium tuberculosis* was isolated in 7% of patients emphasizing the need for including diagnostic tests for tuberculosis in suspected cases of CAP from countries with high prevalence of tuberculosis. Blood cultures, sputum Gram-stain and culture are the most common tests used to identify the etiology of CAP. In addition to this, serological tests are also performed. Resistance to  $\beta$ -lactams and to macrolides is low in India. Chest radiograph is required to confirm the diagnosis of pneumonia, to detect associated lung disease, to assess severity and associated complications and to obtain baseline to assess the response to treatment. Pneumonia mortality risk can be predicted using either Pneumonia Severity Index or CURB 65 [confusion (C), blood urea (U), respiratory rate (R), blood pressure (B) and age  $\geq 65$  years (A)]. Fluoroquinolones are recommended as initial empirical therapy in CAP in some countries, as these drugs are resistant to streptococci in only about less than 3% of cases and have excellent activity against atypical organisms. Fluoroquinolones have also action against *M. tuberculosis* and are recommended for the treatment of multidrug-resistant tuberculosis and for shortening the duration of anti-TB treatment. Initiation of empirical therapy of CAP with fluoroquinolones is therefore, not recommended in countries with high prevalence of tuberculosis as this may have serious consequences of delayed initiation of anti-TB treatment and development of resistance to fluoroquinolone in case the CAP happens to be due to *M. tuberculosis*. In countries



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with high TB prevalence, amoxicillin is the preferred drug for treatment of a patient with CAP with low severity and a  $\beta$ -lactam plus a macrolide for inpatients with CAP and with moderate severity. Intravenous  $\beta$ -lactam and a macrolide are required for patients with high severity. Pneumococcal infections can be prevented with pneumococcal pneumonia vaccines and influenza-related respiratory illnesses can be prevented with influenza vaccinations. Consumption of tobacco is an important risk factor for CAP and smoking cessation is important to reduce the burden of CAP.

## **OSA and Pathophysiology Including Sustained and Intermittent Hypoxia**

**Geraldo Lorenzi-Filho**

Brazil

Obstructive sleep apnea (OSA) is characterized by recurrent upper airway obstruction during sleep. The primary mechanisms linking OSA with poor cardiovascular outcome occur during sleep and are: 1. Mechanical: Generation of negative intrathoracic pressure swings during the futile efforts to breath against an occluded airway. The negative intrathoracic pressure in conjunction with acute (and frequently also sustained) high blood pressure are responsible for increased after load and heart remodeling. The vibration caused by snoring transmitted to the neck may also cause accelerated atherosclerosis at the level of the carotid arteries. 2. Neural: Arousals from sleep, that occur at the end of each obstructive events triggers sympathetic activity. Obstructive events also typically deprives patients from slow wave sleep. Deprivation of slow wave sleep caused by acoustic stimuli triggers metabolic dysfunction in normal subjects; 3. Chemical: Intermittent asphyxia, as characterized by episodes of hypoxia and hypercapnia. These primary mechanisms that occur during sleep triggers a cascade of intermediate pathways that are potentially deleterious to the cardiovascular system. The best studied and probably the most important intermediate mechanism is sympathetic activation that occurs during each episode of airway obstruction. Sympathetic over activation occurs not only during sleep but carry overs during the 24 hour period. In addition OSA may trigger several other mechanisms that are harmful to the cardiovascular system and includes oxidative stress, inflammation, insulin resistance, lipid dysfunction, endothelial dysfunction and accelerated atherosclerosis. There is evidence from animal models that intermittent hypoxia is a key mechanism linking OSA with poor cardiovascular outcome. All these mechanisms help to explain why OSA is associated with increased risk of cardiovascular death due to stroke and myocardial infarction.

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## **GOLD Documents: Current Issues?**

**Jørgen Vestbo**

Denmark

The current GOLD strategy documents builds on the original contents dating back to 2001 and the 2011 revision. A clinical diagnosis of COPD requires a relevant exposure, symptoms and airflow limitation. The 2015 update has slightly expanded the GOLD "square" denoting a symptoms dimension and a risk dimension.

Symptoms are preferably assessed using a comprehensive and systematic assessment, although a simple assessment of breathlessness using the mMRC questionnaire is also possible. Risk is still best assessed using both history of exacerbations (including hospital admissions) and level of FEV1. Recent studies of population samples and patient cohorts have documented that in addition to being a guide to patient management, The A-D categorisation also has predictive value regarding both exacerbations and mortality.

GOLD still emphasises the need for proper assessment of comorbidities, not least cardiovascular comorbidities, and recent studies have shown that in particular Group B patients are at high risk of experiencing comorbidities, leading to a higher than expected mortality in this group.

Preventive measures are underlined, including smoking cessation and reduction of exposure to in- and outdoor pollution.

For non-pharmacological management, the value of both pulmonary rehabilitation and physical activity outside rehabilitation programs are highlighted. For pharmacological management, bronchodilators are central according to GOLD and focus is increasingly on long-acting inhaled bronchodilators. In patients at high risk of exacerbations, these can be combined with anti-inflammatory drugs. GOLD does not recommend use of macrolides for exacerbation prevention. The importance of proper follow-up of all treatments is underlined.

## Thoracic CT Pattern in Lung Cancer: Correlation of CT and Pathological Diagnosis

**Kazuma Kishi**

Japan

High-resolution CT (HRCT) is the main diagnostic tool for the diagnosis of lung cancer. HRCT images are correlated well with pathological findings. In 2011, the International Association for the Study of Lung Cancer, the American Thoracic Society, and the European Respiratory Society proposed a new classification system for lung adenocarcinoma. The new classification system is based on HRCT-pathologic correlation studies, and useful for predicting adenocarcinoma histologic subtype, patient prognosis and management. For example, ground-glass opacity extent within a pulmonary nodule on HRCT can be correlated with the extent of lepidic tumor growth on pathology. On the contrary, the size of the solid component on HRCT is frequently related with invasive component on pathology in lung adenocarcinomas manifesting as ground-glass nodules. In this presentation, I would like to show various HRCT findings of lung cancer by histologic subtypes, especially focus on ground-glass opacity neoplastic lung nodules.



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## CT Assessment of Pulmonary Nodules

**Kazuma Kishi**

Japan

The widespread use of multi-detector row CT in daily clinical practice detects a lot of incidental pulmonary nodules. The differential diagnosis is broad, ranging benign granuloma to malignancy. What is the appropriate management of these indeterminate nodules? The goal of nodule evaluation is to expedite resection of potentially curable lung cancer and to minimize resection of benign nodules. To achieve this goal, not only detailed analyses of morphologic characteristics on high-resolution CT but also the identification of clinical risk factors are important. In addition, it is helpful to use recently published guidelines for the management of pulmonary nodules. In this presentation, I would like to talk about a practical approach to the diagnosis and management of pulmonary nodules with reference to nodule guidelines by the Fleischner Society and American College of Chest Physicians.

## Overview: Sleep-Related Breathing Disorders and Their Treatment

**Kazuo Chin**

Japan

Almost 60 sleep disorders are described in the International Classification of Sleep Disorders (ICSD-3, 2014). Included in ICSD 3 are 17 sleep-related breathing disorders (SRBDs) and 2 isolated symptoms and normal variants (snoring and catathrenia). SRBDs are divided into 4 groups: 1) obstructive sleep disorders, 2) central sleep apnea syndromes, 3) sleep-related hypoventilation syndromes, and 4) sleep-related hypoxemia disorders. Obstructive sleep disorders include adult and pediatric obstructive sleep apnea. Central sleep apnea syndromes include Cheyne-Stokes breathing (CSB), central apnea due to a medical disorder without CSB, central sleep apnea due to high altitude periodic breathing, central sleep apnea due to a medication or substance, primary central sleep apnea, primary central sleep apnea of infancy, primary central sleep apnea of prematurity, and treatment-emergent central sleep apnea. Sleep-related hypoventilation disorders include obesity hypoventilation syndrome, congenital central alveolar hypoventilation syndrome, late-onset central hypoventilation with hypothalamic dysfunction, idiopathic central alveolar hypoventilation, sleep-related hypoventilation due to a medication or substance, and sleep-related hypoventilation due to a medical disorder. There are several treatments for SRBDs such as body weight reduction, CPAP, NPPV, adaptive servo ventilation (ASV), oxygen, surgical therapies, tracheostomy with a ventilator, upper airway stimulation, etc. Patients with SRBDs very often have several comorbidities. Therefore, to administer appropriate therapy for SRBDs, it is important to know the pathophysiology, clinical characteristics, and available treatments and management strategies for SRBDs in adults and children and how to diagnose specific sleep disorders. The participants in this P-G course should know about SRBDs overall and management strategies presently available.

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## Evaluation of Lung Function in Chronic Disease

**Le Thi Tuyet Lan**

Vietnam

Based on the ATS/ERS Interpretation Algorithm of PFTs, each step will be explained and illustrated by the result of the correlative tests.

Measurement of ventilatory function with spirometry, body plethysmograph and measurement of gas exchange by diffusing capacity are used.

Identifying the presence or absence of obstruction, confirming the restriction with lung volumes will help to differentiate the obstruction, restriction or mixed patterns.

Some ways of classification of severity of airflow limitation and lung volumes impairment are presented.

DLCO interpretation and severity classification of diffusing capacity are explained.

The combination of the lung volumes, airflows and diffusing capacity results permits the differentiating 6 groups of lung functions :

- 1.Normal.
- 2.Pulmonary vascular disorders
- 3.Chest wall and neuromuscular disorders.
- 4.Interstitial lung diseases and pneumonitis.
- 5.Asthma and chronic bronchitis and
- 6.Emphysema

Other factors such as performance, central airways disorders... must be considered.

Some issues of the ATS/ERS interpretation algorithm will be pointed out.

The conclusion will emphasize the fact that PFTs indicate only the syndrome or the assumption of one disease. The combination of careful patient history, clinical examination, imaging and bronchoscopy are often required to make an exact diagnosis.

## Methods of Sleep Testing: Polysomnography and Portable Monitoring

**Michelle Cheong**

Hong Kong

Polysomnography is a sleep study, a diagnostic tool in sleep medicine used to identify sleep related breathing disorder, narcolepsy, periodic limb movement, hypersomnia, parasomnias and REM behavior disorder. A polysomnogram recorded a number of channels wire attachments to patients and wires for each channel of record data lead from the patient and converge to a headbox which is connected to a sleep monitoring recording system. During sleep, the computer monitoring can display all attached channels and recording all attached channels the whole night. After completing the whole night sleep recording, the recorded data will analyzes any breathing irregularities or abnormal cardiac rhythm, leg movement and arousals, sleep stages and sleep efficiency including oxygen saturation during sleep. Polysomnography is one method of sleep testing used in most hospital and sleep laboratory. Besides polysomnography, portable monitoring sleep systems currently popular because of their recording machine is smaller, convenient, patient can used this machine to sleep at home and they can hook sensors by themselves. Portable monitoring sleep test in the current market include sleep monitoring with brain activity signals, sensors like effort bands to test chest movement during sleep, airflow to monitor breathing and also saturation during sleep. The data can be wired or wirelessly transferred for remote monitoring. Sleep monitoring based on movement is current popular in the market such as fitbit, sleep cycle alarm, sleep tracker, jawbone and wakemate etc. Watch-pat is also portable device monitors changes in peripheral arterial tone and activity, as well as in blood oxygen saturation levels to detect sleep apnea events.

This presentation will detail sleep technology in sleep lab setting and why portable monitoring system is popular in current market.



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## Monitoring of Mechanical Ventilation

Olu Albert, MPH, RRT

USA

Respiratory mechanics (lung/chest wall compliance, resistance, and time constants) has always been the mainstay of mechanical ventilation monitoring. This 30-minute lecture will discuss key definitions using airway graphics to explain airway pressures, volumes, compliance (dynamic and static) and time constants in patient-ventilator interactions. The interconnectedness between the “measured parameters” (Flow, Pressure, Time) and “calculated parameters” (Volume, Compliance and Resistance); and its clinical significance as it relates to patient-ventilator interaction will be discussed. Lastly, the pressure-volume (PV) curves will be used to explain the concept of lower and upper inflection points as it relates to optimal PEEP, over-distention and decrease lung compliance.

## Patient-Oriented PFTs to Assist Clinical Decision-Making

Paul Enright

USA

This workshop is for pulmonary specialist clinicians who want to learn how to more effectively utilize pulmonary function tests to help with the differential diagnosis or to provide objective evidence of improvement or worsening during follow-up exams of patients who have been previously diagnosed. Everyone will have a clicker to respond to questions about which test to order, how you would interpret the results, and how you would change the treatment based on the PFT results. After each 30 minute presentation of cases, everyone will move to one of five tables filled with PFT instruments for a 30 minute demonstration of testing. You can test yourself. Then back to another 30 minute presentation of more cases, alternating throughout the day.

1a. Case presentations: adult smokers (COPD screening) one case with normal PEF (smoking cessation but no need for spirometry), and one case with low PEF, followed by pre- and post-BD spirometry showing severe CAO (FEV1 of 40% pred) with a follow-up exam after one month of tiotropium therapy.

1b. Hands-on pocket spirometry and diagnostic spirometry

2a. Case presentations: patients with episodic wheezing; a 6 year old child with normal spirometry but high eNO; a young adult with obstruction and a large BD response, follow-up after 1 month of moderate dose ICS+LABA; an older adult former smoker with obstruction but small BD response, 1 month follow-up after high dose ICS+LABA; a professional adult patient closet smoker with difficult-to-control asthma, using home monitoring during follow-up).

2b. Hands-on spirometry, PEF meter, and eNO testing

3a. Case presentations of differential diagnosis of patients with slow onset dyspnea and a relatively normal chest x-ray: a patient with normal PFTs (but severe anemia), a patient with previously undiagnosed asthma, a former smoker with emphysema phenotype COPD, a patient with obesity and poor conditioning, a patient with heart failure.

3b. DLCO demonstration

4a. Case presentations: Evaluation of patients with abnormal chest x-ray; initial and 3 month follow-up exams; a young adult with cystic fibrosis, a middle aged adult with IPF or sarcoidosis, a patient with severe kyphoscoliosis (chest wall restriction), an elderly smoker with heart failure and mild COPD, a patient with infiltrates but normal PFTs

4b. Demonstration of 6MWT with pulse oximetry; hands-on DLCO

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5a. Case presentations of patients with a chronic cough: a teenager with exercise-induced asthma, a patient with allergic rhinosinusitis, a patient with gastro-esophageal reflux, a smoker with chronic bronchitis, a patient with eosinophilic bronchitis.

5b. Demonstration of inhalation challenge testing, and perhaps a demonstration of allergen skin testing

6a. Case presentations of unusual patients: a patient with stridor following prolonged intubation (UAO); a patient with a neuromuscular disease; a patient with asbestosis referred for a disability exam; a patient with lung cancer and severe COPD for a pneumonectomy evaluation

6b. Hands-on maximal respiratory pressure tests (MIP and MEP) and flow-volume loops

## Assessment of Exacerbations

Paul Jones

UK

Exacerbations in COPD drive lung function decline, cause poor health, trigger hospital admissions, cause deaths and increase health-care costs. Reducing the impact of exacerbations is a key outcome in COPD guidelines. Definitions of exacerbation differ widely across clinical studies and subjective assessment of symptoms lacks precision. The most widely used definitions have the following components: acute worsening, beyond normal day-to-day variation for 2-3 consecutive days, necessitating a change in treatment. Using this approach, the severity of the exacerbation is determined by the amount of health care utilization, but that will depend on systems of health care delivery. Development of standardised patient reported outcomes such as the EXACT daily diary now provide a more precise measurement of the frequency, severity and duration of exacerbations in clinical studies.

One of the new developments in the understanding of exacerbations is that, on average, patients have twice as many unreported exacerbations as those that are reported. Unreported exacerbations occur in less severe patients, although the reporting rate varies greatly by country. Unreported exacerbations show the same degree of worsening and medium term impact on health status as those that are reported (and therefore treated), but appear to recover more slowly than those that are reported.

Clinicians need to be alert to the possibility that their patients are having exacerbations that they don't report and for which they don't seek treatment. Patients need to be educated to understand that a 'chest infection' is an exacerbation that requires treatment.



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## Asthma-COPD Overlap Syndrome

**Peter Gibson**

Australia

Asthma and COPD are 2 common airway diseases that and co-exist and give rise to the asthma-COPD overlap syndrome(ACOS). This is recognised to arise in several different situations. The spectrum of ACOS includes long standing asthma that leads to incompletely reversible airflow obstruction, asthma and smoking leading to ACOS, COPD with late onset asthma, and COPD with eosinophilic bronchitis. Patients with ACOS experience greater morbidity than either disease alone. Biomarkers to recognise ACOS are under evaluation. Guideline approaches to asthma and COPD vary considerably and are based on evidence from randomised controlled trials. Since patients with ACOS are excluded from clinical trials, the evidence base for treatment recommendations in ACOS is limited. A clinically useful approach is to consider the specific disease components and direct treatment accordingly. This includes assessment and treatment in 4 domains , such as the airway component(which includes obstruction and inflammation), comorbidity, risk factors(such as smoking, nutrition, obesity), and behavioural issues. Preliminary evidence supports better outcomes when ACOS is managed in this way. Future developments will need to include clinical treatment trials in ACOS and studies of relevant mechanisms.

## **Flex-rigid and Rigid Thoracoscopy: When to Choose and the Drawback**

**Pyng Lee MD FCCP**

Singapore

Thoracoscopy, medical thoracoscopy, pleuroscopy and video-assisted thoracic surgery (VATS) are terms used interchangeably to describe a minimally invasive procedure that provides the physician a window to the pleural space. Historically rigid endoscopic instruments such as stainless steel trocars and telescopes are used. Following the advent of the flex-rigid pleuroscope similar in design and handling to the flexible bronchoscope and compatible with standard light source and video processor available in most bronchoscopy suites, flex-rigid thoracoscopy has made significant inroads as a diagnostic and therapeutic tool. The current debate should not focus on “who and where” to perform thoracoscopy but rather “when and how” to use rigid and flex-rigid instruments in different clinical scenarios.

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## Diagnostic and Therapeutic Thoracoscopy

**Pyng Lee MD FCCP**  
Singapore

Thoracoscopy provides the physician a window to the pleural space, biopsy of the parietal pleura under direct visual guidance, chest tube placement and pleurodesis for recurrent pleural effusions or pneumothoraces in selected patients. Since its inception thoracoscopy was used to lyse adhesions to achieve therapeutic pneumothorax in pulmonary tuberculosis. Following effective anti-tuberculous drugs, it fell into oblivion except for few centers in Europe. The procedure enjoyed resurgence when thoracic surgeons introduced the technique for minimally invasive surgery known as video-assisted thoracic surgery (VATS). VATS is performed under general anaesthesia with single lung ventilation while pleuroscopy is performed by the pulmonologist in an endoscopy suite using non-disposable rigid or flex-rigid instruments, local anaesthesia and conscious sedation. Pleuroscopy is useful for the diagnostic work up of pleural effusions while talc poudrage is an effective therapeutic option for patients with recurrent pneumothoraces and pleural effusions. Pleuroscopy has a place in interventional pulmonology and diagnostic and therapeutic indications will be discussed.

## **Pleural Disease: What Should We Know?**

**Richard W. Light, M.D.**

USA

The most common etiology for a pleural effusion worldwide is congestive heart failure. The effusions due to heart failure are usually bilateral, symmetrical and are not associated with chest pain or fever. They disappear when the heart failure is treated. If the patient is on diuretics, the pleural fluid may meet Light's criteria for an exudative effusion and the transudative nature can be verified by demonstrating a serum-pleural fluid gradient  $>3.1$ .

The presence of a malignant pleural effusion indicates that the patient cannot be cured by surgery. The diagnosis is most easily made with cytology. If the cytology is non diagnostic, image guided biopsy of the pleura or thoracoscopy are effective in making a diagnosis. If the patient is dyspneic and if the dyspnea is relieved with a therapeutic thoracentesis, efforts should be made to control the pleural fluid. The two most popular means of controlling the pleural fluid are the insertion of an indwelling pleural catheter or the injection of an agent into the pleural space to create a pleurodesis.

Parapneumonic effusions are those associated with a pneumonia. When a patient with a parapneumonic effusion is first evaluated, the pleural fluid should be sampled to ascertain whether therapy in addition to antibiotics is indicated. If the gram stain or culture is positive, if the pH is less than 7.20, if the effusion occupies more than 50% of the hemithorax, or if the glucose is less than 60 mg/dl, a chest tube should be inserted. If the pleural fluid is loculated, consideration should be given to the intrapleural administration of the combination of 5 mg DNase and 10 mg tPA.



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## Sleep Apnea and Heart Failure

**Takatoshi Kasai**

Japan

Sleep apnea is often observed in patients with heart failure (HF). Sleep apnea consists of two types: obstructive and central sleep apnea (OSA and CSA, respectively). OSA results from upper airway collapse, whereas CSA arises from attenuations in central respiratory drive. In patients with OSA, blood pressure is frequently elevated as a result of sympathetic nervous system overactivation. The generation of exaggerated negative intrathoracic pressure during obstructive apneas further increases left ventricular (LV) afterload, reduces cardiac output, and may cause the progression of HF. Intermittent hypoxia and post-apneic reoxygenation cause vascular endothelial damage and possibly atherosclerosis and consequently coronary artery disease and ischemic cardiomyopathy. CSA is also characterized by apnea, hypoxia, and increased sympathetic nervous activity and, when present in HF, is associated with increased risk of death. In patients with HF, abolition of coexisting OSA by continuous positive airway pressure (CPAP) improves LV function and may contribute to the improvement of long-term outcomes. Although treatment options of CSA vary compared with OSA treatment, CPAP and other forms of positive airway pressure therapy improve LV function and may be a promising adjunctive therapy for HF patients with CSA. In this session, pathophysiology and treatment option of sleep apnea including both OSA and CSA will be summarized.

## Useful Techniques in Airway Stenosis

Teruomi Miyazawa

Japan

Inoperable central airway stenosis due to a malignant tumor is a relatively common condition and may be life threatening. Because of the poor prognosis, palliative methods are needed to maintain airway patency. In patients with severe malignant airway stenosis, interventional bronchoscopy is considered as a method of maintaining airway patency.

Flow limitation during forced expiration is affected by the relationship between transmural pressure and the cross-sectional area of the airway. The wave-speed is dependent on the stiffness of the airway wall, and on the cross-sectional area itself. The flow-limiting segment (FLS) occurs originally when the cross-sectional area of the airway is narrowest. On the basis of wave-speed concepts of maximal expiratory flow limitation, stenting at the FLS improved expiratory flow limitation by increasing the cross-sectional area supporting the weakened airway wall, and relieving dyspnea.

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## Pathogenesis of COPD and Disease Phenotype of COPD

Tomoko Betsuyaku

Japan

Chronic inflammation and oxidative stress is a prominent feature of smokers' lungs and COPD. Cells lining terminal bronchioles are important source of antioxidants as well as inflammatory chemokines. Aging and cigarette smoke (CS) exposure are thus major risk factors for pathogenesis of COPD. Smoking cessation is regarded as an important strategy for prevention and treatment of COPD. However, little is known what makes the CS-induced injury chronic even after quitting smoking.

To address this question, we have investigated the cellular and molecular changes that occur in cells lining terminal bronchioles in response to CS. We have successfully utilized laser capture microdissection in combination with quantitative RT-PCR in order to quantify the cell-type specific mRNAs in animals in vivo. Our results showed that a variety of bronchiolar genes are affected by smoking and that the profile is dramatically changed by chronic exposure. We then tested whether the duration of CS exposure affects recovery from CS-induced inflammation after cessation of smoking. Our data help in dissecting the mechanisms by which the CS-induced injury become chronic even after smoking cessation, also suggests bronchiolar epithelium could be a target site to prevent them to be chronic.

## Lung Cancer Stem Cell

Toshiaki Kikuchi

Japan

The functional interplay between cancer cells and marrow stromal cells (MSCs) is of great interest because of the MSC tropism for tumors. In this study, we investigated human MSC-secreted paracrine factors that potentially contribute to regulation of cancer stem cell subpopulations. Treatment with MSC-conditioned media led to decreased proportion of the cancer stem cell-enriched compartment, which is detected as the side population and the quiescent (G0) cell cycle fraction, in human lung cancer cells, but not in non-lung cancer cells. Of note, the lung cancer-specific suppression of the cancer stem cell-enriched compartment is ensured by fibroblast growth factor (FGF) 10 secreted from MSCs; the MSC-mediated suppression of the compartment was attenuated by neutralization of FGF10 and was substituted by recombinant FGF10 protein. Moreover, the supplementary FGF10 alleviated mRNA expression of stemness marker genes encoding OCT3/4 and SOX2. Consistently, addition of FGF10 to the lung cancer cell culture impaired formation of floating spheres in serum-free condition, suggesting that self-renewal capacity of lung cancer cells was diminished by FGF10. The clinical relevance of these findings was confirmed by in vivo tumor growth as well as in vitro proliferation, in which the FGF10 treatment rendered the lung cancer cells to be more responsive to an anticancer agent probably due to the reduction of chemoresistant cancer stem cells. Collectively, these results suggest that the pharmacological strategy using FGF10 in conjunction with chemotherapeutic agents may exhibit a therapeutic potency in lung cancer treatment.

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## Clinical Application of Pulmonary Function Testing

Yasutaka Nakano, MD., Ph.D.

Japan

Structural changes of the lung may occur in the subsequence of pulmonary diseases. These lung structural changes influence the patients to show the abnormal results of the pulmonary function testing. Thus, physicians usually use pulmonary function testing as a tool to detect the structural changes of the lung and to estimate and diagnose the underlying diseases. We use pulmonary function testing to diagnose chronic obstructive pulmonary disease (COPD), asthma, interstitial lung diseases, and so on. We use pulmonary function testing to evaluate the treatment. We use pulmonary function testing to follow the disease progression. We use pulmonary function testing to know that the subject does not have any lung diseases. In this lecture, clinical application of pulmonary function testing which is seen at the daily respiratory clinic will be discussed. Representative pulmonary diseases, such as COPD, asthma and interstitial lung diseases will also be discussed.



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## Pathology of Asbestos-Related Disease

**Andrew Churg**

Canada

This lecture will briefly review the pathology of asbestos-related disease, and more specifically comment on controversial areas. The differences between chrysotile and commercial amphibole (amosite, crocidolite) asbestos in terms of producing disease will be emphasized. Asbestos induces a variety of benign pleural diseases including pleural effusions, visceral pleural fibrosis, pleural plaques, and rounded atelectasis. Fiber burdens studies show that a vastly greater dose of chrysotile compared to amosite or crocidolite asbestos is required to cause a pleural plaque. The presence of pleural plaques may indicate individuals at risk of developing mesothelioma but not lung cancer. The pathologic criteria for the diagnosis of asbestosis have been recently refined; in particular actual interstitial fibrosis and not just fibrosis of airway walls is now required, along with 2 or more asbestos bodies/cm<sup>2</sup> of tissue. Fiber burden studies show that asbestosis is a dose-response disease and high doses are required for its development. Asbestosis clearly predisposes to the development of lung cancer, but the possible association of asbestos exposure and lung cancer in the absence of asbestosis remains extremely controversial. The development of mesothelioma is strongly dependent on fiber type. Fiber burden studies complement epidemiologic studies in showing that chrysotile asbestos (with its contaminant tremolite) can cause pleural mesothelioma, but only at very high fiber burdens, whereas amosite (and crocidolite) can induce pleural mesothelioma at several hundred fold lower concentrations. High (asbestosis level) exposure to amosite or crocidolite can produce peritoneal mesothelioma, but there is no convincing evidence that chrysotile can produce peritoneal mesothelioma.

# DAY 2 FRIDAY, 14<sup>TH</sup> NOVEMBER

## OSA: Organisation of Care to Reduce Disease Burden

Atul Malhotra

USA

Obstructive sleep apnea (OSA) is a common disorder with major neurocognitive and cardiovascular sequelae. Nasal continuous positive airway pressure (CPAP) is the treatment of choice for OSA based on randomized trials as it can have transformative benefits for some patients. However, other patients have trouble tolerating CPAP and thus alternative therapies are being sought by studying underlying mechanisms. The pathogenesis of the condition is complex but involves an interaction of anatomical factors, upper airway dilator muscle dysfunction, instability in ventilatory control, and other factors. Recent studies suggest that different OSA patients develop the disease for varying reasons. While some OSA patients have primarily anatomical problems, others have primarily dysfunction in upper airway dilator muscles while others have instability in ventilatory control driving apnea occurrence. Some patients likely have combinations of abnormalities driving OSA. Individualized therapy is being considered whereby uvulopalatopharyngoplasty may be highly effective in patients with velopharyngeal compromise. Hypoglossal nerve stimulation may be particularly effective for patients with dysfunction in upper airway dilator muscles. Agents such as oxygen and acetazolamide may help to improve or eliminate OSA in patients with unstable ventilatory control. Combination therapy may be helpful for patients with multiple underlying mechanisms. The arousal threshold is also receiving increasing attention such that patients with a low arousal threshold may be amenable to sedative/hypnotic agents. The raising of arousal threshold may allow the accumulation of respiratory stimuli during stable sleep which can activate pharyngeal dilator muscles and maintain stable breathing. Research is ongoing in this area to define the pathophysiological mechanism underlying OSA using strategies that are readily accessible in the clinic. Such approaches will be critical for the concept of personalized medicine to be widely applied to OSA patients.

## **Can We Apply Personalized Medicine to the Management of Childhood Obstructive Sleep Apnea: Newer Treatment Modalities**

**Carole Marcus**

USA

The obstructive sleep apnea syndrome (OSAS) is common, affecting 2-4% of otherwise healthy children, and a much higher percentage of children with risk factors such as prematurity, Down syndrome, craniofacial anomalies or neuromuscular disease. OSAS result from a combination of anatomic and neuromotor factors. The primary treatment in children is adenotonsillectomy, but approximately 20% may not improve due to ongoing structural abnormalities or neuromotor deficits. This talk will discuss risk factors for those who are less likely to improve after surgery. Continuous positive airway pressure (CPAP) is usually the second line of therapy, but adherence remains a major barrier to its use. Factors predicting CPAP adherence include age, race and maternal education; family interactions are important for CPAP adherence in adolescents. Mode of ventilation is unlikely to affect adherence. Alternative therapeutic methodologies for children with OSAS include weight loss (whether medical or surgical), anti-inflammatory medications, orthodontic treatment and craniofacial surgery. In addition, the role of newer modalities such as nasal expiratory positive airway pressure are being studied. The pros and cons of each therapy, and criteria for patient selection and management, will be presented.

# DAY 2 FRIDAY, 14<sup>TH</sup> NOVEMBER

## Community Based Management of Chronic Obstructive Pulmonary Disease

**Chau Ngo Quy, MD, PhD**  
Vietnam

Introduction: COPD is one of the chronic diseases, cause a serious public health problem in over the world. Community based management (CBM) of chronic obstructive pulmonary disease is a model, which focus on patient. Getting optimal care requires an individualized, patient-centered approach, not only COPD management but also the systemic effects and comorbidities.

Model of CBM: Based on the Bodenheimer T's model to improve of chronic illness care, they suggest the model of care for COPD patient and believe that patient-provider interactions result in care that improves outcomes.

Objectives of CBM:

COPD Prevention: increasing knowledge of community about risk factors of COPD, especially smoking and improving access to community-based smoking cessation.

Early diagnosis of COPD: Provider spirometry and filters for community, increasing programs for screening focus on individuals with chronic respiratory symptoms and or history of exposure smoking and biomass fuel.

Management of COPD and co-morbidities: Providing pharmacological and non-pharmacological managements for patients at primary care level. In addition, never forget to screen comorbidities. Patient's education about self-management, using drug delivery devices, exercise training and action plans. To achieve these goals, it is necessary to reduce the gap between GP and specialists and expand the range of community-based ambulatory services for COPD and develop integrated referral pathways and protocols.

Conclusions: COPD is a chronic, progressive and complex disease that requires an individualized, patient-centered approach. Community based management may help to its management.

## **Real-life studies – A Poor Relation or Important Partner to the Respiratory RCT?**

**David Price**  
Singapore

RCTs are recognised as the gold standard for assessing efficacy, but real-life studies play a complimentary role in evaluating effectiveness. While RCTs are designed to study narrow populations in highly controlled conditions, real-life observational studies and pragmatic trials examine broader populations in less controlled settings. Due to their different designs, conflicting results sometimes arise. These might best be resolved by assessing how rigorous the methods and analyses of each study are.

The different study designs and patient populations of RCTs and real-life studies enable them to answer different important clinical questions. RCTs are suitable for obtaining drug licenses. On the other hand, real-life studies can evaluate questions about practical issues unanswerable by RCTs, such as patient and physician behaviours and preferences, adherence to therapy, inhaler technique, and ethical questions. For example, in the MASCOT trial examining paediatric asthma therapy step-up, recruited patients were already on optimised therapy. Randomisation to alternative therapies, as required for examining step-up, would have been unethical, so the trial was cancelled and a similar real-life study was done instead. Real-life studies have also been used to evaluate treatment effects beyond indications established in RCTs, such as statin use in COPD.

In support of the complementarity of RCTs and real-life studies, the 2014 Global INitiative for Asthma recommendations have recently been updated to suggest that both efficacy (e.g. RCTs) and effectiveness studies (e.g. real-life studies) should be considered when making choices about controller therapy. Real-life studies are increasingly becoming established as important partners to respiratory RCTs.



# DAY 2 FRIDAY, 14<sup>TH</sup> NOVEMBER

## Latent TB: To Treat or Not to Treat

Dean E. Schraufnagel

USA

Basic premises of tuberculosis are: 1. progress toward elimination is too slow; 2. active tuberculosis comes from latent tuberculosis; 3. tuberculosis spreads before the diagnosis is made; and 4. treating latent disease is effective.

Past successes of treating latent disease have largely been with isoniazid, but this drug is not effective against dormant bacilli. Since latent tuberculosis mainly involves dormant organisms, why not use drugs that kill dormant organisms? In fact, the rifamycins are active against dormant bacilli and do shorten regimens. Pyrazinamide, metronidazole, moxifloxacin, aminoglycosides, capreomycin, linezolid, clofazimine, bedaquiline, delamanid, and PA-824 all have activity against nonreplicating bacilli (depending on the assay).

The decision whether or not to treat latent tuberculosis is based on risk-benefit considerations that are over 40 years old and developed to balance the risk of developing tuberculosis occurring in an individual against the toxicity of taking isoniazid for 12 months. They did not consider the long-term protection against tuberculosis versus the risk of side-effects occurring only during the treatment period. Nor did they take into account public health. Shorter courses and more effective medicine change these considerations. The rifapentine once-weekly course was as effective as the standard isoniazid course and had less toxicity (0.5%) and a higher completion rate (82%). With shorter and safer therapy, the risk-benefit balance has swung toward treating all persons with latent tuberculosis.

## Current Situation in Asia Pacific: What We Have Learned

**Dr Chi Chiu Leung**

Hong Kong

The Asia-pacific region is on track to meet all the three 2015 targets in the reduction of tuberculosis (TB) incidence, prevalence and mortality. However, the burdens of TB, drug-resistant TB and latent TB infection (LTBI) remain huge in this most populous region of the world. Many TB cases are still being missed in South-east Asia. Detection and treatment of multidrug-resistant (MDR) / extensively drug-resistant (XDR)-TB remain formidable challenges in the region as a whole.

Achieving the new 2025 and 2035 milestones of 50% and 90% reduction in TB incidence would require accelerated annual rates of decline of 6.7% and 14.9% respectively (c.f. just 2% now). Timely and efficient scaling-up of integrated, patient-centred care and prevention on a population scale is therefore crucial. Furthermore, innovative ways in implementation of both old and new diagnostic and treatment tools are urgently required to curb the further emergence and transmission of drug-resistant TB.

Controlling ongoing transmission alone will not curb endogenous reactivation from LTBI within the human life-span. Revolutionizing improvements in existing LTBI screening and treatment tools are necessary for the daunting task of screening the entire population and treating 1/3 of them. The existing BCG vaccine offers only partial and unreliable protection against pulmonary TB, the crucial transmission link for this airborne infection. With the absence of long-lasting immunity after natural infection, novel ways of augmenting the immune response would be required to realize a major breakthrough in this critical area.

# DAY 2 FRIDAY, 14<sup>TH</sup> NOVEMBER

## Advanced Technologies for Molecular Diagnosis of Cancer

Eiso Hiyama, Keiko Hiyama

Japan

In life science field, high throughput genomic and proteomic analysis so-called 'omics analysis' using next-generation sequencing and mass-spectrometry has spawned a variety of global analysis methods to assess abnormalities in the genome, epigenome, transcriptome, and proteome which are useful to detect new biomarkers for development, inflammation, and carcinogenesis. The markers revealed by these analyses is important both to identify potential therapeutic targets and/or to suggest molecular markers that might predict patient outcome. To identify these markers in the patients with cancer, the cancer liquid diagnosis using plasma free DNA and CTCs (circulating tumor cells) are now developed as minimum invasive method. Moreover, to distinguish the difference among cells and to detect intracellular changes, we developed imaging mass-spectrometry and single cell analysis so called 'cellomics'. Single-cell analysis has attracted attention in many fields of biological studies as a tool to survey the precise mechanisms of cellular and molecular behavior. The development of sensitive mass spectrometry allows the study of molecules in single cell or small regions. In this technology, nanospray-mediated sampling and ionization named Live Single-cell Mass Spectrometry can be used for real-time analysis. And imaging mass-spectrometry is also available for detecting the molecules and drugs in the cells and tissues mounted on slide. This session focuses into the recent advances of molecular diagnostic technologies applicable to pulmonary diseases including lung cancer.

## **Molecular Diagnosis of Lung Cancer**

**Eiso Hiyama, MD. PhD.**

Japan

Identification of sensitive biomarkers predictive of diagnosis, prognosis and drug sensitivity could have a clinically significant impact on cancer treatment strategies. Recently, molecular-targeted therapies have been developed for cancer treatment, especially for lung cancer treatment. Non-small cell lung cancer (NSCLC) patients with epidermal growth factor receptor (EGFR) gene mutations have shown a dramatic response to EGFR tyrosine kinase inhibitors (EGFR-TKI) such as gefitinib and erlotinib. In addition, echinoderm microtubule-associated protein-like 4-anaplastic lymphoma kinase (EML4-ALK) fusion protein is present in approximately 5% of the patients with adenocarcinomas and Crizotinib is an oral tyrosine kinase inhibitor (TKI), which silences the protein product of the ALK fusion gene and has recently been approved for the treatment of NSCLC aberrantly expressing ALK. In neuroblastoma, one of the major childhood cancers, ALK activated mutations in tyrosine kinase domain are also present in approximately 5% of the patients and reported as responsible genes for familiar or multifocal neuroblastomas. Crizotinib is currently under development for lung cancer and neuroblastoma patients as clinical trials. In this paper, I reviewed the current development of molecular diagnosis of cancers, especially in the aspect for target therapies against lung cancer.

# DAY 2 FRIDAY, 14<sup>TH</sup> NOVEMBER

## Recent Advances in Cardiovascular Comorbidity in OSA

Geraldo Lorenzi-Filho

Brazil

Obstructive sleep apnea (OSA) is an underdiagnosed condition characterized by recurrent episodes of obstruction of the upper airway leading to sleep fragmentation and intermittent hypoxia during sleep. Obesity predisposes to OSA, and the prevalence of OSA is increasing worldwide because of the ongoing epidemic of obesity. Recent evidence has shown that surrogate markers of cardiovascular risk, including sympathetic activation, systemic inflammation, and endothelial dysfunction, are significantly increased in obese patients with OSA versus those without OSA, suggesting that OSA is not simply an epiphenomenon of obesity. Moreover, findings from animal models and patients with OSA show that intermittent hypoxia exacerbates the metabolic dysfunction of obesity, augmenting insulin resistance and nonalcoholic fatty liver disease. In patients with the metabolic syndrome, the prevalence of moderate to severe OSA is very high (~60%). In this population, OSA is independently associated with increased glucose and triglyceride levels as well as markers of inflammation, arterial stiffness, and atherosclerosis. Several cohort studies have consistently shown that OSA is associated with increased cardiovascular mortality, independent of obesity. Taken together, these results support the concept that OSA exacerbates the cardiometabolic risk attributed to obesity and the metabolic syndrome. Recognition and treatment of OSA may decrease the cardiovascular risk in obese patients. However, most evidence comes from small trials. There is a urgent need for large randomized multi-center trials to investigate the impact of the treatment of OSA with CPAP in hard end points such as mortality.



## **Moving Towards TB Elimination: Experience and Lessons Learned Through ERS Engagement in Europe**

**Giovanni Battista Migliori**  
Italy

In May 2014, the World Health Assembly passed a resolution approving a new global tuberculosis strategy with ambitious targets aiming at a major reduction of TB incidence and mortality by 2035.

Settings where TB elimination could be pursued are low-TB incidence countries with <100 cases per million people.

WHO and ERS established an expert group and, in consultation with more than 30 country representatives, developed a framework of measures to accelerate TB control towards elimination.

This adaptation addresses the fundamental challenges in low-incidence settings with the objective to reach a “pre-elimination phase” defined as <10 cases per million people by 2035 to achieve elimination before 2050.

Low-incidence countries have essentially four major challenges: (1) TB concentrated in vulnerable and high-risk groups; (2) re-activation of latent infection is more important than recent transmission; (3) cross-border migration; (4) dwindling visibility, clinical expertise and political commitment.

Facing these challenges requires interventions in 8 priority areas: from ensuring government commitment to focusing interventions on vulnerable people and migrants, and investing in surveillance, research, and global aid.

Consensus on new targets followed intensive dialogue among country representatives with some concerned that targets are too ambitious: major reduction in incidence and mortality would be possible only with the availability of new tools allowing rapid diagnosis in vulnerable populations, large-scale screening and treatment of latent infection in high-risk groups, until a vaccine is available.

Indeed, the possibility of providing chemoprophylaxis to a vast number of people at risk is one of the controversial issues to be addressed in future efforts.

# DAY 2 FRIDAY, 14<sup>TH</sup> NOVEMBER

## Current Molecular Targets in NSCLC

Hagiwara Koichi

Japan

Targeted strategies have become the mainstream of the lung cancer treatment. Most of the molecular targeting drugs are tyrosine kinase inhibitors. Because the tyrosine kinases have stemmed from a common ancestral gene, the molecular structure is similar, and therefore a single drug inhibits multiple tyrosine kinases. Therefore, using drugs that have already on the market, we may be able to target several driver oncogenes. Such drug and target pair include gefitinib, erlotinib, and afatinib against EGFR mutated lung cancer, crizotinib and alectinib against lung cancer with ALK fusion gene, crizotinib against lung cancer with ROS1 fusion gene, vandetanib against lung cancer with RET fusion gene, and sorafenib and vemurafenib against BRAF mutated lung cancer. These indicate that we need to test multiple genes that are “druggable” before determining the treatment regimen of lung cancer. The framework that enables multiple genetic tests may be constructed by utilizing a high-speed DNA sequencer. Clinical samples are limited in amount, and the turn-around time should be short for the genetic information is readily utilized before initiation of the treatment. I would like to consider how such system may be constructed and how it is applied to the clinical oncology.

## Updates of Lung Cancer

Hagiwara Koichi

Japan

Lung cancer is one of the most difficult cancers to cure. More than half of the patients are found their disease in an advanced, inoperable stage. Up until ten years ago, they had been treated chemotherapy and/or radiotherapy: the overall survival was less than one year, and nobody survived more than 2 years. The discovery of the mutation of the epidermal growth factor receptor (EGFR) changed the entire picture of the treatment. When mutation-positive patients are treated with the regimen containing EGFR tyrosine kinase inhibitor (EGFR-TKI), the overall survival extends to 2 to 3 years. Concurrent administration of chemotherapy and EGFR-TKI is expected to further extend overall survival. The discovery of oncogenic fusion genes, EML4-ALK, has made ALK-TKI as a therapeutic option for the lung cancer positive for the gene. For adenocarcinoma without EGFR mutation, a new chemotherapy agent, pemetrexed, and anti-angiogenic monoclonal antibody, bevacizumab, have demonstrated an apparent efficacy. The discovery of molecular mechanism that causes cancer, and the results of the clinical trials investigating the effect of molecular targeting agents, advances the science of lung cancer. I would like to review the progression of this field.

# DAY 2 FRIDAY, 14<sup>TH</sup> NOVEMBER

## Phenotyping Lung Disease Using Optical Coherence Tomography (OCT)

**Harvey O Coxson**  
Canada

Airway diseases are a growing burden around the world. However, the pace of new drug and biomarker discovery has lagged behind those of other common disorders such as cardiovascular diseases and diabetes. One major barrier in airways research has been the inability to accurately visualize large and small airway remodeling using non or minimally invasive instruments. Optical coherence tomography is a new bronchoscopic imaging technique that has generated considerable interest because the spatial resolution approaches that of histology. While relatively more invasive than computed tomography, it has the advantage of not exposing the patient to ionizing radiation. Thus, with the aid of OCT, we may be able to accurately determine and quantify the extent of airway remodeling in asthma and chronic obstructive pulmonary disease. Therefore, these new imaging techniques are very likely to play a front-line role in the study of airways disease and will, hopefully, allow clinicians to phenotype individuals, thereby personalizing their treatment.

## **Omalizumab Improves Quality of Life and Asthma Control in Chinese Patients with Moderate-to-Severe Asthma: A Randomised Phase III Study**

**Jing Li, Jian Kang, Janice Canvin, Changzheng Wang, Jing Yang, Michael Humphries, Nanshan Zhong**  
China

**Introduction:** Omalizumab, an anti-IgE monoclonal antibody, has been found to be effective and safe in the treatment of patients of different ethnicities with moderate-to-severe allergic asthma. We report here the effect of omalizumab on the quality of life, asthma control and safety in Chinese patients with moderate to severe allergic asthma.

**Methods:** This was a randomised, double blind, parallel group, placebo controlled, phase III study to assess the quality of life, asthma control and safety of 24 weeks of omalizumab therapy in Chinese patients, aged 18-75 years, with moderate-to-severe persistent allergic asthma. Asthma Quality of Life Questionnaire (AQLQ) and Asthma Control Questionnaire (ACQ) scores were assessed at baseline and at week 24. Asthma exacerbation rates were also analysed.

**Results:** Among the 608 patients included in the full analysis set, at week 24 a higher proportion of patients treated with omalizumab (n=306), vs. placebo (n=302), achieved clinically relevant improvements in AQLQ (58.2% vs. 39.3% [analysed n=182 vs. 178];  $p < 0.001$ ; change from baseline [ $\Delta$ BL]=0.51 vs. 0.10) and ACQ (49.5% vs. 35.5% [analysed n=210 vs. 211];  $p = 0.003$ ;  $\Delta$ BL=-0.51 vs. -0.34) scores. Although not powered to study differences in exacerbation rates ( $p = 0.097$ ), exacerbations in winter months were less frequent in the omalizumab group vs. placebo (2 vs. 21). Adverse event and serious adverse event rates were comparable in both groups. One death from asthma exacerbation occurred in the omalizumab group.

**Conclusions:** Omalizumab improves quality of life and asthma control in Chinese patients with moderate-to-severe persistent allergic asthma with a good safety profile.



# DAY 2 FRIDAY, 14<sup>TH</sup> NOVEMBER

## **Airway Secretion: Role of PDE4 in Airway Mucus Hypersecretion in COPD**

**Jun Tamaoki**  
Japan

Airway mucus hypersecretion is one of the characteristic features of COPD, and a large amount of secretions stagnated in the respiratory lumen may cause airflow limitation, impairment of mucociliary transport, and recurrent respiratory infection. Although there are several mucoactive agents, these symptoms are generally difficult to treat. A recent GOLD guideline recommends PDE4 inhibitors for patients with severe COPD, but the role of PDE4 in mucus hypersecretion remains uncertain. We studied the effects of ibudilast, a PDE4 inhibitor, on MUC5AC expression in NCI-H292 cells in vitro, and found that exposure of cells to TGF $\alpha$  increased MUC5AC protein and mRNA expression, and this effect was inhibited by pretreatment with ibudilast in a concentration dependent manner. We then conducted an open, non-controlled trial, and examined the effect of ibudilast on sputum production and its impact in patients with COPD whose symptoms were resistant to conventional bronchodilators, mucoactive agents, macrolides or inhaled corticosteroids. Treatment of patients with ibudilast for 8 weeks caused favorable influences on sputum hypersecretion, judging from CASA-Q, a cough and sputum assessment questionnaire. Additionally, ibudilast significantly shortened nasal clearance time measured by saccharine test and decreased solid composition of the sputum (dry/wet weight ratio), indicating a reduction of mucus glycoprotein synthesis and an improvement of airway mucociliary clearance. These results suggest that PDE4 plays a role in airway mucus hypersecretion and that the inhibition of this enzyme may be one of the important strategies for the treatment of COPD patients especially with bronchitic phenotype.

## Second Line Treatment and Beyond Targeted Therapy in NSCLC

Kazuhisa Takahashi

Japan

Lung cancer is the leading cause of cancer death worldwide. However, the prognosis of patients with advanced non-small cell lung cancer (NSCLC) is gradually improving. This phenomenon may be due to several reasons. One is the development of molecular targeting drugs, including epidermal growth factor receptor (EGFR)-tyrosine kinase inhibitors (TKI) and anaplastic lymphoma kinase (ALK) inhibitors. The second reason is the introduction of second and later line(s) chemotherapy into clinical practice, such as regimens using docetaxel, pemetrexed and erlotinib. The third reason is the introduction of maintenance therapy. In fact, the PARAMOUNT study, a randomized phase III study comparing the continuation of pemetrexed with a placebo as maintenance therapy following induction treatment consisting of cisplatin plus pemetrexed in patients with non-squamous NSCLC, revealed that continuation maintenance with pemetrexed prolongs overall survival. In this symposium, I would like to provide an overview of the current status and future perspectives of second-line treatment in patients with NSCLC. I will also touch on the efficacy of continuation of EGFR-TKI beyond progress disease.

# DAY 2 FRIDAY, 14<sup>TH</sup> NOVEMBER

## Metabolic Disorders and Comorbidity in OSA

Kazuo Chin, Yoshihiro Toyama

Japan

Prevalence of obstructive sleep apnea (OSA) has been increasing in parallel with the obesity epidemic, and intermittent hypoxemia, frequent arousals and sleep fragmentation by OSA cause systemic inflammation, sympathetic activation and oxidative stress. These responses lead to hypertension and cardiovascular consequences, insulin resistance and diabetes mellitus, dyslipidemia, and metabolic syndrome. On the other hand, short sleep and sleep restriction also alter sympathetic nervous system activity and increase inflammatory markers such as C-reactive protein, tumor necrosis factor- $\alpha$  and interleukin-6, and now there is a growing body of evidence to suggest that short sleep duration is a risk factor for mortality, hypertension and cardiovascular consequences, insulin resistance and diabetes mellitus, and, metabolic syndrome. Thus, each of OSA or/and short sleep can induce inflammatory responses, sympathetic activation and subsequent metabolic dysregulations. Thus, it is important to consider the associations among several factors such as OSA, sleep duration, sleep fragmentation and sex differences and how combined effects of OSA, short sleep duration and sleep fragmentation affect over all morbidities. In this symposium, we discussed 1) the relationships among body mass index (BMI), respiratory disturbance index (RDI) and sleep duration, 2) hypertension, sleepiness, sleep duration and OSA, 3) glucose metabolism, diabetes mellitus, sleepiness and OSA, 4) dyslipidemia, serum lipid profile, sleep duration and OSA, and 5) metabolic syndrome, OSA and sleep duration. In conclusion, OSA, sleep duration and metabolic dysregulations are interrelated each other. However, because of complexity of these interactions, all of the associations or mechanisms have been not elucidated.

## Management Recommendations

**Kittipong Maneechotesuwan**

Thailand

There has been no evidence-based recommendation for the treatment of asthma COPD overlap syndrome (ACOS) because of the exclusion of smoking asthmatics and COPD patients with reversible airflow obstruction from almost all clinical trials. Patients with ACOS experience acute exacerbations with higher frequency and greater severity than lone COPD. Pharmacotherapeutic considerations for ACOS require an integrated approach, first to identify the relevant clinical phenotypes, then determine the best available classes of drugs that provide the beneficial effects on important outcomes including lung function, acute exacerbations, quality of life and mortality. In addition, it is also important to target treatments to disrupt pathobiologic processes particularly small airway inflammation and smooth muscle dysfunction that give rise to pathophysiologic pattern in ACOS. Current drug treatments that can effectively prevent acute exacerbations of asthma and COPD and control symptoms may be applied to ACOS for the time being until the presence of clinical trials to demonstrate their effectiveness in controlling both symptoms and reducing exacerbations in ACOS either in a single or combination manner. Revised GINA guideline 2014 has recommended to start the initial therapy for ACOS by commencing treatment as for asthma until further investigation and adding long-acting beta2 agonist (LABA) as necessary. If the further assessment suggests COPD, symptomatic treatment with bronchodilator or combination therapy should be started, but not ICS alone as monotherapy. Other therapeutic strategies for ACOS should be included, including smoking cessation, pulmonary rehabilitation, vaccination and treatment of comorbidities.

# DAY 2 FRIDAY, 14<sup>TH</sup> NOVEMBER

## Role of Spirometric Full Flow-Volume Curve in Searching Causes of Dyspnea

**Le Thi Tuyet Lan**  
Vietnam

Dyspnea is the common chief complaint of the patients with respiratory diseases. Besides clinical examination and imaging, pulmonary function tests are crucial for making diagnosis the cause of dyspnea.

Spirometry is the basic test and is used widely. But up to now, some doctors use only the expiratory (upper) part of the flow-volume curve.

We use the full flow-volume curve of spirometry to differentiate many causes of dyspnea. The abnormalities of flow-volume curve could be divided into 2 groups : restriction and obstruction. The later one has three subgroups:

1. Only in the expiratory part (variable, intrathoracic obstruction).
2. Only in the inspiratory part (variable, extrathoracic obstruction).
3. In both parts (fixed obstruction in upper airway).

The flow-volume curve of the diseases in each subgroup are illustrated and additional test are included where needed to have a final diagnosis.

Besides the common diseases such as asthma and COPD, the expiratory part of the flow volume curve can help to orient the physician to less frequent disease such as vascular ring, mediastinal mass, tracheomalacia, endobronchial tuberculosis and even achalasia.

The emphasized point is the need of using the inspiratory (lower) part of the flow-volume curve : many vocal cord disorders, tumor of thyroid gland are detected based on the lower part of the curve.

The fixed obstruction, presented as plateaus in both inspiratory and expiratory parts could orient the diagnosis toward stricture or tumor of the trachea. The stricture sequelae of tracheo-bronchial tuberculosis is common and often was misdiagnosed as asthma.

Other abnormalities such as sleep apnea, foreign body can also be shown on flow volume curve of the spirometry.

Conclusion : The dyspneic patients may have disorders in the inspiratory, expiratory part or both.

The flow-volume curve must be used routinely in full form in order to help doctors in orientation toward an accurate diagnosis.



## **Small Airways in Asthma: Pathogenesis**

**Masakazu Ichinose**

Japan

Asthma is an inflammatory airway disease from the central to small airways. Especially, small airways are important because the mucosal area is wide and difficult to reach inhalation agents. Recently, it has been reported that the concentration of nitric oxide originating in the small airways is elevated in asthmatic patients with frequent exacerbations, suggesting that prolonged inflammation in small airways could be a key target for assessing the future risk of asthma. Airway remodeling in small airways via fibrotic changes in the airway wall causes persistent airflow limitation. These fibrotic changes are due to oxidative stress and inflammatory transcriptional up-regulation. In this talk, I will cover the importance of small airway diseases in asthma and discuss how to treat it.

# DAY 2 FRIDAY, 14<sup>TH</sup> NOVEMBER

## Clinical Importance of Digital Imaging of Terminal Airspace Enlargement in COPD

**Michiaki Mishima**

Japan

The clinical importance of the digital imaging of terminal airspace enlargement in COPD mainly from our data will be presented. Haruna A et al. reported that emphysematous change had strong association with mortality and may predict respiratory mortality in COPD (Chest, 2010). Tanabe N et al. demonstrated that exacerbations are involved in emphysema progression in patients with chronic obstructive pulmonary disease (AJRCCM, 2011). Mishima M et al. reported that the cumulative size distribution of the LAA clusters followed a power law characterized by an exponent D (fractal dimension), and showed that D value is a sensitive and powerful parameter for the detection of the terminal airspace enlargement (PNAS, 1999). Recently, Gotoh S et al. developed a novel method to efficiently isolate alveolar type two (AT2) cells from induced pluripotent stem (iPS) cells in the by way of endodermal lineage. This method may be useful to address the pathogenesis of COPD and regenerative medicine to repair the destroyed lung (Stem Cell Reports, 2014).

## **Clinical Application of iPS Cell in the Respiratory Field Including COPD and Asthma**

**Michiaki Mishima**

Japan

Induced pluripotent stem (iPS) cells, developed by Shinya Yamanaka in Kyoto University who won the Nobel Prize in 2012, theoretically have a potential to differentiate into any cells in the body.

In this paper, the updated our outcome of iPS research in the respiratory field collaborated with Prof. Yamanaka will be demonstrated. Since alveolar type I cells differentiated from alveolar type II (AT2) cells, the key cells for lung regeneration is AT2 cells. We have been developing a strategy to induce AT2 cells from iPS cells by way of endodermal lineage. Recently, Gotoh S et al. developed a novel method to efficiently isolate alveolar type AT2 cells from induced iPS cells by way of endodermal lineage. This method may be useful to address the pathogenesis of COPD or asthma and regenerative medicine to repair the destroyed lung (Stem Cell Reports, 2014; 3(3): 394-403). We expect that this investigation must convey the new paradigm in the respiratory medicine.

# DAY 2 FRIDAY, 14<sup>TH</sup> NOVEMBER

## **The Implications of Real-Life (Comorbid Conditions, Inhaler Technique And Lifestyle Factors) on Asthma Management: Is There Any Evidence Available?**

**Omar S Usmani**  
United Kingdom

Randomized controlled trials (RCTs) in asthma include tightly-controlled, well-characterised populations that represent only a subgroup of the broadly heterogeneous patient population treated in everyday clinical practice.

Patients with asthma, including those with comorbid conditions (e.g. rhinitis, obesity), those who smoke, or those with poor inhaler technique are not included in RCTs due to highly selective inclusion/exclusion criteria. Hence, concerns exist about the extent to which RCT data can be accurately extrapolated to reflect treatment effectiveness and long-term safety in real-life asthmatic patient populations. RCTs should be complemented by a diversity of approaches that involve analysing the totality of the evidence base.

Guidelines focus on therapeutic drug in the management of patients with asthma, but often do not evaluate the differences between inhaler devices or take into account inhalation technique that can modulate the effectiveness of the drug in day-to-day real-life practice. Adherence to inhaler therapy is near optimal in RCTs, but in clinical practice we struggle as practitioners to engage our patients to take their medications as prescribed. Smokers are typically excluded from asthma RCTs, but their inclusion in observational asthma studies and pragmatic trials provides a way of assessing the relative effectiveness of different treatment options to manage this important clinical subgroup constituting approximately 20% of patients with asthma.

Is there any available evidence? This presentation will highlight how real-life effectiveness research can use observational or clinical trial designs with emphasis on high external validity to complement classical efficacy RCTs with their high internal validity.

## Does the Treatment of the Small Airways Matter?

Omar S. Usmani

UK

There is increasing academic interest and clinical awareness in the understanding of the contribution of the small airways to the clinical expression of disease in patients with asthma. Pathological evidence shows small airways disease is present throughout the airway tree in patients with all severities of asthma. Physiological data clearly reveal that the main site of airflow limitation in asthma and chronic obstructive pulmonary disease (COPD) is that of the small airways. There is reinvigoration in physiological tests to identify distinct airways responses in this region from those of large airways. New imaging modalities are also allowing investigators to understand structure function relationships within the airway tree. These techniques are now being utilised to investigate whether targeting inhaled therapies to the small airways is of benefit to patients. In order to achieve this, it is recognised that aerosol drug particle size and patient inspiratory flow are the major determinants influencing the extent, distribution and site of inhaled drug deposition within the airways and regional targeting of the small airways. With the technology to direct inhaled therapy to the small airways coupled with the ability to assess small airway responses the challenge now is to understand whether small airways therapy translates to better patient centred outcomes such as asthma control.



# DAY 2 FRIDAY, 14<sup>TH</sup> NOVEMBER

## **Lung Function in Various Diseases. How to Use Pulmonary Function Tests Most Effectively to Guide Correct Diagnosis and Treatment**

**Paul Enright**  
USA

PFT results only increase or decrease the pre-test probability of disease. You should be uncertain that the results are normal or abnormal when they are near the lower limits of the normal range. Use as few parameters as possible when interpreting the results.

The most common reasons for ordering PFTs when managing patients are for 1) dyspnea evaluations (order spirometry, DLCO, and BNP), 2) infiltrates on chest x-ray (order spirometry and DLCO), 3) chronic cough (if spirometry is normal, consider an inhalation challenge test), and 4) to verify treatment response during follow-up visits (measure change in FEV<sub>1</sub>, FVC, or DLCO).

Lack of a bronchodilator response does not rule out asthma nor confirm COPD. More than half of patients with severe COPD are BD “responders.” Hyperinflation and air trapping are proportional to obstruction severity, so lung volume tests are not helpful for patients with airway obstruction and a chest x-ray. About 20% of patients with severe COPD also have heart failure, so order a BNP to screen for heart failure. DLCO is the second most valuable PFT for clinicians, and is now available as a small instrument. A low DLCO predicts oxygen desaturation during exercise. The six-minute walk test provides an objective measure of exercise limitation and is easy to perform in outpatient settings.

## Future Therapies of Asthma

Peter Barnes

UK

Inhaled corticosteroids (ICS) and long-acting  $\beta$ 2-agonists (LABA) will continue to be the mainstay of asthma therapy in the future, including patients with ACOS - who require maximal bronchodilatation for the COPD component and corticosteroids to deal with the asthmatic component. In addition, these patients are likely to benefit from a long-acting muscarinic antagonist (LAMA), several of which are now becoming available. ACOS patients may therefore be suitable for triple therapy (ICS+LABA+LAMA), although developing such combinations may be challenging. Twice daily (ciclesonide/formoterol/tiotropium and budesonide/formoterol/glycopyrrolate) and once daily triples (fluticasone furoate/vilanterol/umeclidinium and mometasone/indacaterol/ glycopyrrolate) are already in development. The main challenge in future asthma therapies is in treatment of patients with severe disease who are relatively resistant to the anti-inflammatory effects of corticosteroids. This has led to a search for alternative anti-inflammatory treatments, such as phosphodiesterase-4 and p38 MAP kinase inhibitors, although these drugs are limited by side effects. More specific therapies include cytokine blockers, such as anti-IL-5 and anti-IL-13, which may be suitable for specific phenotypes of asthma with eosinophilia or ACOS patients with eosinophilia. ACOS patients may have predominantly neutrophilic inflammation and this may be targeted by antagonists of the chemokine receptor CXCR2, non-antibiotic macrolides and by blockers of IL-17. An alternative approach is to target the corticosteroid resistance mechanisms, which involve a reduction in histone deacetylase-2 through the activation of phosphoinositide-3- kinase- $\delta$ . This pathway may be targeted by existing treatments, including theophylline, nortriptyline and macrolides, or by newly developed inhaled PI3K $\gamma/\delta$  inhibitors. Since this pathway is driven by oxidative stress novel antioxidants, such as Nrf2 activators are also in development.

# DAY 2 FRIDAY, 14<sup>TH</sup> NOVEMBER

## What Happens to a Child with Untreated Obstructive Sleep Apnoea?

Rosemary SC Horne

Australia

During childhood sleep is at a lifetime maximum. Sleep disordered breathing (SDB) is common in childhood and affects up to 35% of children. SDB forms a spectrum of severity from primary snoring, where there are no associated gas abnormalities or sleep disruption to obstructive sleep apnoea (OSA) which is associated with repetitive hypoxic events and frequent arousals from sleep. Primary snoring is most common form of SDB, with OSA occurring in 1-5% of children. SDB of all severities is associated with significant adverse daytime consequences including behaviour, attention and learning. SDB is also associated with adverse effects on the cardiovascular system including elevated heart rate and blood pressure and impaired autonomic cardiovascular control. In children, SDB is primarily due to enlarged tonsils and adenoids and the primary treatment is surgical removal of these tissues. Surgery is however usually only carried out in children with more severe disease, with the majority of children with primary snoring not having surgical intervention. Despite the frequency of adenotonsillectomy there have been limited studies of the effectiveness of this treatment is ameliorating the behavioural, neurocognitive and cardiovascular effects and most of these studies have only followed children up in the short term (i.e. < 1 year). There are even fewer studies of what happens to children who are not treated. Data from longer term follow up studies (3-4 years) will be presented. These studies highlight the need to consider the age of the child and suggest that the threshold for treatment in children should be revised.

## Phenotyping of COPD from Asian Perspective

Sang-Do Lee

Republic of Korea

Clinicians have been long aware that neither the traditional distinctions of “emphysema” versus “chronic bronchitis” nor the traditional clinical phenotypes of “blue bloater” and “pink puffer” are sufficient to categorize patients that suffer from chronic obstructive pulmonary disease (COPD). With an increased understanding of pathophysiologic variation, COPD now clearly represents a spectrum of overlapping diseases with important extrapulmonary consequences.

A “phenotype” describes the outward physical manifestations of a particular disease, and comprises anything that is part of the observable structure, function or behavior of an individual. Such phenotypic distinctions in COPD include: frequent exacerbator, pulmonary cachectic, rapid decliner, airways hyperresponsiveness, impaired exercise tolerance, and emphysema versus airways disease. These variable manifestations, each with unique prognostic, clinical and physiologic implications, represent distinct phenotypes within COPD. While all of these phenotypes have smoking as a common risk factor, the other risk factors that determine these phenotypes remain poorly understood. An individual smoker has variable expression of each phenotype and there is mounting evidence that COPD phenotypes have different clinical outcomes. These phenotypes can be broadly classified into one of three groups: clinical, physiologic and radiographic.

To understand the heterogeneity of COPD in Asian countries we organized Asian Network for Obstructive Lung Diseases (ANOLD) in 2008. Through this network we found that characteristics of COPD patients in Asian countries vary and the history of exposure to biomass fuels or dusty jobs was related to frequency of symptoms, severe airflow limitation, and poor quality of life. We also evaluated whether there are subgroups of COPD patients with distinct phenotypes and found subgroups of COPD patients with distinct phenotypes. The fractions of the COPD subgroups among four Asian regions were different and might suggest that there are substantial differences in the severity and a potential subtype in Asian regions.

# DAY 2 FRIDAY, 14<sup>TH</sup> NOVEMBER

## Compensation for Asbestos-Related Diseases in Korea : Current Situation

**Soon-Hee Jung, M.D., Ph.D**  
Republic of Korea

In Korea, the asbestos related diseases (ARDs) patients and the bereaved can be compensated through Asbestos Damage Relief Law enacted by the Ministry of Environment in 2011 to provide fair, prompt relief to victims and to the bereaved of ARDs and to address the health damage caused by asbestos exposure. The ARDs include malignant mesothelioma, lung cancer, and asbestosis. The damaged patients can be compensated by medical expense, medical treatment allowance, relief benefit adjustment money, funeral service expense. The special bereaved families can be compensated by special condolatory expense for the bereaved and special funeral service expense. The applicants can be certificated by three steps through Judgment Committee, Review Committee, and Re-review Committee.

During the last three years after enforcement of asbestos damage relief act in 2011, 1,740 asbestos-related diseases (ARDs) patients and the bereaved applied for the compensation of asbestos damage and 1,261 (72.5%) applicant were certified. The percent of applicants among certified applicants was 50.9% in MM, 40.6% in asbestosis and 8.5% in lung cancer. The certified percent of patients was 91.2% in MM, 66.2% in asbestosis, and 8.0% in lung cancer. The certified bereaved was 77.5% in MM, 1% in asbestosis, and 9.3% in lung cancer. For the certification of MM, the patient and the bereaved have to apply with pathologic report and an application letter. They can be compensated through the pathologically confirmation after the review of pathologists among the committee members. However, we have experienced many MM cases with diagnostic pitfalls, diagnosed at the several hospitals because of absence of diagnostic guidelines of MM. Asbestosis and lung cancer were same situation.

So we have studied the project of diagnostic guideline of ARDs to provide a fair, prompt relief to victims of ARDs in Korea through the financial support of Korea Environment Industry & Technology Institute (KEITI).



## Malignant Pleural Mesothelioma - Diagnosis and Management

Takashi Nakano

Japan

Mesothelioma is an uncommon neoplasm arising from the mesothelial cells of the pleura, peritoneum, pericardium and tunica vaginalis. A total of 80% of all mesothelioma are pleural in origin. The increasing incidence of mesothelioma in almost all industrialized countries is characterized by its association with commercially used asbestos, and its long latency period of 40 years. High iron content of amphiboles fibers contributes to their higher carcinogenic potential with generating reactive oxygen and nitrogen species. Paradoxically, oxidoreductase enzyme thioredoxin acts as one of antioxidants, and its overexpression has been demonstrated in mesothelioma cells, and serum level is significantly increased in patients with malignant pleural mesothelioma(MPM). Early stage MPM frequently present with asymptomatic pleural effusion as the first clinical sign. Therefore fluid cytology is the first step for diagnosis and thoracoscopy is the key investigation to obtain sufficient material allowing immunohistochemical characterization and adequate visual examination in the pleural cavity. Cisplatin+pemetrexed is the standard first line chemotherapy, which has remained unchanged for 10 years since the randomized trial in 2003. Radical surgery extrapleural pneumonectomy is associated with 4-9.5% mortality and >25% serious complications. Pleurectomy/Decortication(P/D) is well tolerated and produces low mortality and morbidity, but does not offer a macroscopic complete resection in most cases. However, retrospective analyses demonstrated better outcomes among those who underwent P/D. The role of surgery has been the subject of debate, however, surgical MCR and control of micrometastasis and invasion are pivotal role in the multimodality therapy for MPM.

# DAY 2 FRIDAY, 14<sup>TH</sup> NOVEMBER

## Fluid Shift in Patients with the Sleep Disordered Breathing

**Takatoshi Kasai**

Japan

The high prevalence of sleep disordered breathing (SDB) in patients with fluid retention such as heart failure (HF), renal failure (RF) and drug resistant hypertension (DRH) led us to hypothesize that fluid retention and, more specifically, nocturnal shift of dependent fluid rostrally while recumbent during sleep, is involved in the pathogenesis of SDB. Recently, our group demonstrated that in response to an application of lower body positive pressure, neck circumference increased, the pharyngeal cross-sectional area decreased, and pharyngeal resistance and collapsibility increased simultaneously with a reduction in leg fluid volume. We also demonstrated that in patients with HF, RF, and DRH, there were direct relationships between the volume of fluid displaced gravitationally from the legs overnight and both the overnight increase in the neck circumference and the severity of SDB. These data suggest that rostral fluid shift in patients with SDB behaves in a way that would predispose to upper airway obstruction during sleep. In this session, I would review these data and discuss possibilities to utilize this concept for the treatment of SDB.

## Pulmonary Stem Cell: Role in Lung Disease Pathogenesis and Therapy

Toshiaki Kikuchi

Japan

Recent studies suggest that bronchiolar progenitors that exist within bronchioles are capable of long-term self-renewal to maintain the normal airway epithelium. In this study, for development of novel therapeutic strategies to modulate the stem cell-like capacity, we characterized the gene expression profile of mouse bronchiolar progenitors by using the Agilent microarray system. Bronchiolar progenitor and club cell (formerly known as Clara cell) subsets were isolated according to the surface phenotype of CD31<sup>neg</sup> CD45<sup>neg</sup> CD34<sup>neg</sup> Sca-1<sup>pos</sup> with low and high autofluorescence, respectively. Among genes that represented significant differences of expression in bronchiolar progenitors compared to club cells, we picked an up-regulated gene, and referred to the gene product as bronchiolar progenitor factor 1 (BPF1) on an assumption that BPF1 may be requisite for the bronchiolar progenitor behavior. Contrary to the assumption, the genetic deficiency of BPF1 increased the number of bronchiolar progenitors, and decreased lung inflammation following naphthalene-induced lung injury. In further gene expression microarray experiments, we found a gene with the most remarkable up-regulation in bronchiolar progenitors of BPF1-deficient mice compared to those of wild-type mice, and referred to the gene product as bronchiolar progenitor factor 2 (BPF2). Upon treatment with intravenous administration of recombinant protein BPF2, wild-type mice displayed the similar phenotype to BPF1-deficient mice; the BPF2 treatment dampened the naphthalene-induced inflammation with the increased number of bronchiolar progenitors. These results suggest that BPF2 may provide novel therapeutic approaches to inflammatory lung disorders.

# DAY 2 FRIDAY, 14<sup>TH</sup> NOVEMBER

## Epithelial Mesenchymal Transition in Airway Epithelial Cells

Yasuhiro Yamauchi

Japan

Tissue fibrosis has been suggested to be associated with an abnormal wound healing process derived from sustained chronic injury and consequent excessive healing. Persistent injury from various stimuli activates the epithelial cells, which produce abundant several cytokines, chemokines, and growth factors. These mediators promote chronic airway inflammation, which induce excessive repair process and provide fibrotic change in tissue. The cells related with fibrosis have been reported to originate at residual fibroblasts, bone marrow-derived progenitors of fibroblasts, or mesenchymal cells that underwent epithelial-mesenchymal transition (EMT) from epithelial cells. Transforming growth factor (TGF)- $\beta$  is one of the key mediators involved in the pathogenesis of fibrosis, since TGF- $\beta$  regulates production of extracellular matrix proteins, proliferation, and differentiation of fibrosis and myofibroblasts. Also, TGF- $\beta$  is a potent inducer of EMT and would play important roles on the fibrosis through induction of EMT process. Since chronic inflammation induces abundant TGF- $\beta$  and associates with the formation of fibrosis, several inflammatory mediators in the airway, such as tumor necrosis factor (TNF)- $\alpha$ , interleukin-1 $\beta$ , and TNF superfamily 14 which is called as LIGHT, would affect EMT process in association with TGF- $\beta$ . The mechanisms of EMT in airway epithelial cells under chronic airway inflammation would be summarized.

## Phenotyping Lung Disease Using Computed Tomography

Yasutaka Nakano, MD., Ph.D.

Japan

Structural changes of the lung may occur in the subsequence of pulmonary diseases. These lung structural changes influence the function of the lung. Structural and functional changes of the lung work together and lead to symptoms. Part of the structural changes of the lung could be assessed using computed tomography (CT).

CT is a precise tool to measure the specific gravity. Using this function, CT has been used to evaluate and quantify the structure of the lung. This type of approach is now called as 'Quantitative Computed Tomography (QCT)' analysis. In chronic obstructive pulmonary disease (COPD), emphysematous lesions and airway lesions are evaluated using QCT analysis. CT extent of emphysematous lesions are calculated as the percentage of low attenuation volume at the threshold of -950 Hounsfield units (%LAV-950) and airway lesions are quantified as the square root of wall area of a hypothetical airway with an internal perimeter of 10 mm (SRWA-Pi10). Using these two parameters patients with COPD could be divided into phenotypes: airway dominant, emphysema dominant, and mixed (airway plus emphysema). The difference of these phenotypes will also be discussed in this lecture.



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## Phenotyping Lung Disease using Magnetic Resonance Imaging (MRI)

Yoshiharu Ohno

Japan

When magnetic resonance (MR) imaging was first implemented, many investigators were interested in this new technique for not only brain, but also other areas including chest. As a result, from the 1980s to the early 1990s, MR imaging was tested to evaluate different lung diseases as well as mediastinal, pleural and cardiac diseases by many physicists and radiologists. However, the MR systems, sequences and other applications at that time were very primitive and limited, adequate image quality within an appropriate examination time could not be realized. Therefore, it could not be demonstrated that MR could be substituted for computed tomography (CT), pulmonary angiography and/ or nuclear medicine studies. Until 2000, MR imaging was therefore used only for some minor clinical indication.

In the 2000s, however, technical advances were reported by many basic and clinical researchers, in particular for lung MR imaging, which has been one of the more challenging fields for MR imaging. State of the art pulmonary MR imaging can provide not only functional and metabolic information, but also morphological information with relatively high spatial resolution within appropriate examination time, and may therefore be able to perform as a substitute and/or in a complimentary role in management of patients with pulmonary and/ or cardiopulmonary diseases.

This lecture covers 1) state-of-the-art pulmonary MR techniques applied for phenotyping of lung diseases, and 2) future direction of pulmonary MR imaging.

## Therapeutic Effects of Histone Deacetylase Enzyme 6 Inhibitors (HDAC6i) in a Murine Asthma Model

Yuan Ren  
China

Background and purpose: Airway inflammation, airway remodeling and airway hyperresponsiveness are major aspects of asthma pathology. Histone deacetylase inhibitors have a wide range of effects that demonstrate therapeutic effects in animal models of chronic inflammatory diseases. In this study, we investigated the effect of Tubastatin A Hcl, a selective HDAC6 inhibitor, on the development of chronic allergic airway disease mice with airway inflammation, airway remodeling and airway hyperresponsiveness. Methods: Wild-type BALB/C mice were immunized intraperitoneally three times with ovalbumin (OVA) + aluminum hydroxide gel (on weeks 0, 1, 2) and nebulized 1 week (early phase) or 8 weeks (prolonged phase). Ovalbumin-exposed mice were treated with Tubastatin A Hcl or vehicle control. Airway inflammation was assessed by bronchoalveolar lavage fluid cell counts and HE staining of lung tissue sections. Airway remodeling was assessed by Alcian blue-Periodic acid Schiff staining and Masson trichrome staining. Airway hyperresponsiveness was assessed by plethysmography measurement of airway resistance. Results: Tubastatin A Hcl treatment relieved airway inflammation compared with vehicle treated mice ( $P < 0.05$ ), but its effect was worse than dexamethasone treatment. However, Tubastatin A Hcl treatment reduced the quantity of goblet cell ( $P < 0.05$ ), subepithelial collagen deposition ( $P < 0.05$ ) and attenuated airway resistance ( $P < 0.05$ ). Conclusion: These results demonstrate that treatment with HDAC6 inhibitors can reduce airway inflammation, airway remodeling and airway hyperresponsiveness, suggesting that blockade of HDAC6 may be a useful treatment for bronchial asthma.

# DAY 3 SATURDAY, 15<sup>TH</sup> NOVEMBER

## Smoking-Related Interstitial Lung Disease: New Ideas and New Entities

Andrew Churg

Canada

HRCT surveys have found evidence of interstitial lung disease (ILD) in 8 to 15% of smokers. The recent pathology literature has described a new form of ILD in smokers under the names “smoking-related interstitial fibrosis,” “airspace enlargement with fibrosis,” and “RBILD with fibrosis.” To avoid confusion with other forms of smoking-related ILD, we suggested that this lesion be called “respiratory bronchiolitis with fibrosis” (RBF). RBF is characterized pathologically by localized patches of quite marked subpleural paucicellular interstitial fibrosis mixed with emphysema and smoker’s macrophages. We have shown that this lesion is often visible on HRCT as distinctive subpleural upper or mid-zonal circumscribed patches of reticulation surrounding emphysematous spaces. Some cases also demonstrate patchy ground glass opacities. Pulmonary function tests typically show mild airflow obstruction, sometimes with a disproportionately decreased diffusing capacity. In a survey of 200 heavy smokers, we observed this lesion on HRCT in 7% of patients. The lesion was radiologically stable in 86% and showed mild progression in 14%. However, in no case did the patient develop a diffuse fibrosing lung disease. RBF is often mistaken pathologically and radiologically for a functionally impairing ILD. We propose that, like smoker’s respiratory bronchiolitis (RB), most cases of RBF have no functional consequences; but, analogous to RBILD, some patients with RBF have evidence (reticulation, decreased diffusing capacity) of a ILD; ie, RBFILD. RBFILD may account for a substantial proportion of the ILD seen in HRCT surveys of cigarette smokers. The relationship of RBILD and RBFILD to DIP will be discussed.

## Tuberculosis, Tobacco and Smoking Cessation

Asif Mujtaba Mahmud

Bangladesh

Tuberculosis and Tobacco are a deadly pair

Smokers are roughly twice as likely to become infected and develop active TB compared to nonsmokers. They are usually diagnosed at a more severe stage of the disease, since cough is often neglected as smoker's cough. Passive smoking increases the risk of TB, especially in children

Smokers with TB, have a poorer prognosis, a greater risk of relapse, and are less likely to comply with TB treatment. In addition, tobacco increases the risk of dying from TB. There is a "dose response" relationship between smoking and TB – that is, the more cigarettes smoked daily, or the longer duration someone has smoked, increases the vulnerability to TB

Smoking Cessation Reduces the Risk of TB

Tobacco control has been neglected as a means of reducing TB. The strong links between TB and smoking make smoking cessation imperative for TB patients as well as people at risk for TB. Proven tobacco control initiatives should be integrated within TB control strategies, especially in low-income and middle-income countries, where the tobacco industry is aggressively expanding its markets. Smokers who quit reduce both their risk of becoming infected with TB and dying from it.

The message for health care providers, particularly clinicians and policymakers, is clear: Aggressive and sustained tobacco control is important in achieving effective TB control. This should be a priority for governments in high burden countries who are fighting relentlessly to achieve the Millennium Development Goals.

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## Smoking-Related Diseases in Asia-Pacific: Current Data

**Chau NGO QUY, MD, PhD**

Vietnam

The Asia–Pacific region is home to a large heterogeneous population whose respiratory health is influenced by diverse social, economic and environmental factors. The most prevalent causes of respiratory morbidity and mortality are tobacco smoking, infection, and air pollution. Tobacco smoking is a significant contributor to respiratory ill-health and death in the Asia–Pacific region as it is worldwide. Tobacco use is one of the biggest contributors to the epidemic of non-communicable disease in the Western Pacific Region.

This review aims to summarize current respiratory health issues in the Asia–Pacific region including smoking-related diseases especially COPD, lung cancer, cardiovascular disease. Among the Asia Pacific Region’s developing countries, tobacco use adversely impacts five of the top 10 diseases and injuries: Cerebrovascular disease, lower respiratory infections, chronic obstructive pulmonary disease, ischemic heart disease... Among the developed countries tobacco ranks as the leading health risk factor of over 50% of the population attributable fraction for COPD and lung cancer. Tobacco use also plays a role in the causation of ischemic heart disease and cerebrovascular disease, which are two leading diseases that make up over 15% DALYs.



## Managing Respiratory Infection in ICU

Curtis N. Sessler, M.D., FCCP

USA

Respiratory infections are common in the ICU setting. There are a number of different aspects of the care of these patients that are important to consider and review. Many patients with community-acquired pneumonia require ICU hospitalization, particularly for the management of acute respiratory failure and/or septic shock. Pneumonia is the most common infectious cause of septic shock. There are important features of septic shock management, including antimicrobial therapy, shock assessment and treatment, and supportive care that will be discussed. Key aspects of mechanical ventilation of patients with severe pneumonia, including pneumonia-related ARDS are evolving and will be reviewed. Further, critically ill patients who are receiving mechanical ventilation can develop nosocomial infection – ventilator-associated pneumonia (VAP), which is associated with worse patient outcomes. There are important strategies designed to reduce the chances of developing VAP, which will be reviewed. The important role of multi-drug resistant microorganisms in VAP will be emphasized and the impact on antimicrobial management discussed. In summary, the speaker will address key concepts in the epidemiology, prevention, and management of respiratory infections in the ICU.

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## Electronic Cigarettes – An Unrecognized Threat

Dean E. Schraufnagel

USA

One of the most important health challenges of today is the non-communicable diseases, which kill more than 36 million people annually and cause untold suffering that stress the health care systems of all countries. Attenuating risks today from tobacco, excess alcohol, physical inactivity, and unhealthy diets will mean a healthier tomorrow. The United Nations deemed them so important that it held a high-level meeting on their prevention and control in 2011.

Of these risks, tobacco use is the most harmful and modifiable. Important gains have been made in tobacco control using strategies such as “denormalizing” its use, but a new challenge has arisen, electronic nicotine delivery systems. Heavily marketed worldwide as fashionable, their popularity has spread exponentially. E-cigarettes are unregulated, readily available to all ages, and produced in flavors that appeal to young people. These products have not been adequately studied for safety, their effects on the health of populations, or as smoking cessation devices.

The Asian Pacific Society of Respiriology and its partner organizations in Forum for International Respiratory Societies presented a joint statement in association with a follow-up high-level meeting of the United Nations on non-communicable diseases in July 2014. The statement advised that “The potential benefits of electronic cigarettes to an individual smoker should be weighed against harm to the population of increased social acceptability of smoking and use of nicotine...” It recommended that “Electronic nicotine delivery devices should be restricted or banned until more information about their safety is available. If they are allowed, they should be regulated as medicine or tobacco products.”

## TB Control and MDR-XDR Prevention in India

**Dr. V. K. Vijayan**

India

The estimated incidence of new tuberculosis cases in India is 2.2 million and the prevalence is 2.8 million cases. Approximately 5% of TB patients have an estimated HIV positivity. Drug resistant TB has been reported in 2.2% of new cases and 15% of previously treated cases. India accounts for about 23.3% of the global prevalence and is the highest TB burden country in the world. Considering the magnitude of the problem and to achieve a control, the Revised National TB Control Programme (RNTCP) was launched in 1997 in India and RNTCP has expanded across the country in a phased manner. The objectives of the programme are to achieve and maintain cure rate of at least 85% among New Sputum Positive (NSP) patients and to achieve and maintain case detection of at least 70% of the estimated NSP cases in the community. The current focus of the programme is on ensuring "universal access" to good quality early diagnosis and treatment for all TB patients. The program is covering the entire nation since March 2006 reaching over a billion population (1164 million). Annually more than 1.5 million TB patients are placed on DOTS treatment under RNTCP. In 2011, RNTCP has achieved the new sputum positive case detection rate of 71% and treatment success rate of 88% which is in line with the global targets for TB control. Throughout the country a network of more than 600,000 trained directly observed treatment (DOT) providers provide directly observed treatment (DOT) to more than a 1.5 million patients diagnosed as TB each year. The success of the RNTCP is the result of a comprehensive and appropriate strategy, systematic and timely planning, robust systems of quality assurance for diagnosis and treatment, methodological logistics management, well defined human resource development strategy including trainings, clear defined technical and operational guidelines and a built in supervision and monitoring mechanism. All states in India are implementing the "Supervision and Monitoring strategy", detailing guidelines, tools and indicators for monitoring the performance from the peripheral health institution (PHI) level to the national level. Regular internal and external evaluations ensure quality program implementation. The program is focusing on the reduction in the default rates among all new and retreatment cases and is undertaking steps for the same. quality assured anti-TB drugs for the full course of treatment to the patients through patient wise boxes. Decentralized treatment is provided to the patients as near to their home as possible. The programme is in the process of establishing a network of accredited culture and drug susceptibility testing (DST) Intermediate Reference Laboratories (IRLs) across the country in a phased manner for diagnosis and follow up of MDR TB patients. Multi Drug resistant TB (MDR TB) services have been initiated in all states in the country. RNTCP has involved NGOs and private practitioners, corporate hospitals and medical colleges as part of Public Private Mix (PPM) for successful implementation of the programme.

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The positive impact of the programme can be seen from the fact that TB mortality in the country has reduced from over 39 per hundred thousand population in 1990 to 29 hundred thousand population in 2010. The prevalence of TB in the country has reduced from 459 per hundred thousand population in 1990 to 256 per hundred thousand population by the year 2010 (WHO Global TB Report, 2011). As per the National Strategic Plan of Government of India to be implemented during 2012-2017, the vision is to have a “TB-free India” with the goal of Universal Access to quality TB diagnosis and treatment for all pulmonary and extra pulmonary TB patients including drug resistant and HIV associated TB.

## Moving COPD Beyond FEV1: Role of Pulmonary Imaging

Harvey O Coxson

Canada

Forced expiratory volume in 1 second (FEV1), measured using spirometry, provides a straightforward, widely-available and inexpensive global measurement of airflow limitation and lung function. For decades, FEV1 has remained the main intermediate endpoint used in research studies and for the development of new COPD therapies. Not surprisingly, therapies that acutely improve FEV1, dominate as COPD therapies. However, in COPD patients, the relationship of FEV1 with symptoms and outcomes such as exacerbations and mortality is weak, and importantly, FEV1 does not take into account the heterogeneity of COPD nor its different phenotypes. Thoracic imaging provides a way to quantify airway remodeling, emphysematous destruction, regional ventilation abnormalities (ventilation defects) and gas trapping in ex-smokers in whom FEV1 may be normal and in COPD patients with very modest lung function deterioration. In individual patients and in COPD cohort studies, thoracic imaging using x-ray computed tomography (CT), and magnetic resonance imaging (MRI) (conventional  $^1\text{H}$  as well as hyperpolarized noble gases such as  $^{129}\text{Xe}$ ,  $^3\text{He}$ ) and optical coherence tomography can be used to directly visualize the structural and functional consequences of COPD and thus provide a clearer picture of COPD mechanisms, disease progression and response to therapy. We briefly describe pulmonary imaging methods that provide a way to visualize and quantify with high spatial and temporal resolution, regional ventilation abnormalities, gas trapping, emphysema and airway remodeling in COPD. Finally we discuss the implications of recent imaging findings and their impact on future biomarker and therapy research aimed at improving COPD outcomes.



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## **What's New in Pulmonary Hypertension; How Do You Manage a COPD Patient Associated with Pulmonary Hypertension?"**

**Hiroshi Kimura, MD**

Japan

In 2013 the most recent world conference on pulmonary hypertension (PH) was performed in Nice. The classification of PH was updated and it remained to minor revision. The concept of classification would be basically considered that the same group has the common pathologic and hemodynamic characteristics and also common therapeutic approaches. Aspire Registry, however, shows that outcomes and characteristics differed not only between PH groups but within PH groups. Three-year survival for PAH (Group 1), PH with left heart disease (PH-LHD; Group 2), PH with chronic lung disease (PH-CLD; Group 3), and chronic thromboembolic PH (CTEPH; Group 4) was 68%, 73%, 44%, 71%, respectively. Even within groups, PAH is a heterogeneous condition; 3-yr survival for PAH with systemic sclerosis (PAH-SSc), idiopathic PAH (IPAH), and Eisenmenger's PH was 52%, 63% and 85%, respectively. It is conceivable that the different forms of PH present with either a predominance of pulmonary arterial remodeling such as IPAH or vein remodeling such as pure pulmonary veno-occlusive disease (PVOD) or a contribution of both such as SSc-PAH. PVOD/PCH (pulmonary capillary hemangiosis) remain difficult disorders to classify since they share the same characteristics as IPAH but exhibit many differences, so PVOD/PCH were classified to Group 1' in the world conference in Dana Point in 2008. Survival in PH-CLD was most poor among all groups. Moreover, Group 3 was inferior and Group 4 was superior compared with Group 1. In order to discuss therapeutic strategies for PH-CLD, one severe PH case will be presented in the session.

## Redefining COPD

Jørgen Vestbo

Denmark

The well-known heterogeneity of COPD has led to the concept of phenotypes as a way of understanding and treating separate components of the disease separately – a way of introducing precision medicine into COPD.

A phenotype of COPD was defined by Han et al as “a single or combination of disease attributes that describe differences between individuals with COPD as they relate to clinically meaningful outcomes (symptoms, exacerbations, response to therapy, rate of disease progression, or death).”

Some of the most important phenotypes of COPD are the frequent exacerbator, chronic bronchitis, having systemic inflammation and being a rapid decliner. Exacerbations are frequent in COPD but there is ample evidence that some patients have frequent exacerbations and some rarely have any. Predictors are available and specific treatments aimed at reducing the burden of exacerbations. Future treatments are likely to be guided by biomarkers. Chronic bronchitis is strongly associated with smoking and in younger patients a predictor of poor prognosis; specific treatments are lacking. Systemic inflammation is still poorly understood but relates to both exacerbations and risk of patterns of comorbidities.

I propose that phenotypes should be evaluated according to underlying activity of COPD. Biomarkers for this are urgently needed but required for further progress in the understanding and management of COPD.

# DAY 3 SATURDAY, 15<sup>TH</sup> NOVEMBER

## Identifying Early Signs and Symptoms of impending Asthma Exacerbations in Children

Katherine Rivera-Spoljaric, MD, MSCI  
USA

Despite advances in the treatment of asthma, exacerbations remain common. The National Heart Lung and Blood Institute's National Asthma Education and Prevention Program Guidelines recommend early treatment of asthma exacerbation as "key in management", but do not provide specific information or guidance about signs and symptoms that may present before lower respiratory symptoms. We aimed to describe early signs and symptoms of an impending asthma exacerbation in children, and to study how these symptoms vary during a well-period and illness periods.

To collect and study these signs and symptoms we performed focus groups followed by administration of surveys with a 57-symptom inventory (Asthmatic Child Early Signs and Symptoms (ACcESS) inventory) to 200 caregivers of children aged 1-12 years with physician diagnosed persistent asthma recruited from our pediatric asthma clinics. We then recruited caregivers (same inclusion criteria) to perform daily diary cards based on the symptom inventory during a 4-week well-period (ACQ<1.5) in the summer (n=19) and a 16-week winter period (n=27) where there was likelihood of loss of control (>2 consecutive days of increased LR symptoms).

A wide variety of symptoms were reported as preceding an asthma exacerbation. We categorized these into three main categories: non-respiratory (NR), upper respiratory (UR), and lower respiratory (LR). NR symptoms were further subdivided into changes in eating habits, appearance, activity, and behavior. The most common NR symptoms included decreased activity (31%), decreased appetite (25%), irritability (20%), difficulty falling asleep (19%), and paleness (11%). A total of 25% of caregivers reported runny nose as an early symptom and 68% reported cough. The very first symptom was NR in 25%, UR in 15%, and LR in 60%. Ninety-seven percent of caregivers were able to identify a first symptom, and 89% reported that this first symptom was present always or almost always. There was no difference in symptom reporting or symptom reporting category (NR, UR, LR) according to child's age, gender, ethnicity, race, or use of asthma controller, or the caregiver's age, gender, ethnicity, race, insurance type, income, and education.

During the 4-week well period, there was little symptom variability across all symptom categories. During the 16-week winter period, overall there was a trend for increased likelihood of reporting NR symptoms in the 3 days before loss of control episodes, including changes in behavior, mood, and appearance as compared to period where asthma was controlled. Some NR symptoms were consistently increased over the 3-4 days prior to an episode, with others increased only the day prior to an episode of loss of control. Overall, UR symptoms were not increased during the days prior to a loss of control episode.

In conclusion, caregivers identify a wide variety of signs and symptoms as occurring before an asthma exacerbation. These include non-respiratory symptoms and upper respiratory symptoms, in addition to the typical lower respiratory symptoms of asthma. These symptom patterns are not different by specific parental or child characteristics. The symptoms that were significantly increased before episodes of loss of control were mostly NR in nature. Furthermore, these symptoms do not significantly vary during a well period, and some children may exhibit symptom patterns with recurrent illnesses. A study is currently underway to assess whether augmentation of inhaled corticosteroids triggered by an ACcESS inventory-guided asthma management plan will change asthma morbidity by decreasing severity of asthma exacerbations.

# DAY 3 SATURDAY, 15<sup>TH</sup> NOVEMBER

## A Challenge to Overcome Drug Resistance in Lung Cancer

**Kazuhisa Takahashi**

Japan

Lung cancer is the leading cause of cancer death worldwide. Despite progress in the use of platinum-based chemotherapy, the median survival time of patients with inoperable non-small cell lung cancer (NSCLC) remains poor. One recent advancement in the treatment of inoperable lung cancer is the development of molecular targeting drugs, including gefitinib, erlotinib and afatinib, that target the epidermal growth factor receptor (EGFR). However, most patients with NSCLC harboring EGFR activating mutations who respond to EGFR-TKI become resistant to treatment nine to 14 months after the start of therapy via various molecular mechanisms, including gatekeeper mutations in EGFR, such as the T790M mutation, in which threonine (T), at the 790th amino acid, is converted to methionine (M), cMET amplification, hepatocyte growth factor (HGF), overexpression and PTEN downregulation. Several other molecular mechanisms of acquired resistance have been identified, including the epithelial-mesenchymal transition (EMT) and involvement of cancer stem cells (CSCs). We have intensively investigated precise EGFR-TKI resistance mechanisms that function independent of T790M and recently clarified several novel potential mechanisms. In this lecture, I would like to review the mechanisms underlying EGFR-TKI resistance and introduce our recent novel findings, which may identify potential novel molecular targets in patients with advanced NSCLC.



## Lung Cancer Screening program: Any Good News?

**Kazuma Kishi**

Japan

In the 1990's, investigators first in Japan and then the United States began studying low-dose CT screening for lung cancer. These low-dose CT trials were designed as single-arm studies, which led to the detection of more early stage lung cancers. However, the single arm studies did not determine whether low-dose CT screening affected lung cancer mortality due to lead-time, length-time, and over-diagnosis biases. To answer the question whether screening with low-dose CT reduces lung cancer mortality, several randomized control trials have been conducted. Findings of the US National Lung Screening Trial showed a 20% reduction in lung cancer mortality and a 6.7% decrease in all-cause mortality. Recently, the US Preventive Service Task Force recommended that screening should be implemented. However, many questions remain, including whom to screen, how often, and for how long. Furthermore, costs and effects on the health care system remain unclear. In this presentation, I would like to briefly review the history of lung cancer screening, discuss the results of the National Lung Screening Trial, and address some of the unanswered questions.

# DAY 3 SATURDAY, 15<sup>TH</sup> NOVEMBER

## **Pulmonary Hypertension Associated with Pulmonary Disease/Hypoxia**

**Masayuki Hanaoka**

Japan

The mean pulmonary arterial pressure (mPAP) in patients with COPD is an extent of more than 20 mmHg at rest, of which pulmonary hypertension is mild or even absence. COPD cases corresponding to severe pulmonary hypertension have been reported to be at most 1%; it is not a small number on estimation that the number of patients with COPD is approximately 200 million in the world. Comparing with the forced expiratory volume in 1 second and lung diffusion capacity, the severity of pulmonary hypertension is the crucial factor in determination of the prognosis. However, since the airflow obstruction does not correlate with the severity of pulmonary hypertension, when the respiratory functions could not explain the reduction of exercise tolerance, it is very important to examine the pulmonary circulation system. It is lack of evidences of treatment for pulmonary hypertension associated with COPD by the medicines those are approved for clinical management of pulmonary arterial hypertension (PAH). Because PAH therapeutic agents may contribute to mismatch of ventilation-perfusion, it is not recommended for the administration of pulmonary hypertension associated with COPD at present time. However, as the clinical course of a number of severe cases of pulmonary hypertension due to COPD is similar to that of PAH, PAH therapeutic agents might be considered reasonably in the treatment of severe pulmonary hypertension associated with COPD.

## **Bronchial Thermoplasty in the Management of Asthma**

**Michael E. Wechsler, MD**

USA

Asthma remains one of the most common diseases worldwide and results in significant societal healthcare costs and morbidity and mortality to those afflicted. In spite of currently available medications, many asthmatics have severe disease with debilitating symptoms and/or life threatening exacerbations. Bronchial thermoplasty (BT) is a bronchoscopic device-based therapy that utilizes thermal energy to disrupt airway smooth muscle. In addition to improving asthma related quality of life, BT has been shown to reduce exacerbations and improve important measures of asthma control with good safety and efficacy data, now demonstrated out to 5 years.

# DAY 3 SATURDAY, 15<sup>TH</sup> NOVEMBER

## Recent Strategies for Chronic Thromboembolic Pulmonary Hypertension

**Nobuhiro Tanabe**

Japan

Chronic thromboembolic pulmonary hypertension (CTEPH) is a form of pulmonary hypertension caused by non-resolving thrombi in the pulmonary arteries. It was previously believed that recurrent pulmonary embolism might be a major cause of CTEPH. However, recent evidence suggests that venous thromboembolism might be a trigger, and in situ thrombosis in the pulmonary artery and small vessel vasculopathy similar to pulmonary arterial hypertension could be important for progression of CTEPH. In addition CTEPH is more prevalent in women in Japan, in contrast to Western countries. A Japanese multicenter study reported that a form of CTEPH related to deep vein thrombosis is associated with HLA-B\*5201. Pulmonary ventilation-perfusion scans are necessary for screening of CTEPH. Furthermore, contrast CT scans can detect segmental emboli and are useful for the differential diagnosis of pulmonary vascular disease. However, pulmonary angiography is still the gold standard for assessment of surgical accessibility and small vessel disease. The prognosis of this disease was poor in the 1980's (5-year survival ~40%). Pulmonary endarterectomy with median sternotomy under intermittent deep hypothermia has decreased operative mortality to <5% at Chiba University as well as other expert centers. Advances in balloon angioplasty techniques have resulted in marked improvement in pulmonary hemodynamics, quality of life and survival for inoperable patients in Japan. A soluble guanylate cyclase activator (riociguat) has just become available for patients with inoperable or persistent/recurrent pulmonary hypertension after surgery. It must be emphasized that this disease is treatable once patients with dyspnea on exercise have been accurately diagnosed with CTEPH.

## **Diversity and Heterogeneity in The Respiratory Problems in Asia Pacific: The Global Burden of Respiratory Disease**

**Norbert Berend**

Australia

Respiratory diseases make up four of the top ten causes of death globally (lower respiratory infections, COPD, tuberculosis and lung cancer). There is a worldwide shift from infectious to non-communicable disease and the World Health Organisation lists respiratory disease as one of the four major non-communicable diseases for the coming decade. In Asia Pacific chronic lung disease and lung cancer are fuelled by high smoking prevalence in many countries as well as high levels of air pollution, while tuberculosis and lower respiratory infections remain major problems. Air pollution is now recognised to be a major cause of cardiovascular disease, stroke and respiratory disease. The shift to less polluting nuclear power in the region has slowed as a result of the Fukushima nuclear power plant disaster. Childhood deaths in Asia Pacific lead the world although rapid progress in lowering childhood and maternal mortality is being made. There is mixed news about improvements in extending life expectancy with large variations across the region since 1990. With reduction in mortality and an ageing population there is an increase in the burden of disability with its resultant increased costs of healthcare. Respiratory disease is deserving of greater attention by governments and health care organisations.



# DAY 3 SATURDAY, 15<sup>TH</sup> NOVEMBER

## Assessment of Quality of Life in COPD

**Paul Jones**

UK

COPD is a complex condition with multiple pathogenic pathways causing symptoms that are directly related to the lungs such as breathlessness and cough, and other symptoms unrelated to the chest, such as fatigue, sleep disturbance, muscle weakness and depression. Whilst breathlessness and exercise limitation are important determinants of impaired health and poor quality of life, they account for less than half of the poor health status that patients report. Comprehensive assessment of COPD requires health status measurement.

The older questionnaires such as the St George's Respiratory Questionnaire (SGRQ) were too complex to use in routine practice, but the COPD Assessment Test (CAT) was designed for use in routine clinical care and only takes 2-3 minutes to complete. There is good evidence that the CAT performs the same way in Asia as in the rest of the world.

A CAT score of 10 appears to be a reasonable threshold for starting regular bronchodilator therapy and score >20 in patients with a history of exacerbations predicts a high probability of another exacerbation within a few weeks. That study was performed in Asia. A recent review provides a very comprehensive review of the performance of the CAT (Gupta et al *Eur Respir J* 2014; 44: 873).

An analysis of clinical trials using the SGRQ has shown that patients in low-medium socio-economic countries have very large improvements in health status just by entering a clinical trial, even if they receive placebo. However they also show a similar or slightly greater improvement with treatment compared to patients in high-income countries.

In summary, sophisticated health status measurement can be performed routinely in the clinic, more simply and quickly than spirometry.

## Dynamic Change of House Dust Mites and Its Components-sIgE&slgG4 in Specific Immunotherapy

Pei-yan Zheng, Bao-qing Sun\*, Ni-li Wei, Hui-min Huang, Guang-qiao Zeng

China

**Objective:**This project aimed to analyze the levels of specific IgE (sIgE) to Dermatophagoides pteronyssinus (Der p) and its main components including Der p1, Der p2 and Der p10 before specific immunotherapy (SIT), and observe the dynamic change of sIgE and slgG4 to Der p, Der p1 and Der p2 after SIT, so as to evaluate the significance of applying of SIT of Der p in clinical diagnosis.

**Methods:**The dynamic change of the sIgE, slgG4 and correlation between each antibody before and after SIT were analyzed.

**Results:**The positive rate of serum sIgE in vitro to Der p10 from patients with mild-moderate rhinitis or coexisting asthma was only 1.6%. Moreover, there was no significant difference on the levels of serum sIgE and slgG4 to Der p10 before and after SIT ( $p<0.05$ ).After 17 weeks of SIT the levels of serum sIgE to Der p, Der p1 and Der p2 increased continuously ( $t=4.78, 6.21, 4.21, p<0.01$ ).However, along with SIT, sIgE decreased significantly again after 57 weeks of SIT, which resulted in no difference as pre-SIT.The levels of slgG4 to Der p, Der p1 and Der p2 were dynamically observed and recorded in each of the three periods. The levels of slgG4 increased significantly along with the process of SIT. After 57 weeks of SIT, the increasing range of Der p slgG4 reached to the maximum, followed by Der p1 and then Der p2.In each of the three periods, there was a significant correlation between sIgE and slgG4 to Der p, Der p1 and Der p2 representing on the change of increasing range ( $p<0.01$ ). And the order of the correlative degree of the sIgE in each period was: Der p and Der p1 reached the highest, followed by Der p and Der p2, and the lowest were Der p1 and Der p2.There was also a correlation between sIgE and slgG4 to Der p and Der p2 ( $p<0.05$ ).

**Conclusions:**The results showed that SIT was a dynamic immune process and sIgE and slgG4 to Der p and its components reflected the immune status of the immune process.

# DAY 3 SATURDAY, 15<sup>TH</sup> NOVEMBER

## **TB and HIV: The Perfect Storm**

**Prof. Charles Y. Yu, MD,FACP,FPCP,FPCCP**  
Philippines

This presentation gives us a overview of the diagnosis and management of TB and HIV when they overlap in patients as well as MDR- TB and HIV. There were an estimated 194,000 TB patients with HIV in the Asia pacific region in 2012. An estimated 11-13% of new cases of Tb were HIV positive and ,people living with HIV (PLWH) have a 20 fold risk of developing TB in their lifetimes.

Discussions will concentrate on the peculiarities attendant to dealing with both TB and HIV, including timing of treatments, diagnostic issues, drug interactions, updates on diagnosis and management as well as treatment recommendations when dealing with drug sensitive as as drug resistant TB in the HIV patient. Approaches to optimize treatment outcomes will be highlighted.

## **Advances in Pneumothorax Management: COPD vs Tuberculosis Pneumothorax**

**Pyng Lee MD FCCP**  
Singapore

Spontaneous pneumothorax (SP) refers to air in the pleural cavity which occurs without preceding trauma or obvious precipitating cause, and is sub-divided into primary and secondary. Secondary spontaneous pneumothorax (SSP) occurs as a complication of underlying pulmonary disorder most often chronic obstructive pulmonary disease (COPD) while primary spontaneous pneumothorax (PSP) affects an individual without clinically apparent lung disease. Annual incidence of SSP is 6.3/ 100,000 population in males and 2/ 100,000 in females while PSP affects 18-28 males/100,000 population and 1.2-6 females/ 100,000. Together they account for 130 million dollars in health care expenditures. The course of SP is variable with recurrence rate 25-54% and presence of lung disease is a major determinant. The American College of Chest Physicians and BTS guidelines recommend VATS staple bullectomy and parietal pleural abrasion for SSP since surgical options are more effective. Medical chemical pleurodesis with tetracycline and its derivatives or graded talc may be appropriate if the patient declines or is unfit for surgery. Management of persistent air leak due to COPD and PTB with autologous blood patch and potential role conferred by bronchoscopic valves will be discussed.

# DAY 3 SATURDAY, 15<sup>TH</sup> NOVEMBER

## Update in Pleural Disease

**Richard W. Light, M.D.**

USA

In recent years it has been shown that a pleural fluid N-terminal BNP level greater than 1500 is diagnostic of congestive heart failure. Serum N-terminal BNP levels are as efficient as pleural fluid N-terminal BNP levels in making the diagnosis.

The approach to patients with undiagnosed after a diagnostic thoracentesis had typically been to perform thoracoscopy. However, recently it has been shown that image guided pleural biopsy (CT scan or ultrasound) in patients with pleural thickening or pleural nodules is almost effective as thoracoscopy. The advantage of image guided pleural biopsy is that it is less invasive.

The management of patients with complicated parapneumonic effusions is difficult. It has now been shown that the intrapleural administration of 5 mg DNase and 10 mg tPA twice a day for three days increases the rate at which the pleural fluid disappears and also decreases the duration of hospitalization and the need for surgical intervention. The intrapleural injection of a fibrinolytic itself such as tPA is ineffective in treating complicated parapneumonic effusions.

Patients with malignant pleural effusions often have the quality of their lives decreased by dyspnea. Recently the insertion of an indwelling catheter by which the pleural fluid can be intermittently drained has become accepted as a treatment for malignant pleural effusions. The advantages of the indwelling catheter as compared with pleurodesis are that the patient does not need to be hospitalized and the total number of hospital days associated with management of the pleural effusion is reduced.



## Improving Outcome of CAP

Ronald Grossman MD FRCPC FCCP FACP

Canada

Community-acquired pneumonia (CAP) is a common infection and is associated with potentially lethal consequences. The most important bacterial pathogen in CAP is *Streptococcus pneumoniae*, though a variety of other organisms, including *Haemophilus influenzae* and the atypical pathogens, *M. pneumoniae* and *Legionella pneumophila*, are also implicated. The majority of patients with mild-to-moderate CAP are treated in the community setting with empiric antimicrobial therapy. Patients with more serious disease or who are elderly or have co-morbidities may be hospitalized, though even in this setting, empiric antimicrobial therapy is usually started. A number of severity scoring systems have been developed to assist in “the admit to hospital” decision and “the transfer to ICU” decision. The CURB65 rule and the PORT score have been validated in a variety of clinical settings. Biomarkers such as procalcitonin have been demonstrated to shorten antibiotic duration. All admitted patients should receive their first dose of antibiotic therapy as soon as possible after arrival to the hospital. While 4 hours has been the recommended standard, it is not clear that every patient must be treated that quickly because the diagnosis of pneumonia often takes longer than 4 hours to establish and treatment should not be started until other diagnostic possibilities have been excluded. Guideline directed therapy improves outcomes especially mortality. Treatment failure rates even with guideline-driven empiric therapy have been reported as high as 15%. Resistant and unusual microorganisms and noninfectious causes are usually responsible. Knowledge of local resistance rates may allow better initial empiric therapy choices. Shortening the course of antibiotic therapy and rapidly switching from intravenous to oral therapy, and discharging patients from hospital when they have stabilized, have all been demonstrated to shorten hospital without compromising outcomes. Pneumococcal vaccination, particularly the new 7-valent conjugated vaccine offered only to children, reduces invasive pneumococcal disease in vaccinated children and in unvaccinated adults via herd immunity. The older 23 valent polysaccharide vaccine is not effective in preventing pneumonia but may reduce invasive pneumococcal disease. The new 13-valent protein conjugate vaccine is effective in reducing pneumonia in the elderly and may supplant the 23-valent polysaccharide vaccine for routine use in adults.

# DAY 3 SATURDAY, 15<sup>TH</sup> NOVEMBER

## Pulmonary Infection in Chronic Respiratory Disease

Ronald Grossman MD FRCPC FCCP FACP

Canada

The convergence of emerging resistance, newly recognized pathogens, altering healthcare systems, and changing patient type yields a more complex situation for today's prescriber. The need to treat more resistant pathogens in a rapid manner reduces the empiric options available. Treatment failures with first line antibiotics are common especially among patients with chronic respiratory exposed frequently to antibiotics. COPD patients are at risk of frequent exacerbations if they have more severe disease (FEV1 usually < 50% predicted), a history of frequent exacerbations (usually  $\geq 2$ /year) and symptoms of chronic bronchitis. The strongest predictor of an AECOPD in a given year is the presence of an exacerbation in the previous year. Elevated systemic biomarkers also predict frequent exacerbations but treatment with a statin does not change this observation. Antibiotics reduce the likelihood of clinical failure and respiratory fluoroquinolones appear to be the most potent of treatment options. COPD patient exhibiting severe airflow obstruction, isolation of a potentially pathogenic microorganism, or at least one hospital admission due to COPD exacerbation in the previous year are very likely to have concomitant bronchiectasis. Preventive therapy with influenza or pneumococcal vaccines is not very effective. Chronic antibiotic administration may be necessary in some selected cases.

Non-cystic fibrosis bronchiectasis is the best example of pulmonary infection in chronic respiratory disease. Most cases are either idiopathic or post-infectious. H influenzae and Pseudomonas are the most common isolates but a broad variety of organisms are found in the bionome. Treatment of the underlying condition, promotion of bronchial hygiene, identification of acute exacerbations and administration of antibiotics, suppression of the microbial load, reduction of the excessive inflammatory response, and control of bronchial hemorrhage are the basic principles of management. Patients chronically colonized by Pseudomonas have a faster decline in annual FEV1 compared to those not colonized. Long term oral and/or inhaled antibiotics are gaining an increasing role in the management of these patients. Chronic suppressive therapy with inhaled antibiotics looks especially promising.

## **Addressing the Significant Burden of Pneumococcal Disease in Elderly – Investigating the Impact of Co-morbidities and Risk Factors**

**Sita Andarini**  
Indonesia

Lower respiratory tract infection is a leading cause of death worldwide in any age group. Older adults are at increased risk of invasive pneumococcal disease, due to contributing immunologic factors, such as changes in the aging immune system (immunosenescence). These changes include complex changes in the innate immunity and adaptive immunity. Older age, current smoking, diabetes mellitus, congestive heart failure, lung cancer, COPD, asthma, were independently associated with and increased risk for all causes of community acquired pneumonia. In Asia Pacific, the common comorbidities associated with CAP include bronchopulmonary diseases, smoking history, cardiovascular disorders, malignancy and neurologic disorders. Comorbidities and risk factors add mortality rate in elderly. In the current presentation, the impact of comorbidities and risk factors on the burden of pneumococcal disease in elderly are discussed.

# DAY 3 SATURDAY, 15<sup>TH</sup> NOVEMBER

## Infection in Interstitial Lung Disease

**Sita Andarini**

Indonesia

Idiopathic Pulmonary Fibrosis is a disease characterized by progressive deterioration of lung function which leads to respiratory failure. IPF has median survival time of 3 years after diagnosis, –worse than survival of some cancer. IPF occurs among elderly, some with history of smoking. Environmental factor might induce IPF in susceptible individual. Respiratory infection has been sought to have role in disease pathogenesis and progression. Viruses might play in part in acute exacerbation, and increase in mortality. Infection in IPF showed related with increase morbidity and mortality. In this presentation, the role of infection in disease pathogenesis and progression in IPF were presented.

## **Comorbidities in COPD**

**Suzanne Hurd**

USA

COPD patients have been shown to have a higher prevalence of osteoporosis, anxiety/panic attacks, heart trouble, heart attack, and heart failure; COPD patients with comorbid conditions have been shown to have poor clinical outcomes. In this presentation, the recommendations from the Global Initiative for Chronic Obstructive Lung Disease (GOLD) for treatment of COPD of patients with comorbid conditions will be presented. The recommendations for diagnosis of Asthma COPD Overlap Syndrome will also be discussed.



# DAY 3 SATURDAY, 15<sup>TH</sup> NOVEMBER

## Latest Update on Interventional Bronchoscopy

Teruomi Miyazawa

Japan

Functional Bronchoscopy and Interventional Pulmonology, which includes laser ablation and stenting, bronchoscopic lung volume reduction (BLVR) with bronchial valve or coil for COPD, bronchial thermoplasty for bronchial asthma and targeted lung denervation for COPD. It is important to take into account the respiratory physiological characteristics of individual patients. For example, in the case of airway stenosis, an appropriate stent must be accurately placed at the flow-limiting segment to obtain optimal results. In this sense, pathological conditions can be evaluated in real-time by measuring the airway pressure gradient between the airway pressures at both sides of the stenosis using a bronchial catheter during bronchoscopy.

In addition, collateral ventilation positive is problematic for BLVR with bronchial valve placement for COPD. In this regard, interlobar collateral ventilation evaluation using the Chartis System is useful for the preoperative selection of patients. In cases of heterogeneous upper lobe emphysema without collateral ventilation, valve placement is most effective.

Ideal functional bronchoscopy is defined as bronchoscopy with an ultrathin catheter bronchoscope equipped with sensors for measuring pH, temperature, O<sub>2</sub>, CO<sub>2</sub>, airflow, pressure, etc. Local lung function is examined by inserting the bronchoscope into the segmental or sub-segmental bronchi, smaller airways, or peripheral bronchi. An O<sub>2</sub> sensor can be used for evaluating segmental bronchial ventilation, and a CO<sub>2</sub> sensor can be used for evaluating the perfusion pulmonary blood flow of segmental lobar bronchi based on CO<sub>2</sub> output.

## Preventing Chronic Lung Infections: Lessons from Cystic Fibrosis

Thomas Ferkol, MD

USA

Cystic fibrosis (CF) is the most common, life-shortening inherited disease of Caucasians. An autosomal recessive defect, occurring in approximately 1 in 3500 live births in the United States based on data from neonatal screening, the life expectancy of a child born with CF has gradually improved and is now over 38 years. Cystic fibrosis is caused by defects in the cystic fibrosis transmembrane conductance regulator (CFTR), a cyclic adenosine monophosphate (cAMP)-regulated chloride channel expressed on the surface of airway epithelial cells and the serous cells of the submucosal glands. The CFTR is functionally linked to other apical chloride channels, and aberrant expression or function of the CFTR in the airway leads not only to reduced chloride conductance but dysregulation of epithelial sodium channel activity. Failure of chloride secretion and massive sodium hyperabsorption results in dehydration of the airway surface. The dessicated secretions obstruct the airways and reduce mucociliary clearance, thus permitting bacterial infection to become established and allowing the inflammatory response to be amplified. Other gaps in innate defenses, including impaired antibactericidal activity, contribute to bacterial persistence and chronic infection in the CF airway.

Cystic fibrosis patients acquire bacterial pathogens in an age dependent manner that over time will chronically infect their airways. Initially, *Staphylococcus aureus* is isolated from the lungs of infants and young children, and its prevalence, particularly methicillin-resistant strains, has greatly increased. *Pseudomonas aeruginosa* emerges as the predominant organism over time and most CF children have had lung infection with *P. aeruginosa* by 3 years of age. Approximately 80% of CF adults in the United States are persistently infected with *P. aeruginosa*. Acquisition of *S. aureus* and *P. aeruginosa* from the lungs of CF patients is associated with a poorer prognosis, but recent studies treating initial acquisition of *P. aeruginosa* have shown that it can be eradicated with aggressive antibiotic therapy. While patients reacquired the organism later, the realization that early *P. aeruginosa* infection can be effectively eradicated has changed treatment strategies for CF. No longer are antibiotics solely used to treat symptomatic disease; now they are used to treat early positive *P. aeruginosa* cultures, even in the absence of symptoms, though data showing that early eradication impacts later lung function are lacking at this time.

# DAY 3 SATURDAY, 15<sup>TH</sup> NOVEMBER

## IPF: What We Have Learned

**Toshihiro Nukiwa**

Japan

While the pathogenesis of idiopathic pulmonary fibrosis (IPF) is yet unclear, we have questioned and learned considerable basics on the clinical diagnosis and therapeutic responses. Let us think over what we have learned in these 15 years. The first step was to move to positive fibrosis histologic photos from X-ray negative films. VATS procedure enabled less invasive sampling. Development of HRCT also enabled the diagnosis of typical IPF without surgery. This accumulation of experiences led to the Consensus Statement of ATS/ERS in 2000. What was the second step? When Japanese doctors started the clinical trial of pirfenidone in patients with IPF, we were fumbling to write a protocol. What should be the primary endpoint? What kind of patients should be enrolled? Because we had a 10-year experience of using staging system in IPF, we planned to enroll patients with slowly progressive (i.e. stage II to III). We know that the difference between the pirfenidone and placebo arms should be significant in a period of 52 weeks, and if pirfenidone has a power to slow the fibrosing course, proper patients will be in stages II or III. This strategy was proved right and we succeeded in the phase III clinical trial. Pirfenidone was approved in 2008 in Japan, and is now on the market all over the world through CAPACITY and ASCEND studies. More recently, using nintedanib, a tri-kinase inhibitor, doctors succeeded in TOMORROW and IMPULSIS studies. FDA has just approved it. Now we have two drugs with different mechanism for patients with IPF. The next challenge will be the timing of drug administration and choice of combination. We are in the exciting time of novel intervention for IPF.

## Acute Exacerbation of IPF

Yasuhiro Kondoh

Japan

The clinical course of idiopathic pulmonary fibrosis (IPF) is usually chronic, but some patients may experience episodes of acute respiratory worsening. Although these episodes may occur secondary to common conditions such as pneumonia, pulmonary embolism, pneumothorax or cardiac failure, the term acute exacerbation of IPF (AE-IPF) has been used when a cause cannot be identified for the acute respiratory worsening. Because AE-IPF is a crucial and lethal complication of IPF, its prevention and better management is an urgent need.

AE-IPF is a diagnosis of exclusion, so diagnostic approach includes physical examination, laboratory tests, biomarkers, HRCT, BAL, and echocardiogram. Possible pathophysiology for AE-IPF are accelerated primary disease process, and occult etiology. BAL and lung surgery are known as precipitating factors for AE-IPF. PaO<sub>2</sub>/FIO<sub>2</sub>, CRP, LDH, KL-6, % lymphocytes in BAL, CT patterns, and CT extent are reported to be prognostic factors.

Corticosteroids with or without immunosuppressant are commonly prescribed and are thought as mainstream therapy. Noninvasive positive pressure ventilation should be a first-line mechanical ventilation, and invasive mechanical ventilation may be a reasonable intervention in a minority. Because of poor prognosis, experimental therapies such as low molecular weight heparin, recombinant thrombomodulin, and PMX hemoperfusion therapy were reported to be possible candidate therapies. Extracorporeal membrane oxygenation has been used mainly as a bridge to transplantation.

# DAY 4 SUNDAY, 16<sup>TH</sup> NOVEMBER

## What is in the Pipeline in Asthma Therapy?

**Christine Jenkins**

Australia

Despite the greater understanding of refractory or difficult to treat asthma that has come with phenotyping, asthma remains a highly treatable condition. In the majority of people, currently available treatments prevent exacerbations, control daily symptoms and maintain lung function, reduce airway inflammation and airway hyperresponsiveness and prevent lung function decline over time. So, considerations of future therapies should address not only the proportion of people with currently untreatable disease, but also those whose disease is eminently treatable but for whom daily preventer therapy is too big a challenge. Currently available therapies may fail due to the requirement for long term daily adherence, the lack of perceived benefit, the belief that reliever therapy is a reliable rescue, and the burden of cost. New treatments, targeted at refractory asthma, such as anti-TNF $\alpha$  agents have been disappointing, although monoclonal antibody treatments targeted at severe eosinophilic asthma such as mepolizumab (anti-IL-5) have shown reduced asthma exacerbations, but surprisingly also a dissociation between preventing attacks and modifying daily symptoms and lung function. Other monoclonal antibody treatments such as dupilumab, lebrikuzimab and reslizumab targeted at specific interleukins in the TH-2 pathways offer hope to a small proportion of patients but the cost of these treatments means they may only be affordable for a tiny minority initially. For the rest we must employ key management strategies that make current therapies more effective and safe. Refinement of current treatments such as once daily ICS-LABA, inhaled steroid sparing therapies, greater understanding of bronchial thermoplasty, and new oral medications also provide opportunities for better asthma outcomes.



## **Asthma in Asia Pacific: The Burden**

**Christopher Lai**

Hong Kong

The International Study of Asthma and Allergies in Childhood (ISAAC) studies have provided valuable data on asthma prevalence in schoolchildren worldwide, including those in AP. In the Phase 3 study, conducted in the early millennium, the regional prevalence of “wheeze in the past 12 months”, was 8.8%, ranging from 0.8% in Tibet (China) to almost 30% in Ho Chi Minh City (Vietnam). Comparison between Phase I (1994-95) and Phase 3 data showed the annual change in current wheeze was 0.07%, ranging from -0.55% in Hong Kong and Manila (Philippines) to 0.52% in Bandung, (Indonesia). While there is no good comparative data on asthma prevalence in adults in AP, various studies using different definitions of asthma revealed the rates ranged from 1.7% in Japan to 10.3% in India.

Although asthma mortality has shown a decreasing trend in many developed countries worldwide, the disease is still causing significant morbidity, with about 1 in 3 patients suffering from uncontrolled asthma in AP. This poor control is likely the consequence of under-usage of inhaled steroid, and over-estimation of control (both on the part of patients and physicians). Uncontrolled asthma, as defined by GINA, is associated with a higher risk of exacerbation that requires urgent health care utilization, including emergency room attendance, unscheduled medical visits and hospitalization. Tackling these issues related to poor asthma control, as well as identifying the risk factors associated with the development of asthma, may help reduce the burden of disease.

# DAY 4 SUNDAY, 16<sup>TH</sup> NOVEMBER

## **LABA, LAMA and Other Bronchodilators in Asthma Treatments**

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Bronchial asthma is characterized by chronic inflammation, responsive to corticosteroids, a powerful anti-inflammatory agent. Current guidelines (GOLD 2014) recommended inhaled corticosteroid combined with long acting beta 2 agents is preferred, when dose of inhaled corticosteroids alone failed to control asthma (Bateman 2008 NAEDP 2007). At the receptor level addition of LABA facilitate steroid effect. Fixed-dose combination of LABA/ICS had given patients more palpable effect of symptoms improvement, increasing adherence to ICS intake lowered frequency of dosing and maintain disease control. Thus, the addition of LABA to ICS had become a preference to patients, improving compliance and overall better disease control. Quick acting LABA had further enhanced their role in being able to act as a reliever (GOLD 2014). The safety of LABA in Asthma had been questioned (Martinez 2005). The Cochrane systematic review however had shown evidence that when LABA is added to ICS beneficial effects are seen in asthma control.

The use of long acting anti-muscarinic agents (LAMA) in asthma is currently limited. In a recent study (Kert Jens 2012) when LAMA was added to poorly controlled asthmatics already on ICS and LABA, there was less worsening of exacerbation that required systemic corticosteroids with sustained bronchodilation.