APSR 2015

Congress of the Asian Pacific
Society of Respirology

PROGRAMME BOOK

December 3 - 6, 2015

Kuala Lumpur Convention Centre
Kuala Lumpur, Malaysia

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AN INITIAL MAINTENANCE THERAPY FOR PATIENTS WITH COPD WHO ARE BREATHLESS ON EXERTION[1]

NEW

DON'T WAIT – RECOMMEND ANORO® ELLIPTA® INSTEAD OF TIOTROPIUM. GIVE PATIENTS THE IMMEDIATE RESULTS THEY NEED[2,3]

ANORO® ELLIPTA®
umeclidinium/vilanterol
breathe...

References: 1. Anoro® Ellipta® 62.5/25 Malaysia Prescribing Information PI01MAL based on EUSPC 27Mar15. **2.** Donohue JF et al. Efficacy and safety of once-daily umeclidinium/vilanterol 62.5/25 mcg in COPD. Respir Med 2013; 107:1538-1546. **3.** Data on file: Anoro® Ellipta® (Umeclidinium/Vilanterol) - A comparison of shuttle walking test endpoints in exercise studies in patients with COPD. RF/UCV/0111/15.

COPD – Chronic Obstructive Pulmonary Disease

ABBREVIATED PRESCRIBING INFORMATION FOR ANORO ELLIPTA. Product Name & Active Ingredient: ANORO Ellipta 62.5/25 micrograms inhalation powder, pre-dispensed. **Indications:** ANORO is indicated as a maintenance bronchodilator treatment to relieve symptoms in adult patients with chronic obstructive pulmonary disease (COPD). **Dosage and Administration:** Adults: The recommended dose is one inhalation of ANORO 62.5/25 micrograms once daily. ANORO should be administered once daily at the same time of the day each day to maintain bronchodilation. The maximum dose is one inhalation of ANORO 62.5/25 micrograms once daily. Special population: No dosage adjustment is required in elderly patients over 65 years, renal impairment and mild or moderate hepatic impairment. The use of ANORO has not been studied in patients with severe hepatic impairment and should be used with caution.There is no relevant use of ANORO in the paediatric population (under 18 years of age) in the indication for COPD. **Contraindications:** Hypersensitivity to the active substances or to any of the excipients like Lactose monohydrate and Magnesium stearate. **Warnings & Precautions:** Asthma: Umeclidinium/vilanterol should not be used in patients with asthma since it has not been studied in this patient population. Paradoxical bronchospasm: As with other inhalation therapies, administration of umeclidinium/vilanterol may produce paradoxical bronchospasm that may be life-threatening. Treatment with umeclidinium/vilanterol should be discontinued immediately if paradoxical bronchospasm occurs and alternative therapy instituted if necessary. Not for acute use: Umeclidinium/vilanterol is not indicated for the treatment of acute episodes of bronchospasm. Deterioration of disease: Increasing use of short-acting bronchodilators to relieve symptoms indicates deterioration of control. In the event of deterioration of COPD during treatment with umeclidinium/vilanterol, a re-evaluation of the patient and of the COPD treatment regimen should be undertaken. Cardiovascular effects: Cardiovascular effects, such as cardiac arrhythmias e.g. atrial fibrillation and tachycardia, may be seen after the administration of muscarinic receptor antagonists and sympathomimetics, including umeclidinium/vilanterol. Patients with clinically significant uncontrolled cardiovascular disease were excluded from clinical studies. Therefore, umeclidinium/vilanterol should be used with caution in patients with severe cardiovascular disease. Antimuscarinic activity: Consistent with its antimuscarinic activity, umeclidinium/vilanterol should be used with caution in patients with urinary retention or with narrow-angle glaucoma. Hypokalaemia: Beta2-adrenergic agonists may produce significant hypokalaemia in some patients, which has the potential to produce adverse cardiovascular effects. The decrease in serum potassium is usually transient, not requiring supplementation. No clinically relevant effects of hypokalaemia were observed in clinical studies with umeclidinium/vilanterol at the recommended therapeutic dose. Caution should be exercised when umeclidinium/vilanterol is used with other medicinal products that also have the potential to cause hypokalaemia. Hyperglycaemia: Beta2-adrenergic agonists may produce transient hyperglycemia in some patients. No clinically relevant effects on plasma glucose were observed in clinical studies with umeclidinium/vilanterol at the recommended therapeutic dose. Upon initiation of treatment with umeclidinium/vilanterol plasma glucose should be monitored more closely in diabetic patients. Coexisting conditions: Umeclidinium/vilanterol should be used with caution in patients with convulsive disorders or thyrotoxicosis, and in patients who are unusually responsive to beta2-adrenergic agonists. Excipients: This medicinal product contains lactose. Patients with rare hereditary problems of galactose intolerance, the Lapp lactase deficiency or glucose-galactose malabsorption should not take this medicinal product. Drug interaction: Caution must be taken when co-administring with beta-adrenergic blockers, metabolic and transporter based interactions drugs,other antimuscarinics/sympathomimetics, hypokalaemia, other medicinal products for COPD. Pregnancy and Lactation: Administration of drugs during pregnancy should only be considered if the expected benefit to the mother is greater than any possible risk to the foetus/neonate. **Adverse Events:** Frequencies are defined as: very common (≥ 1/10), common (≥1/100 to <1/10), uncommon (≥1/1000 to <1/100), rare (≥ 1/10,000 to <1/1000) and very rare (<1/10,000) including isolated reports.

System Organ Class	Adverse reactions	Frequency
Infections and infestations	Urinary tract infection	Common
	Sinusitis	Common
	Nasopharyngitis	Common (Most common-9%)
	Pharyngitis	Common
	Upper respiratory tract infection	Common
Nervous system disorders	Headache	Common
Cardiac disorders	Atrial fibrillation	Uncommon
	Supraventricular tachycardia	Uncommon
	Rhythm idioventricular	Uncommon
	Tachycardia	Uncommon
	Supraventricular extrasystoles	Uncommon
Respiratory, thoracic and mediastinal disorders	Cough	Common
	Oropharyngeal pain	Common
Gastrointestinal disorders	Constipation	Common
	Dry mouth	Common
Skin and subcutaneous tissue disorders	Rash	Uncommon

Please read the full prescribing information prior to administration, available from:
GlaxoSmithKline Pharmaceutical Sdn Bhd (3277-U) Level 6, Quill 9, 112 Jalan Semangat, 46300 Petaling Jaya, Selangor Darul Ehsan, Malaysia.
Abbreviated Prescribing Information Version 01 based on PI01MAL based on EUSPC 27Mar15
Date of revision: 9 October 2015

MY/UCV/0003/15 11/15

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For Medical/Healthcare Professionals Only

Theravance

Content

Welcome Message by Congress President

Dear friends and colleagues,

I would like to personally welcome each of you to the 20th Congress for the Asian Pacific Society of Respirology – '*Selamat Datang ke Kuala Lumpur'*.It is an exciting time for APSR and Malaysian Thoracic Society. Together, we continue to grow, whilst confronting a time of rapid change. We are meeting these changes during a time of larger nation-wide and global change. The challenge is for us to remain relevant, adaptable and responsive.

I would like to give you an idea of what you can expect and what we hope to achieve over the next few days. Our scientific programme in the form of symposia, plenary lectures, interactive sessions and hands–on workshops will cover scientific advances ranging from a molecular level, to clinical medicine and respiratory health in the population. It is our sincere hope that you will go away with the satisfaction of having gleaned new information, ideas and skills that can benefit you, whether in your research, health or clinical settings.

The world of respiratory health is an exciting area in which to work. Amongst other changes, we are transforming the way we operate to continuously improve our ability to deliver health care. In this congress, the APSR has committed itself to collaborate in the development of e-health platforms to better deliver and improve accessibility to healthcare for our patients. We are committed to bring inspired people together in meetings like this, to ensure our work always remains at the cutting edge.

Our industry partners and sponsors have continued to meet the challenges of our field and to excel despite the economy setback. We should all be very proud of where we are today and excited about where we are headed. Partners and sponsors from the industry, we thank you.

Terima kasih, to each of you, in attending our conference and bringing your expertise to our gathering. As experts in your field, you have the vision, knowledge, the wherewithal and the experience to help us pave our way into the future. You are truly our greatest asset today and tomorrow, and we could not accomplish what we do without your support. Throughout this conference, I ask you to stay engaged, keep proactive and help us shape the future of respiratory health.

My personal thanks to the APSR Central Committee and secretariat, under the stewardship of Professor Dr Michiaki Mishima and Professor Dr Kwun M Fong, for giving Malaysian Thoracic Society an opportunity to organise this congress. Finally, I would like to thank Professor Yong-Kek Pang, our Scientific Chairperson and the very dynamic local congress committee who have tirelessly contributed their invaluable time and effort in making this congress a success. Enjoy the congress and enjoy our country Malaysia!

Terima kasih!

Roslina A Manap

President, Malaysian Thoracic Society
Congress President, APSR 2015

About the Asian Pacific Society of Respirology

The Asian Pacific Society of Respirology 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.

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 Respirology, a journal of international repute

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Bronchoscopy and Interventional Technique	**Takashi Ishida**
Cell and Molecular Biology	**Yasuhiro Yamauchi**
COPD	**Diahn-Warng Perng**
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Clinical Respiratory Medicine	**Philip Eng**
Critical Care Medicine	**Yoshiki Ishii**
Environmental& Occupational Health and Epidemiology	**Soon-Hee Jung**
Interstitial Lung Disease	**Masahito Ebina**
Lung Cancer	**Kwun Fong**
Paediatric Lung Disease	**Albert Li**
Pulmonary Circulation	**Hiroshi Kimura**
Respiratory Infections	**Jun-Hee Woo**
Respirology Neurobiology and Sleep	**Kazuo Chin**
Respiratory Structure and Function	**Yasutaka Nakano**
Tuberculosis	**Charles Yu**

About the Malaysian Thoracic Society

The Malaysian Thoracic Society (MTS) was formed in late 1986 and the official inauguration was graced by His Royal Highness DYMM Seri Paduka Sultan Azlan Shah of Perak Darul Ridzuan, who was also the patron of the society at the first MTS scientific meeting in June 1987.

Objective

- To advance the knowledge and practice of thoracic (or respiratory) medicine
- To promote research in the field of respiratory medicine
- To organise regular scientific meetings
- To facilitate collaboration work between qualified individuals or societies in respiratory medicine
- To publish books, magazine, periodicals, leaflets or other literary or scientific works that the society may think desirable for the promotion of its objectives, subject to the approval of the relevant authorities

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AWARDS

APSR Medal

APSR Research Awards

Michiyoshi Harasawa Research Award

Ann J. Woolcock Research Award

Fukuchi Award

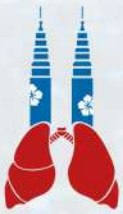
for the Best Paper in Respirology

Young Investigator Awards

From APSR, ATS, ERS and TSANZ

APSR Travel Awards

APSR-IASLC Book Prizes



APSR 2015

Congress of the Asian Pacific Society of Respirology

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.

Takashi Horie

President
Ohta General Hospital Foundation
Japan

In 1986, Dr Takashi Horie made great efforts with our founding members to formulate the basic international respiratory society representing the region of Asian Pacific as the Chairman of the International Relations Committee of the JRS.He had endeavored to find funds for the APSR as the Treasurer of the Society up to 2006 to become competitive level to other international respiratory societies.

Furthermore, at his period of the Chairman, Board of Directors of the JRS, he brought a great bulk of fund to the APSR after positioning the JRS as APSR en bloc member. This en bloc membership was first to be adopted and unprecedented in other regional societies.

Academic achievements are not cardinal qualifying conditions for the medal, but still worthwhile to acknowledge that Dr. Horie mentored four professors in respirology during his chairmanship of the respiratory department of Nippon University in Tokyo.

Due to his great contribution particularly at the inauguration period and through international promotion of the status of the APSR, he is worthy of the 2015 APSR Medal.

Awards

Michiyoshi Harasawa Research Award

The Michiyoshi Harasawa Research Award is given in the memory of the late Dr. Harasawa, who died September 2001. The award is given to the 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 organisation of the Asian Pacific Society of Respirology (APSR) in 1986 that became a highly structured and leading society of pulmonologists in the Asia Pacific.

Hiroshi Kimura

Chairman, Department of Internal Medicine,
Professor of Medicine, Department of Respiratory Medicine
Nara Medical University School of Medicine
Nara, Japan

Education:

1972-1978, Kanazawa University School of Medicine (M.D.) 1985, Chiba University School of Medicine (Ph.D.)

Professional Areas:

Respiratory Medicine / Respiratory Physiology / Pulmonary Circulation / Chronic Obstructive Pulmonary Disease / Lung Cancer / Sleep Medicine / Control of Breathing / Inflammatory Cytokines / Exercise Physiology / High Altitude Medicine

Professional Societies:

- President, The Japan Society for Respiratory Care and Rehabilitation Medicine (October 2013-October 2014)
- President, The Japanese Respiratory Society (April 2014-April 2015)
- The representative senior directors, The Japanese Respiratory Society (2014-Present)
- The board of directors,The Japanese Respiratory Society
- The board of directors, The Japan Society for Respiratory Care and Rehabilitation Medicine
- The board of directors, Japanese Society of Clinical Physiology

Awards

Ann Jane Woolcock Research Award

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

Ian Yang

Thoracic Physician and Director of Thoracic Medicine
The Prince Charles Hospital
Brisbane, Australia

Prof. Yang has an exemplary clinical, research and educational record in respiratory medicine and has made significant contributions to advancing respiratory medicine in our region and globally.

He is a senior Thoracic Physician at The Prince Charles Hospital and is leading the Department as Program Medical Director. His clinical expertise in clinical and translational research is in the field of chronic lung disease, with special interest in gene-environmental interaction in chronic obstructive pulmonary disease (COPD), asthma and lung cancer.

In terms of COPD, the highlights of his research output include original work, the first to demonstrate the importance of genetic polymorphism in influencing COPD exacerbations. Futhermore, the original work of his QU PhD student (Dr Santuyagu Savarimuthu Francis), focused on the pathogenesis of COPD progression – this team's papers in [] have demonstrated novel patterns of dysregulation in gene expression, epigenetics and the immune system occurring in COPD, providing potentially useful disease biomarkers for the future.

Awards

Fukuchi Award for the Best Original Paper in Respirology

Respirology and Wiley publishing are delighted to present the 2015 Fukuchi award for the best original paper published in Respirology, the official journal of APSR in 2014.

This prestigious award has been named after Professor Fukuchi who was the president of the APSR between 2004 – 06 and who was also the editor of Respirology between 1996 – 99. The decision on the best paper published in Respirology was made by world renouned international researchers.

Guo Chen

Resident Doctor
Guangdong General Hospital
Guangzhou, China

Tao Yang

National Center for Cardiovascular Diseases,
Fuwai Hospital,
Chinese Academy of Medical Sciences & Peking Union Medical College,
Beijing, China.

Presentation: Elevated plasma YKL-40 as a prognostic indicator in patients with idiopathic pulmonary arterial hypertension

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.

ATS Young Investigator Awardees

Laura E. Crotty Alexander

Assistant Professor of Medicine
University of California at San Diego

Presentation: E-cigarette and Conventional Cigarette Pro-Virulent Effects on Airway Colonizer and PathogenStaphylococcus aureus

Isaac Almendros

University of Barcelona, Barcelona, Spain

Presentation: Cancer and OSA: Macrophages as Protagonists or Bystanders?

Awards

Young Investigator Awardees

Separately, presenting authors under the age of 35 who are also the member of European Respiratory Society (ERS) are additionally considered for APSR-ERS Travel Award.

ERS Young Investigator Awardees

Caroline Bernadette Olanka King Kay

University of Santo Tomas Hospital
Philippines

Presentation: Performance of TB Diagnostic Committee (TBDC) in Certifying Disease Activity Among Smear Negative Retreatment Cases in District IV of Manila, Philippines from January 2013 to June 2014

Fatim Tahirah Mirza Mohd Tahir Beg

Curtin University
Australia

Presentation: Regression equations to estimate the two-minute walk distance (2MWD) in Malaysian adults aged 40 to 75 years

TSANZ Young Investigator Awardees

Rebecca L Harper

Royal Adelaide Hospital, Adelaide, South Australia

Presentation: MPR2 Upregulation via *in situ* Gene Delivery or via Engineered Endothelial Progenitor Cells Alleviates Pulmonary Arterial Hypertension in a Rat Model

Awards

APSR Travel Awards

Outstanding works by abstract presenters will be further considered for a limited number of Travel Awards. The highly-coveted award will be offered based on the scientific merit of the submitted abstract, and is aimed at partially supporting investigators from developing regions to attend the congress.

Abdulaziz Aljohani (Canada)
Akihisa Mitani (United Kingdom)
Andre Awaloei (Philippines)
Avril Soh (Singapore)
Bingdi Yan (China)
Gooh Yeon Hong (South Korea)
Ho Jung Jeong (South Korea)
Ji Young Park (South Korea)
Jing Liu (China)
Jiunn-Liang Tan (Malaysia)
Ken-ichiro Tanaka (Japan)
Lily Diana Zainudin (Malaysia)
Motoyasu Kato (Japan)
Nurhuda Ismail (Malaysia)
Rie Kinoshita (Japan)
Saleela Ruwanpura (Australia)
Sukhwinder S. Sohal (Australia)
Takashi Niwa (Japan)
Tomohiko Mio (Japan)
Toshiya Inui (Japan)
Wenhua Jian (China)
Yasuhiro Kawai (Japan)
Yoshinori Fuseya (Japan)
You Zhou (China)
Yuji Matsumoto (Japan)

APSR-IASLC Book Prizes

Cheng Lee Daniel Pang (Malaysia)
Hye Seon Kang (South Korea)
Jiaoli Wang (China)
Jiunn-Liang Tan (Malaysia)
Jun-Pyo Myong (South Korea)
Midori Ura (Japan)
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APSR 2015

Congress of the Asian Pacific Society of Respirology

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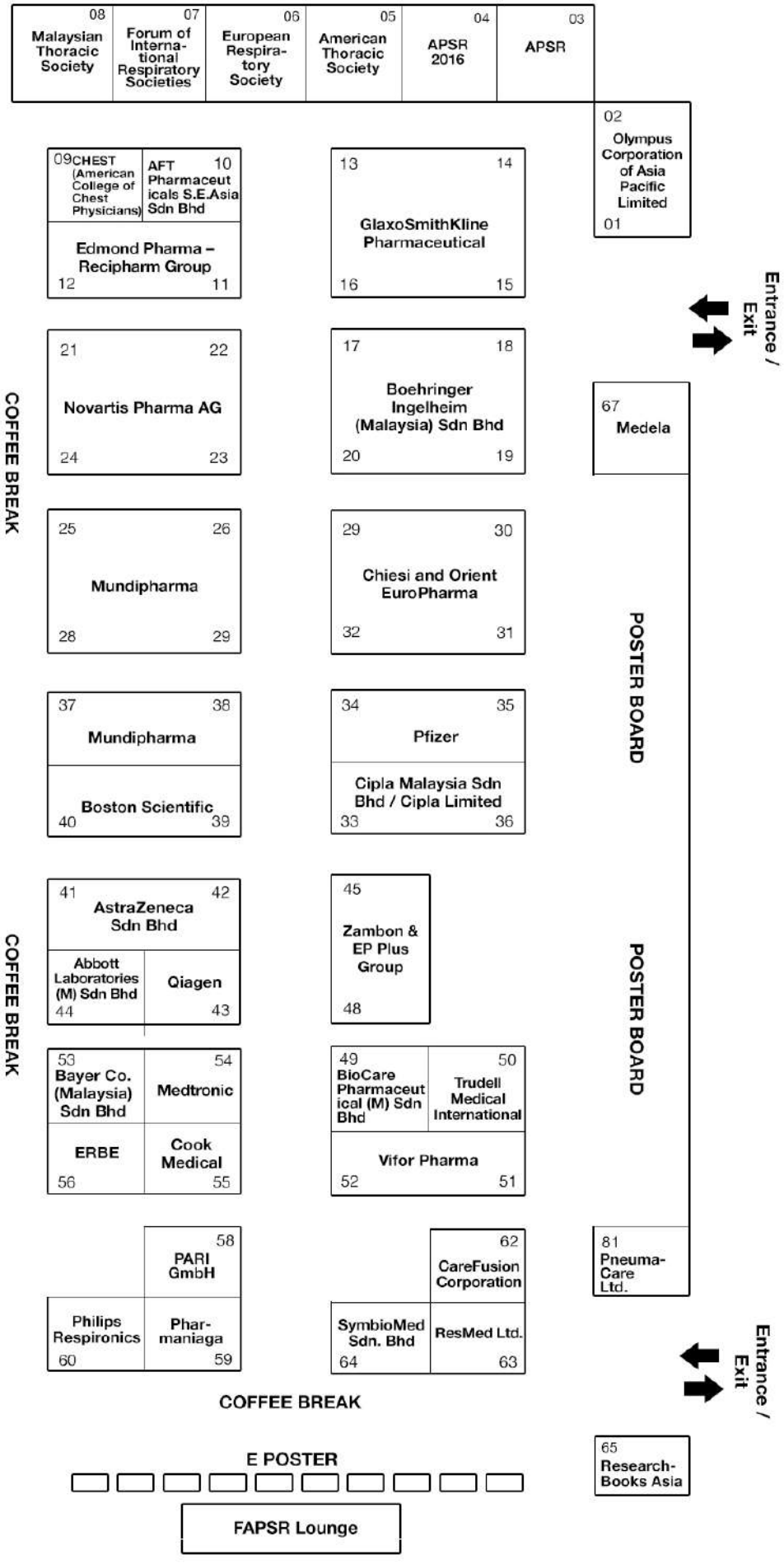
Exhibitors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SPR-23SEPT15-002 (01/10/2015)

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The First Once-Daily Dual Bronchodilator[1]

THE FIRST ONCE-DAILY DUAL BRONCHODILATOR[1]
ULTIBRO™ BREEZHALER®
START A NEW CHAPTER IN COPD[2-4]

ULTIBRO™ BREEZHALER® is indicated as a once-daily maintenance bronchodilator treatment to relieve symptoms in adult patients with chronic obstructive pulmonary disease (COPD).

References: 1. PI Rd 14 Mar 2014; Appr 27 Feb 2015 2. Vogelmeier CF, Bateman ED, Pallante J, et al. Efficacy and safety of once-daily QVA149 compared with twice-daily salmeterol-fluticasone in patients with chronic obstructive pulmonary disease (ILLUMINATE): a randomised, double-blind, parallel group study. *Lancet Respir Med*. 2013;1:51-60. 3. Bateman ED, Ferguson GT, Barnes N, et al. Dual bronchodilation with QVA149 versus single bronchodilator therapy: the SHINE study. *Eur Respir J*. Published online May 2013 as doi: 10.1183/09031936.00200212. 4. Wedzicha JA, Decramer M, Ficker JH, et al. Analysis of chronic obstructive pulmonary disease exacerbations with the dual bronchodilator QVA149 compared with glycopyrronium and tiotropium (SPARK): a randomised, double-blind, parallel-group study. *Lancet Respir Med*. 2013;1:199-209.

NOVARTIS
PHARMACEUTICALS

Novartis Corporation (Malaysia) Sdn Bhd (10920H)
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Seksyen 13, 46200 Petaling Jaya, Selangor, Malaysia.
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ONCE DAILY
ultibro®
breezhaler®
indacaterol maleate/glycopyrronium bromide
inhalation powder

For Healthcare Professionals only

Ultibro/Adv01/MY1510993713

Perfect harmony in asthma control

flutiform®

CONSISTENT DELIVERY

***flutiform*® HDi** produces consistent high fine particle fraction of approximately 40% at different inhalation flow rates[1]

EFFICACIOUS

***flutiform*®** provides greater reduction of exacerbation risk than fluticasone alone[2]

FAST

***flutiform*®** offers rapid and sustained control of asthma symptoms[3]

***flutiform*®** (fluticasone propionate and formoterol fumarate) pressurised inhalation suspension
PRESENTATION: Pressurised inhalation suspension, in a pressurised metered dose inhaler (pMDI), containing fluticasone propionate and formoterol fumarate dihydrate at strengths of 50 mcg/5 mcg, 125 mcg/5 mcg or 250 mcg/10 mcg per actuation. One inhaler contains 120 actuations. **INDICATIONS:** This fixed-dose combination of fluticasone propionate and formoterol fumarate (flutiform® inhaler) is indicated in the regular treatment of asthma where the use of a combination product (inhaled corticosteroid and long-acting β2-agonist) is appropriate: For patients not adequately controlled with inhaled corticosteroids and 'as required' inhaled short-acting β2-agonist , or for patients already adequately controlled on both an inhaled corticosteroid and a long-acting β2-agonist. flutiform® 50 mcg/5 mcg and 125 mcg/5 mcg per actuation are indicated for use in adults and adolescents 12 years and above. flutiform® 250 mcg/ 10 mcg per actuation is indicated in adults only. **DOSAGE AND ADMINISTRATION:** For inhalation use. The patient should be shown how to use the inhaler correctly by a healthcare professional. Patients should be given the strength of flutiform® containing the appropriate fluticasone propionate dose for their disease severity (note that flutiform® 50 mcg/5 mcg per actuation is not appropriate in patients with severe asthma). The appropriate strength should be taken as two inhalations, twice-daily (normally in the morning and evening) and used every day, even when asymptomatic. Prescribers should be aware that in asthmatics, fluticasone propionate is as effective as some other inhaled steroids when administered at approximately half the total daily microgram dose. Total daily dose can be increased if asthma remains poorly controlled by administering a higher strength inhaler. Appropriate doses of the β2-agonist and inhaled corticosteroid (ICS) in separate inhalers, or the ICS alone, should be prescribed if a patient requires doses outside the recommended dose regimens. Patients should be assessed regularly and once asthma is controlled, treatment should be reviewed and stepped down to the lowest effective dose, or an ICS alone. It is extremely important to regularly review patients as their treatment is stepped down. ICSs alone are first line treatment for most patients. flutiform® is not intended for initial treatment of mild asthma. For patients with severe asthma the ICS therapy should be established before prescribing a fixed-dose combination product. Patients on flutiform® must not use an additional LABA. An inhaled SABA should be taken for immediate relief of asthma symptoms arising between doses. The AeroChamber Plus® spacer device is recommended in patients who find it difficult to use inhalers; re-titration should always follow the introduction of a spacer device. Patients should be advised to contact their prescriber when the flutiform® dose indicator is getting near zero. **CONTRAINDICATIONS:** Hypersensitivity to any of the active substances or excipients. **PRECAUTIONS AND WARNINGS:** flutiform® should not be used for the first treatment of asthma, to treat acute asthma symptoms or for prophylaxis of exercise-induced asthma. It should not be initiated during an exacerbation, during significantly worsening or acutely deteriorating asthma, and should not be stopped abruptly. Patients should use their flutiform® maintenance treatment as prescribed, even when asymptomatic. If a patient experiences serious asthma-related adverse events or exacerbations, they should continue treatment but also seek medical advice. Patients should be reviewed as soon as possible if there is any indication of deteriorating asthma control. In the case of sudden and progressive deterioration, which is potentially life-threatening, an urgent medical assessment should be carried out. Use with caution in patients with: pulmonary tuberculosis; quiescent tuberculosis; fungal, viral or other infections of the airway; thyrotoxicosis; pheochromocytoma; diabetes mellitus (consider additional blood sugar controls); uncorrected hypokalaemia; predisposition to low levels of serum potassium; impaired adrenal function (monitor HPA axis function regularly); hypertrophic obstructive cardiomyopathy; idiopathic subvalvular aortic stenosis; severe hypertension; aneurysm or other severe cardiovascular disorders. There is risk of potentially serious hypokalaemia with high doses of β2-agonists or concomitant treatment with β2-agonists and drugs that can induce or potentiate a hypokalaemic effect. Particular caution is recommended in unstable or acute severe asthma and other conditions when the likelihood for hypokalaemia adverse effects is increased. Monitoring of serum potassium levels is recommended during these circumstances. Formoterol may induce prolongation of the QTc interval. Caution must be observed when treating patients with existing prolongation of QTc interval. flutiform® should be discontinued immediately if there is evidence of paradoxical bronchospasm. Systemic effects with an ICS may occur, particularly at high doses for prolonged periods or when combined with potent CYP3A4 inhibitors, but are less likely than with oral corticosteroids. Use of a spacer device may also cause an increased systemic exposure. Increased exposure can be expected in patients with severe hepatic impairment. Prolonged treatment with high doses of corticosteroids may result in adrenal suppression and acute adrenal crisis, particularly in adolescents and children or potentially as a result of trauma, surgery, infection or rapid dose reduction. Patients should be advised that flutiform® contains a small amount of ethanol; however this negligible amount does not pose a risk to patients. flutiform® is not recommended in children under 12 years of age. **INTERACTIONS:** Caution is advised in long-term co-administration with strong CYP3A4 inhibitors (e.g. ritonavir, atazanavir, clarithromycin, indinavir, itraconazo le, nelfinavir, saquinavir, ketoconazole and telithromycin); co-administration should be avoided if possible. Caution is advised with use of non-potassium sparing diuretics (e.g. loop or thiazide), xanthine derivatives, glucocorticosteroids, L-Dopa, L-thyroxine, oxytocin, alcohol or other adrenergic drugs. There is an increased risk of arrhythmias in patients receiving concomitant anaesthesia with halogena ted hydrocarbons. Hypokalaemia may increase the risk of arrhythmias in patients being treated with digitalis glycosides. Concomitant use of β-adrenergic drugs can have a potentially additive effect. Extreme caution should be taken when using formoterol fumarate with drugs known to prolong the QTc interval, such as tricyclic antidepressants or MAOIs (and for two weeks following their discontinuation), as well as antipsychotics (including phenothiazines), quinidine, disopyramide, procainamide and antihistamines. Concomitant use of an MAOI or a similar agent, such as furazolidone or procarbazine, may precipitate hypertensive reactions. β-blockers and formoterol fumarate may inhibit the effect of each other. β-blockers may produce severe bronchospasm in asthma patients, and they should not normally be treated with β-blockers including those that are used as eye drops to treat glaucoma. Under certain circumstances, e.g. as prophylaxis after myocardial infarction, cardioselective β-blockers could be considered with caution. **PREGNANCY AND LACTATION:** flutiform® is not recommended during pregnancy. It should only be considered if benefits to the mother outweigh risks to the foetus. It is not known whether fluticasone propionate or formoterol are excreted in breast milk; a risk to the breast feeding infant cannot be excluded. A decision should be made on whether to discontinue breastfeeding or discontinue/abstain from flutiform®. **SIDE-EFFECTS:** Potentially serious side-effects: hyperglycaemia; depression; aggression; behavioural changes (predominantly in children); paradoxical bronchospasm; agitation; vertigo; palpitations; ventricular extrasystoles; angina pectoris; tachycardia; hypertension; dyspnoea; peripheral oedema; Cushings Syndrome; adrenal suppression; growth retardation; cataract and glaucoma; hypersensitivity reactions and QTc interval prolongation. For detailed information, please refer to the full prescribing information.

REFERENCES: 1. Johal B, Howald M *et al.* Fine particle profile of fluticasone propionate/formoterol fumarate versus other combination products: the DIFFUSE study. Comb Prod Ther 2013;(3):39–51. **2.** Poster presented at: Annual Congress of the European Respiratory Society (ERS); 2013; Barcelona. **3.** Aalbers R *et al.* Onset of bronchodilation with fluticasone/formoterol combination versus fluticasone/salmeterol in an open-label, randomized study. Adv Ther 2012; 29 (11): 958-969.

For Healthcare Professional Use Only.

MY-FLU-0479-V1-1115

Mundipharma Pharmaceuticals Sdn. Bhd. (Reg. No. 665228K)
A-5-01 Level 5, Block A, PJ8, No. 23 Jalan Barat, Seksyen 8,
46050 Petaling Jaya, Selangor Darul Ehsan, Malaysia.
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flutiform®
fluticasone propionate/formoterol

Help prevent pneumococcal disease with the proven protection of Prevenar 13*

Prevenar 13 is now proven to help prevent vaccine-type pneumococcal pneumonia in adults ≥ 65 years of age: Results from the Community-Acquired Pneumonia Immunization Trial in Adults (CAPiTA)-one of the largest vaccine-efficacy trials ever conducted[1]

Prevenar 13 is indicated for active immunization for the prevention of pneumococcal disease caused by Streptococcus pneumoniae serotypes 1, 3, 4, 5, 6A, 6B, 7F, 9V, 14, 18C, 19A, 19F and 23F in adults aged 18 years and older.[2]

- Hypersensitivity (eg, anaphylaxis) to any component of Prevenar 13 or any diphtheria toxoid–containing vaccine is a contraindication to the use of Prevenar 13[2]
- Prevenar 13 does not provide 100% protection against vaccine serotypes nor protect against nonvaccine serotypes[2]

REFERENCES
1. Bonten MJM, Huijts SM, Bolkenbaas M, et al. Polysaccharide conjugate vaccine against pneumococcal pneumonia in adults. N Engl J Med. 2015;372(12):1114-1125. **2**. Prevenar 13 Malaysia PI, PREVENAR 13 - 0315.

ABRIDGED PRESCIBING INFORMATION

NAME OF THE MEDICINAL PRODUCT: Prevenar 13 suspension for injection. The vaccine is a homogeneous white suspension. Pneumococcal polysaccharide conjugate vaccine (adsorbed), 13-valent Conjugated to CRM197 carrier protein and adsorbed on aluminium phosphate (0.125 mg aluminium). **THERAPEUTIC INDICATIONS:** Active immunization for the prevention of pneumococcal disease caused by *Streptococcus pneumonia* serotypes 1, 3, 4, 5, 6A, 6B, 7F, 9V, 14, 18C, 19A, 19F and 23F (including invasive disease,pneumonia and acute otitis media) in infants, children and adolescents from 2 months to 17 years of age. Active immunisation for the prevention of pneumococcal disease caused by *Streptococcus pneumoniae* serotypes 1, 3, 4, 5, 6A, 6B, 7F, 9V, 14, 18C, 19A, 19F and 23F in adults aged 18 years and older. **METHOD OF ADMINISTRATION:** The vaccine should be given by intramuscular injection. The preferred sites are the anterolateral aspect of the thigh (vastus lateralis muscle) in infants or the deltoid muscle of the upper arm in children, adolescents and adults. **POSOLOGY:** Infants aged 2- 6 months: The recommended immunisation series consists of four doses, each of 0.5 ml. The primary infant series consists of three doses, with the first dose usually given at 2 months of age and with an interval of at least 1 month between doses. The first dose may be given as early as six weeks of age. The fourth (booster) dose is recommended between 12-15 months of age. Alternatively, when Prevenar 13 is given as part of a routine infant immunization programme, a series consisting of three doses, each of 0.5 ml, may be considered. The first dose may be given from the age of 2 months, with a second dose 2 months later. The third (booster) dose is recommended between 11-15 months of age. Unvaccinated infants and children ≥7 months of age: Infants aged 7-11 months: Two doses, each of 0.5 ml, with an interval of at least 1 month between doses. A third dose is recommended in the second year of life. Children aged 12-23 months. Two doses, each of 0.5 ml, with an interval of at least 2 months between doses. Children and adolescents aged 2 years to 17 years One single dose of 0.5 ml. Prevenar 13 vaccine schedule for infants and children previously vaccinated with Prevenar (*Streptococcus pneumoniae* serotypes 4, 6B, 9V, 14, 18C, 19F, and 23F). Prevenar 13 contains the same 7 serotypes contained in Prevenar, using the same carrier protein CRM197. In clinical trials, immunogenicity and safety profiles are comparable between the two vaccines. Infants and children Children who have begun immunisation with Prevenar may complete immunisation by switching to Prevenar 13 at any point in the schedule. Young children and adolescents (1-17 years) who are completely immunised with Prevenar should receive one dose of Prevenar 13 to elicit immune responses to the 6 additional serotypes. Adults aged 18 years and older: Prevenar 13 is to be administered as a single dose to adults 18 years and older including those previously vaccinated with a pneumococcal polysaccharide vaccine. The need for re-vaccination with a subsequent dose of Prevenar 13 has not been established. **CONTRAINDICATIONS:** Hypersensitivity to the active substances, to any of the excipients or to diphtheria toxoid. As with other vaccines, the administration of Prevenar 13 should be postponed in subjects suffering from acute, severe febrile illness. However, the presence of a minor infection, such as a cold, should not result in the deferral of vaccination. **SPECIAL WARNINGS AND PRECAUTIONS FOR USE:** Do not administer Prevenar 13 intravascularly. As with all injectable vaccines, appropriate medical treatment and supervision should always be readily available in case of a rare anaphylactic event following the administration of the vaccine. This vaccine should not be given to individuals with thrombocytopenia or any coagulation disorder that would contraindicate intramuscular injection, unless the potential benefit clearly outweighs the risk of administration. Prevenar 13 will only protect against *Streptococcus pneumoniae* serotypes included in the vaccine, and will not protect against other microorganisms that cause invasive disease, pneumonia, or otitis media. As with any vaccine, Prevenar 13 may not protect all individuals receiving the vaccine from pneumococcal disease. Individuals with impaired immune responsiveness, whether due to the use of immuno-suppressive therapy, a genetic defect, human immunodeficiency virus (HIV) infection, or other causes, may have reduced antibody response to active immunization. Safety and immunogenicity data for Prevenar 13 are not available for individuals in specific immunocompromised groups (e.g., malignancy, nephrotic syndrome) and vaccination should be considered on an individual basis. **PREGNANCY AND LACTATION:** There are no data from the use of pneumococcal 13-valent conjugate in pregnant women. Animal studies do not indicate direct or indirect harmful effects with respect to reproductive toxicity. **UNDESIRABLE EFFECTS:** Infants and children aged 6 weeks to 5 years: The most commonly reported adverse reactions were vaccination-site reactions, fever, irritability, decreased appetite, and increased and/or decreased sleep. An increase in vaccination site reactions was reported in children older than 12 months compared to rates observed in infants during the primary series with Prevenar 13. Children and adolescents aged 6-17: The most commonly reported adverse events were decreased appetite and irritability; any vaccination-site erythema; induration/swelling or pain/tenderness; somnolence; poor quality sleep; vaccination-site tenderness (including impaired movement). Adults aged 18 years and older: Decreased appetite, Headaches, Diarrhea, vomiting, Rash, arthralgia, myalgia, Chills, fatigue, vaccination-site erythema, vaccination-site induration/swelling, vaccination-site pain/tenderness, limitation of arm movement, fever. **SPECIAL PRECAUTIONS FOR STORAGE:** Store in a refrigerator (2°C–8°C). Do not freeze.

Product Indications and prescribing information may di ffer in each country. Please use products in accordance with the approved indication(s) and prescribing information in your country

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Prevenar 13*
Pneumococcal polysaccharide conjugate vaccine (13-valent, adsorbed)
*Trademark

WMAPRV0815082-12Oct2015

Life gets better
when the right things come together

Symbicort 160/4.5 mcg can be prescribed for patients 12 years and above.
A lower dose of Symbicort should be prescribed in patients 6-11 years old

Symbicort is indicated for treatment of Asthma & COPD with

- Fast onset-of-action[1]
- Unique SMART indication and flexible SMART dosing for Asthma[2,6]
- Significant and sustained improvement in lung function and exacerbation reduction[2-5]

FOR **ASTHMA**

160/4.5 mcg

1-2 inhalations BID + prn

Dosage for adults only.

*SMART therapy is indicated for Adults 18 years and older
**Not more than 6 inhalations should be taken on a single occasion and 12 inhalations daily for a limited period of time

FOR **COPD**

320/9 mcg

1 inhalation BID

References:
1. Balanag et al., Pulm Pharm Ther 2006 ; 19:139-147. 2. O'Byrne et al. Am J Respi Crit Care Med 2005; 171:129-136. 3. Vogelmeier et al. Eur Respir J, 2005; 26:819-828. 4. Szafranski et al. Eur Respir J, 2003; 21:74-81. 5. Calverley et al. Eur Respir J, 2003; 22:912-919. 6. Symbicort® Prescribing Information.

SYMBICORT Turbuhaler Abbreviated Prescribing Information:

Indications: Asthma- Symbicort is indicated in the regular treatment of asthma where use of a combination (inhaled corticosteroid and long-acting beta2-agonist) is appropriate:- patients not adequately controlled with inhaled corticosteroids and "as needed" inhaled short-acting beta2-agonists or patients already adequately controlled on both inhaled corticosteroids and long-acting beta2-agonists. **COPD**- Symptomatic treatment of patients with severe COPD (FEV1 <50% predicted normal) and a history of repeated exacerbations, who have significant symptoms despite regular therapy with long-acting bronchodilators. **SYMBICORT Turbuhaler 320/9 mcg/inhalation Recommended doses: Asthma - Symbicort maintenance therapy. Adults (18 years and older):** 1-2 inhalations BD. **Adolescents (12-17 years):** 1 inhalation twice daily. **COPD - Adults (18 years and older):** 1 inhalation BD. Symbicort 320/9 micrograms/inhalation is not recommended for children under 12 years of age. **SYMBICORT Turbuhaler 160/4.5 mcg/inhalation Recommended doses: Asthma- (A) Symbicort maintenance therapy. Adults (18 years and older):** 1-2 inhalations BD. **Adolescents (12-17 years):** 1-2 inhalations BD. **(B) Symbicort maintenance and reliever therapy (SMART). Adults (18 years and older):** 2 inhalations per day, given either as 1 or 2 inhalations in either the morning or evening. Patients should take 1 additional inhalation as needed in response to symptoms. If symptoms persist after a few minutes, an additional inhalation should be taken. Not more than 6 inhalations should be taken on any single occasion. **COPD - Adults (18 years and older):** 2 inhalations BD. **SYMBICORT Turbuhaler 80/4.5 mcg/inhalation Recommended doses: Asthma - Symbicort maintenance therapy.** Children (6 years and older): 2 inhalations BD. **Contraindications:** Hypersensitivity (allergy) to budesonide, formoterol or inhaled lactose. **Special precautions:** Dose should be tapered when the treatment is discontinued and should not be stopped abruptly. Sudden and progressive deterioration in control of asthma or COPD is potentially life threatening and the patient should undergo urgent medical assessment. Concomitant treatment with itraconazole, ritonavir or other potent CYP3A4 inhibitors should be avoided. Symbicort should be administered with caution in patients with thyrotoxicosis, phaeochromocytoma, diabetes mellitus, untreated hypokalaemia, hypertrophic obstructive cardiomyopathy, idiopathic subvalvular aortic stenosis, severe hypertension, aneurysm or other severe cardiovascular disorders, such as ischaemic heart disease, tachyarrhythmias or severe heart failure. Formoterol itself may induce prolongation of the QTc-interval. Potentially serious hypokalaemia may result from high doses of beta2-agonists. Possible systemic effects particularly at high doses prescribed for long periods eg. adrenal suppression, growth retardation in children and adolescents, decrease in bone mineral density, cataract and glaucoma. **Undesirable Effects:** Common- palpitations, candida infections in the oropharynx, headache, tremor, mild irritation in the throat, hoarseness, coughing. **Pregnancy and lactation:** No clinical data on exposed pregnancies are available. Data on approximately 2000 exposed pregnancies indicate no increased teratogenic risk associated with the use of inhaled budesonide. Budesonide is excreted in breast milk. However, at therapeutic doses no effects on the suckling child are anticipated. It is not known whether formoterol passes into human breast milk.

Further information available on request
Please consult local full prescribing information before prescribing
For Healthcare Professionals Only

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47810 Petaling Jaya, Malaysia
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AstraZeneca

Symbicort®
budesonide/formoterol

MY_Symbicort_187,906.022_30/09/2014

NEW 5 YEAR DATA

Established long-term effectiveness and safety

Newly published data confirm Bronchial Thermoplasty (BT), delivered by the Alair™ System, as a safe and minimally invasive procedure that provides a long-term reduction in asthma exacerbations for patients with severe asthma.

Fewer respiratory-related emergency room visits

- **84% reduction in emergency room visits** for respiratory symptoms at 1 year compared with sham-controlled patients, with reduction maintained out to 5 years[1,2]

Fewer exacerbations, with effectiveness maintained out to 5 years

- **32% decrease in severe asthma exacerbations** (requiring systemic corticosteroids) at 1 year compared with sham-controlled patients, with reduction maintained out to 5 years[1,2]
  - —The decrease in severe exacerbations over 5 years included a substantial reduction in the use of systemic corticosteroids associated with those exacerbations[2]
- No increase in hospitalizations, asthma symptoms, or respiratory adverse events over 5-year period[2]

View the 5-year clinical trial results at BT5years.com

Brief Statement of Relevant Indications for Use, Contraindications, Warnings, and Adverse Events: The Alair Bronchial Thermoplasty System is indicated for the treatment of severe persistent asthma in patients 18 years and older whose asthma is not well controlled with inhaled corticosteroids and long-acting beta-agonists. The Alair System is not for use in patients with an active implantable electronic device or known sensitivity to medications used in bronchoscopy. Previously treated airways of the lung should not be retreated with the Alair System. Patients should be stable and suitable to undergo bronchoscopy. The most common adverse event of BT is an expected transient increase in the frequency and worsening of respiratory-related symptoms. Rx only.

CAUTION: Law restricts this device to sale by or on the order of a physician. Indications, contraindications, precautions, and warnings can be found with product labeling.

References: 1. Castro M, et al, for the AIR2 Trial Study Group. *Am J Respir Crit Care Med.* 2010;181:116-124. **2.** Wechsler M, et al; for the AIR2 Trial Study Group [published ahead of print September, 2013]. *J Allergy Clin Immunol.* doi:10.1016/j.jaci.2013.08.009.

NOW AVAILABLE from Boston Scientific
for the treatment of severe asthma in adults

Bronchial Thermoplasty™

Cipla

Access to powerful affordable asthma treatment is that simple

Rx

Seroflo Inhaler

Salmeterol 25 mcg +
Fluticasone propionate 50/125/250 mcg / dose

APSR 2015
Congress of Asian Pacific Society of Respirology

Edmond Pharma Silver Sponsor

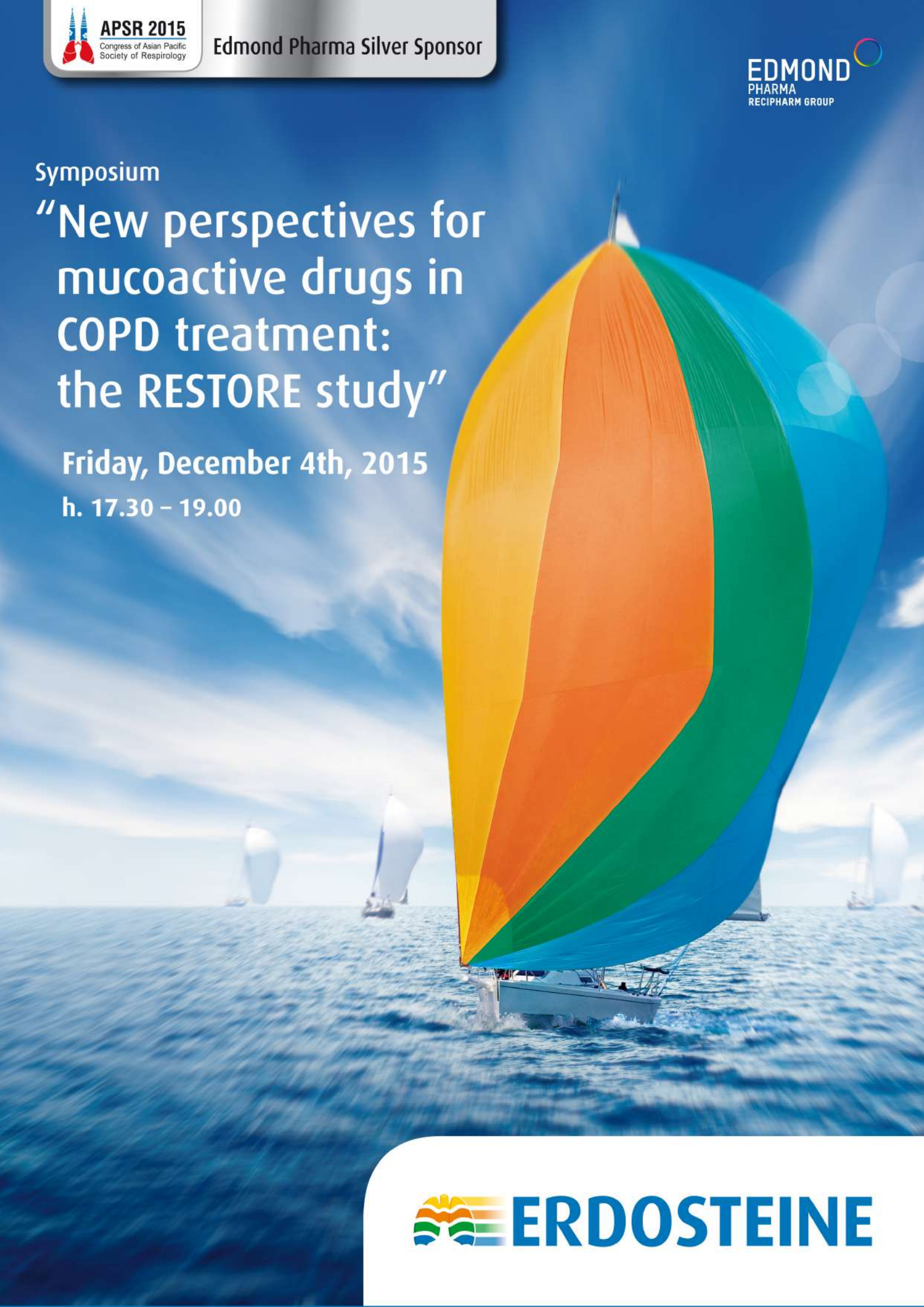
EDMOND PHARMA
RECIPHARM GROUP

Symposium

"New perspectives for mucoactive drugs in COPD treatment: the RESTORE study"

Friday, December 4th, 2015

h. 17.30 – 19.00



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MORE TIME FOR WHAT MATTERS

SUPERDIMENSION™ NAVIGATION SYSTEM WITH LUNGGPS™ TECHNOLOGY

The superDimension™ navigation system with LungGPS™ technology from Medtronic, offers you minimally invasive access to the entire lung to aid in the diagnosis and management of lung disease. With low complication rates[1] and high diagnostic yield[2], your patients can get back to what they enjoy doing most.

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1. Eberhardt R, et al. Electromagnetic Navigation Diagnostic Bronchoscopy in Peripheral Lung Lesions. CHEST. 2007;131:1800-1805.
2. Lee FL, et al. The emerging technique of electromagnetic navigation lung lesions: Promising results in 50 lesions. Cancer Cytopathology. 19 Dec 2013 (online ahead of print). DOI: 10.1002/cncy.21510

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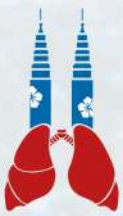
Further, Together

APSR MEETINGS

APSR Business Meetings

APSR Assembly Meetings



APSR 2015

Congress of the Asian Pacific Society of Respirology

APSR Meetings

APSR Business Meetings

Date	Time	Meeting	Venue
Thu 3 Dec	08:00 – 14:00	APSR Executive Committee Meeting	Room 301
	14:00 – 15:00	Councillors/Leaders of en bloc membership Meeting	Room 301
	15:00 – 16:00	APSR Assembly Heads Meeting	Room 301
	16:00 – 17:00	Annual General Meeting (AGM)	Room 301
Fri 4 Dec	09:00 – 10:00	Respirology & Respirology Case Reports Editorial Boards Meeting	Room 303

APSR Assembly Meetings

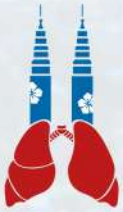
Date	Time	Meeting	Venue
Fri 4 Dec	11:00 – 12:00	Respiratory Neurobiology and Sleep	Room 303
	12:00 – 13:00	Paediatric Lung Disease	Room 303
	13:00 – 14:00	Cell and Molecular Biology	Room 303
	14:00 – 15:00	Asthma	Room 303
	15:00 – 16:00	Respiratory Structure and Function	Room 303
	16:00 – 17:00	COPD	Room 303
Sat 5 Dec	09:00 – 10:00	Environmental & Occupational Health and Epidemiology	Room 303
	10:00 – 11:00	Lung Cancer	Room 303
	11:00 – 12:00	Clinical Respiratory Medicine	Room 303
	13:00 – 14:00	Clinical Allergy & Immunology	Room 303
	14:00 – 15:00	Interstitial Lung Disease	Room 303
	16:00 – 17:00	Pulmonary Circulation	Room 303

Congress Overview

General Information

Guide for Chairs and Speakers

Social Events



APSR 2015

Congress of the Asian Pacific Society of Respirology

Congress Overview

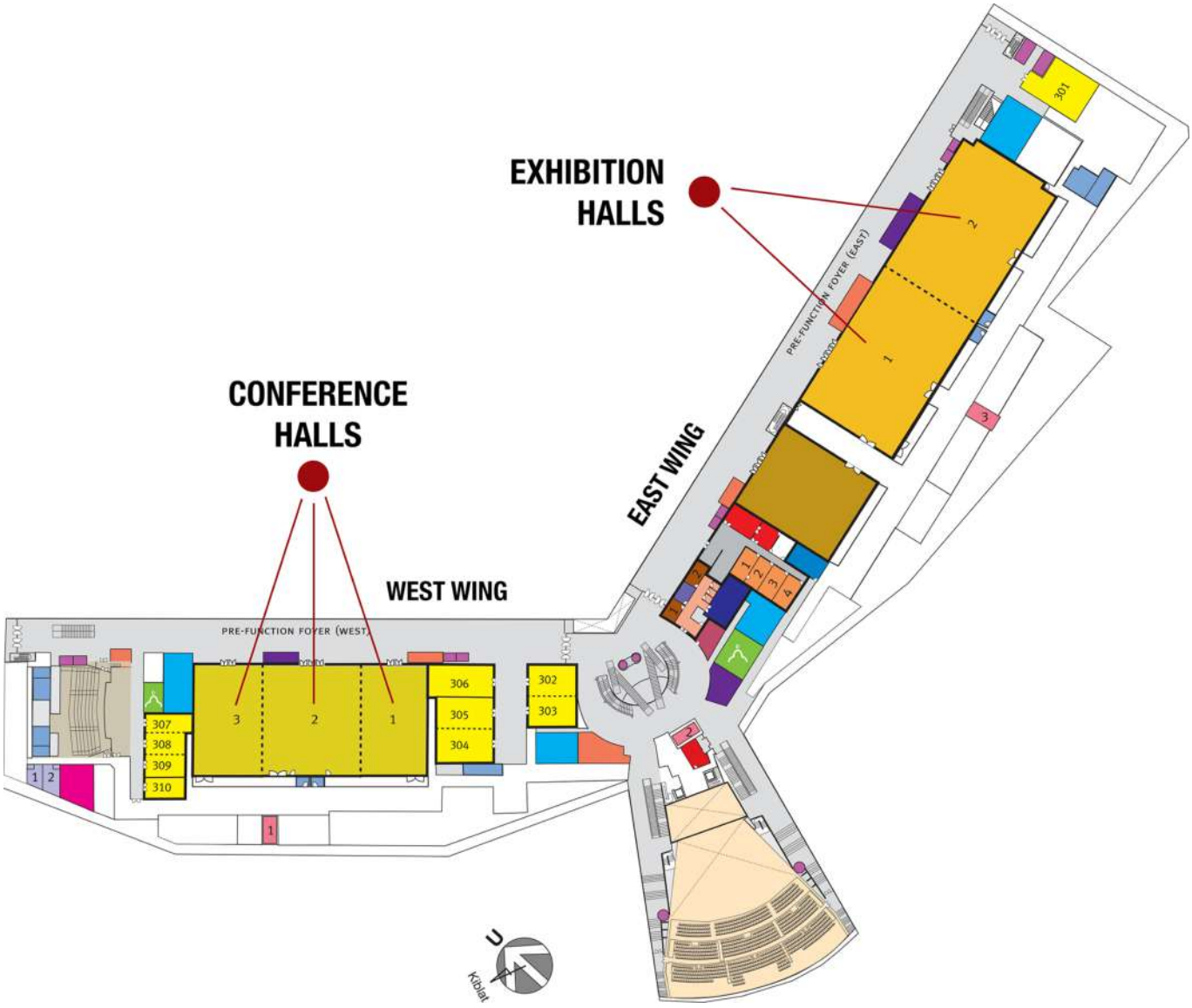
General Information

Congress Venue

Located at the heart of Kuala Lumpur city, the Kuala Lumpur Convention Centre is the city's most technologically-advanced, purpose-built facility.

The Centre is one of the components within PETRONAS' distinctive Kuala Lumpur City Centre (KLCC) development. Designed as a unique "city within a city", the 100-acre development offers a stimulating environment where one can work, live, shop, visit and simply enjoy the distractions of everyday living.

The congress takes place on Level 3 of the Kuala Lumpur Convention Centre spanning all across from the east wing to the west wing. Please refer to the venue map below:



Certificate of Attendance

Attendees may collect their certificate of attendance from 5th December onwards at the certificate collection counter on Level 3, West Wing using your badge.

Certificate of attendance for one-day registration can be collected from 4th December 14:00 onwards.

Accreditation

Delegates attending APSR Congress 2015 will be awarded 20 CPD points under the MMC CPD Grading System.

APSR Congress 2015 has also been accredited for 20 CPD points by the Malaysian Nursing Board.

Participants attending the Respiratory Neurobiology and Sleep Workshop will be awarded 6 CPD points under the MMC CPD Grading System.

Participants attending the other Workshops will be awarded 4 CPD points under the MMC CPD Grading System.

Exhibition

Exhibition takes place at the Ballroom located at Level 3, East Wing. Only attendees with a valid Delegate or Exhibitor badge may access the exhibition area.

Opening hours of the exhibition:

Fri, Dec 4	07:30 – 18:00
Sat, Dec 5	07:30 – 18:00
Sun, Dec 6	08:00 – 12:00

Lost and Found

Please contact the help desk at the Centre Core, Level 3.

Congress Overview

Guide for Chairs and Speakers

Oral Sessions

Chairs: Please come to the session room 10 minutes before your session begins and be seated on the seat allocated for you at the front of the hall marked as "Next Chair". Please take strict note of the scheduled time.

Speakers: For all speakers of oral presentations, please load your presentations at the Speakers Room (Room 307, West Wing, Level 3):

The opening hours for the speakers room are:

Thu, Dec 3	09:00 - 17:00 (Centre Core, Level 3)
Fri, Dec 4	07:30 - 17:00
Sat, Dec 5	07:30 - 17:00
Sun, Dec 6	07:30 - 12:00

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 desk located at the front 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" seat. Please do strictly keep within your presentation time.

Poster Sessions

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

Important Note About Timing

Timings for your poster on the Presentation Day

08:00	Poster pin-up*
17:00	Poster removal**

* Poster boards are located at the Ballroom, East Wing on Level 3. Your poster number will be indicated on the boards.

** Kindly remove your posters by 17:30 on the same day. Any poster that has not been collected will be discarded. The APSR Congress 2015 shall 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:

Cultural Showcase

There will be a cultural showcase comprising Malaysian arts and crafts in parallel with the opening ceremony and welcome reception.

Date: Thursday, 3rd December 2015

Time: 16:00 – 21:00

Venue: Conference Hall Foyer, Level 3

Opening Ceremony & Welcome Reception

Cocktail refreshments would be served from 19:00 onwards

Date: Thursday, 3rd December 2015

Time: 17:30 – 21:00

Venue: Conference Hall 1, Level 3

Gala Dinner

The Gala Dinner program promises you a night of good fun with a treat of Malaysian songs and dances. Tickets can be purchased at the Registration Counter at the Centre Core.

Date: Saturday, 5th December 2015

Time: 19:00 – 22:00

Venue: Banquet Hall, Level 3

Closing Ceremony

Date: Sunday, 6th December 2015

Time: 13:30 – 14:00

Venue: Conference Hall 1, Level 3

Presidential Dinner (by invitation only)

Date: Friday, 4h December 2015

Time: 19:00 – 22:00

Venue: Diamond Ballroom, Mandarin Oriental Hotel

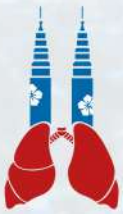
Programme at a glance

Thursday, 3rd December

Friday, 4th December

Saturday, 5th December

Sunday, 6th December



APSR 2015

Congress of the Asian Pacific Society of Respirology

Programme at a glance

Time	Day 1: Thursday, 3rd December 2015				
0800 - 0830	**Registration**				
0830 - 1030	**Respiratory Neurobiology & Sleep** Room 302	**Bronchoscopy & interventional Pulmonology** Room 303	**Thoracic Ultrasound & Pleuroscopy (Part 1)** Room 304	**Smoking Cessation** Room 306	**China-Malaysia Friendship Symposium** Conference Hall 1
1030 - 1100	**Coffee Break**				
1100 - 1230					
1200 - 1230		Sponsored Session: **Boston Scientific**			
1230 - 1330	**Lunch & Registration**				
1330 - 1500	**Respiratory Neurobiology & Sleep** Room 302	**Bronchoscopy & interventional Pulmonology (Part 2)** Conference Hall 2	**Thoracic Ultrasound & Pleuroscopy (Part 2)** Conference Hall 2	**Educational Seminar of the APSR – Interstitial Lung Disease (ESAP)** Room 306	**Non-invasive Ventilation** Room 304
1500 - 1530	**Coffee Break**				
1530 - 1700					
1700 - 1900	**Opening Ceremony** Conference Hall 1				
1900 - 2100	**Welcome Reception** Conference Hall Foyer				

Time	Day 2, Friday 4th December 2015		
0700 - 1700	**Registration**		
0700 - 0800	**Sunrise Session** Conference Hall 1	**Sunrise Session** Conference Hall 3	
0800 - 0815	**Opening Remarks** Conference Hall 2		
0815 - 0900	**Presidential Lecture** Conference Hall 2		
0900 - 1030	Concurrent Session 1A: **Asthma 1** Conference Hall 2	Concurrent Session 1B: **Chest Imaging** Conference Hall 3	Concurrent Session 1C: **Cell & Molecular Biology** Conference Hall 1
1030 - 1100	**Coffee Break**		
1100 - 1230	Concurrent Session 2A: **COPD** Conference Hall 2	Concurrent Session 2B: **Clinical Respiratory Medicine 1** Conference Hall 3	Concurrent Session 2C: **Environmental & Occupational Health & Epidemiology** Conference Hall 1
1200 - 1400	**Lunch**		
1230 - 1330	**Lunch Symposium 1** Boehringer Ingelheim (Malaysia) Sdn Bhd Conference Hall 2	**Lunch Symposium 2** Novartis Conference Hall 1	**Lunch Symposium 3** GlaxoSmithKline Pharmaceutical Conference Hall 3
1400 - 1530	Concurrent Session 3A: **Lung Cancer 1** Conference Hall 2	Concurrent Session 3B: **Respiratory Infection (non-TB)** Conference Hall 3	Concurrent Session 3C: **Respiratory Neurobiology & Sleep 1** Conference Hall 1
1530 - 1600	**Networking & Poster Viewing Sessions**		
1600 - 1730	Concurrent Session 4A: **Lung cancer 2** Conference Hall 2	Concurrent Session 4B: **Interstitial Lung Disease 1** Conference Hall 3	Concurrent Session 4C: **Pleural Diseases** Conference Hall 1
1730 - 1830	**Evening Symposium 1** Edmond Pharma – Recipharm Group Conference Hall 2	**Evening Symposium 2** Cipla Malaysia Sdn Bhd / Cipla Limited Conference Hall 1	
1900 - 2130	**Presidential Dinner** (by invitation only)		

Time	Day 3, Saturday 5th December 2015		
0700 - 1700	**Registration**		
0700 - 0800	**Sunrise Session** Conference Hall 1	**Sunrise Session** Conference Hall 3	
0815 - 0900	**Memorial Lecture: Michiyoshi Harasawa Research Award** Conference Hall 2		
0900 - 1030	Concurrent Session 5A: **Respiratory Neurobiology and Sleep 2** Conference Hall 2	Concurrent Session 5B: **Paediatric Lung Diseases 1** Conference Hall 3	Concurrent Session 5C: **Bronchoscopy & Interventional Pulmonology** Conference Hall 1
1030 - 1100	**Coffee**		
1100 - 1230	Concurrent Session 6A: **Asthma 2** Conference Hall 2	Concurrent Session 6B: **Paediatric Lung Diseases 2** Conference Hall 3	Concurrent Session 6C: **Pulmonary Circulation 1** Conference Hall 1
1200 - 1400	**Lunch**		
1230 - 1330	**Lunch Symposium 4** Boehringer Ingelheim (Malaysia) Sdn Bhd Conference Hall 1	**Lunch Symposium 5** Novartis Conference Hall 2	**Lunch Symposium 6** Chiesi and Orient EuroPharma Conference Hall 3
1400 - 1530	Concurrent Session 7A: **Young Investigator Session** Conference Hall 3	Concurrent Session 7B: **Clinical Allergy & Immunology** Conference Hall 2	Concurrent Session 7C: **Pulmonary Circulation 2** Conference Hall 1
1530 - 1600	**Networking & Poster Viewing Sessions**		
1600 - 1730	**The Great Debate** Conference Hall 2		
1730 - 1830	**Evening Symposium 3** Mundipharma Conference Hall 2	**Evening Symposium 4** AstraZeneca Conference Hall 1	
1900 - 2100	**Gala Dinner** Banquet Hall		

Time	Day 4, Sunday 6th December 2015		
0700 - 1700	**Registration**		
0700 - 0800	**Sunrise Session** Conference Hall 1	**Sunrise Session** Conference Hall 3	
0815 - 0900	**Memorial Lecture: Ann Janet Woolcock Research Award** Conference Hall 2		
0900 - 1030	Concurrent Session 8A: **Tuberculosis 1** Conference Hall 2	Concurrent Session 8B: **Respiratory Structure & Function** Conference Hall 1	Concurrent Session 8C: **Clinical Respiratory Medicine 2** Conference Hall 3
1030 - 1100	**Coffee**		
1100 - 1200	**Lunch Symposium 7** Boehringer Ingelheim (Malaysia) Sdn Bhd Conference Hall 2	**Lunch Symposium 8** Pfizer Conference Hall 1	
1200 - 1330	Concurrent Session 9A: **Tuberculosis 2** Conference Hall 2	Concurrent Session 9B: **Interstitial Lung Diseases** Conference Hall 3	Concurrent Session 9C: **Critical Care Medicine** Conference Hall 1
1330 - 1400	**Closing Ceremony** Conference Hall 2		

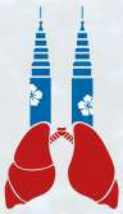
Detailed Programme

Thursday, 3rd December

Friday, 4th December

Saturday, 5th December

Sunday, 6th December



APSR 2015

Congress of the Asian Pacific Society of Respirology

Day 1

3rd December 2015

Smoking Cessation

Venue	**MEETING ROOM 306**
Chairperson	**Helmy Haja Mydin**
0800 - 0830	**Registration**
0830 - 0900	**Updates in Global Tobacco Control** *– Hayden McRobbie (New Zealand)*
0900 - 0930	**Tobacco and the lungs** *– Helmy Haja Mydin (Malaysia)*
0930 - 1000	**Smoking cessation therapies** *– Nurhayati Mohd Marzuki (Malaysia)*
1000 - 1030	Coffee break
1030 - 1100	**The challenges of implementing a smoking cessation service** *– Suthat Rungruanghiranya (Thailand)*
1100 - 1130	**Motivational Interviewing** *– Amer Siddiq Amer Nordin (Malaysia)*
1130 - 1200	**How to deal with the recalcitrant smoker** *– Wee Lei Hum (Malaysia)*
1200 - 1230	**The role of e-cigarettes in smoking cessation** *– Hayden McRobbie (New Zealand)*
1230 -1330	**Lunch**

Interstitial Lung Disease (ESAP)

Venue	**MEETING ROOM 306**
Chairperson	**Sundari Narayani K Ampikaipakan**
1330 - 1400	**Approach to the patient with ILD** – *John Simpson (UK)*
1400 -1430	**Pulmonary function testing in ILD - diagnostic and prognostic indicators** – *Takashi Ogura (Japan)*
1430 - 1500	**HRCT of the ILD – Basic Anatomy, Essential Patterns and Pearls for Interpretation** – *Masashi Takahashi (Japan)*
1500 -1530	**Histopathology of ILD** – *Masashi Takahashi (Japan)*
1530 -1600	Coffee break
1600 - 1630	**Pulmonary hypertension secondary to ILD** – *Sundari Ampikaipakan (Malaysia)*
1630 - 1700	**Advances in the treatment of ILD** – *Masahito Ebina (Japan)*
1700 - 1730	**Diagnostic pitfalls of sarcoidosis** – *John Simpson (UK)*

Bronchoscopy and Interventional techniques

Venue	**MEETING ROOM 303**
Chairpersons	**Siew-Teck Tie, Takashi Ishida**

PART 1

0800 - 0810	**Course introduction** *– Siew-Teck Tie (Malaysia)*
0810 - 0840	**Biopsy of the peripheral lung nodule - The role of Navigational Bronchoscopy** *– Chung-Ming Chu (Hong Kong)*
0840 - 0910	**Conventional TBNA - Is there a role in the era of EBUS?** *– Jamsak Tscheikuna (Thailand)*
0910 - 0940	**EBUS TBNA - Tips to improve the yield** *– Takehiro Izumo (Japan)*
0940 - 1010	**Airway debulking - Which is the best option?** *– Jamalul Azizi Abdul Rahaman (Malaysia)*
1010 - 1030	Coffee break
1030 - 1100	**Airway stenting in malignant obstruction** *– Hideo Saka (Japan)*
1100 - 1130	**Airway stenting in benign stricture** *– Philip Eng (Singapore)*
1130 - 1200	**Role of navigational bronchoscopy in the surgical management of lung cancer** *– Calvin S.H. Ng (Hong Kong)*
1200 - 1240	**Industry-sponsored Symposium** (Sponsored By: Boston Scientific) Chairperson: Jamalul Azizi Rahaman (Malaysia) **Bronchial Thermoplasty: How I do it** *– Gerard Cox (Canada)*
1240 - 1340	**Lunch**

Bronchoscopy and Interventional techniques

Venue	**CONFERENCE HALL 2**
Chairpersons	**Siew-Teck Tie, Soon-Hin How**

PART 2

1340 - 1700

Bronchial Thermoplasty
– Gerard Cox (Canada)

Rigid Bronchoscopy and airway stenting
– Hideo Saka (Japan)

EBUS-TBNA
– Takehiro Izumo (Japan)

Navigational Bronchoscopy
– Chung-Ming Chu (Hong Kong)

Endobronchial debulking
– Jamalul Azizi Rahaman (Malaysia)

Thoracic Ultrasound & Pleuroscopy

Venue	**MEETING ROOM 304**
Chairperson	**Fauzi Md Anshar**

PART 1

0800 - 0820	**Registration**
0820 - 0830	**Welcome Speech & Course Outline** *– Fauzi Md Anshar (Malaysia)*
0830 - 0900	**Thoracic ultrasound imaging - Introduction and normal anatomy** *– Anushya Vijayanathan (Malaysia)*
0900 - 0930	**Thoracic ultrasound imaging** • **Effusion** • **Infection** • **Pleural thickening** • **Lymph nodes** • **Lung masses** *– Anushya Vijayanathan (Malaysia)*
0930-1000	**Medical Pleuroscopy - Introduction** *– Anantham Devanand (Singapore)*
1000-1030	Coffee break
1030 - 1100	**Flexible pleuroscopy - How I do it** *– Pyng Lee (Singapore)*
1100 - 1130	**Rigid pleuroscopy - How I do it** *– Anantham Devanand (Singapore)*
1130 -1200	**Indwelling Pleural Catheter (IPC)** *– Hilmi Lockman (Malaysia)*
1200 - 1230	**Q & A**
1230 - 1330	**Lunch**

Thoracic Ultrasound & Pleuroscopy

Venue	**CONFERENCE HALL 2**
Chairperson	**Hilmi Lockman**

PART 2

1330 – 1700

Practical Session

4 groups - Ultrasound/ IPC/ Flexible pleuroscopy/ Rigid pleuroscopy

– *Pyng Lee (Singapore)*

– *Anushya Vijayanathan (Malaysia)*

– *Anantham Devanand (Singapore)*

– *Hilmi Lockman (Malaysia)*

– *Paras Doshi (Malaysia)*

– *Mat Zuki Mat Jaeb (Malaysia)*

– *Mohd Arif Mohd Zim (Malaysia)*

– *Fauzi Md Anshar (Malaysia)*

Respiratory Neurobiology & Sleep

Venue	**MEETING ROOM 302**
Chairperson	**Ahmad Izuanuddin Ismail**

0800 - 0820	**Registration**
0820 - 0830	**Welcome speech** *– Ahmad Izuanuddin Ismail (Malaysia)*
0830 - 0920	**Updates on Sleep-Related Breathing Disorders in International Classification of Sleep disorders 3rd Edition (ICSD – 3)** *– Kazuo Chin (Japan)*
0920 - 1010	**Polysomnographic assessment of SDB (OSA, CSA, Sleep-Related Hypoventilation in ICSD – 3)** *– Naricha Chirakalwasan (Thailand)*
1010 - 1040	Coffee break
1040 - 1130	**Sleep-Disordered Breathing in children and its effects** *– David Gozal (USA)*
1130 - 1220	**Clinical & Practical Pearls (1)** **Case discussion on Central Sleep Apnoea and therapy** *– Chol-Shin (South Korea)*
1220 - 1400	**Lunch**
1400 - 1450	**Clinical & Practical Pearls (2)** **Case discussion on Excessive Daytime Sleepiness (EDS)** *– Naricha Chirakalwasan (Thailand)*
1450 - 1540	**Advances in management of Sleep Disordered Breathing (SDB)** *– Atul Malhotra (USA)*
1540 - 1630	**Clinical & Practical Pearls (3)** **Case discussion on Parasomnia** *– Syed Hassan Syed Ahmad Almashoor (Malaysia)*

Non-Invasive Ventilation

Venue	**MEETING ROOM 304**
Chairpersons	**Ai-Khiang Goon, Asiah Kassim**

1330 - 1400	**The basics of NIV** *– Stefano Nava (Italy)*
1400 - 1430	**How to improve patient-ventilator synchrony** *– Stefano Nava (Italy)*
1430 - 1445	**Q & A**
1445 - 1515	**Non-Invasive Ventilation in Children - Evidence Based Considerations** *– David Gozal (USA)*
1515 - 1545	**How to set NIV in acute respiratory failure** *– Stefano Nava (Italy)*
1545 - 1600	Coffee break
1600 - 1730	**Illustrative case discussions** • Mask selection and fitting, • Ventilator settings, • and troubleshooting of various problems *– Stefano Nava (Italy)* *– David Gozal (USA)*

China-Malaysia Friendship Symposium

Venue	**CONFERENCE HALL 1**
Chairpersons	**Chun Xue Bai, Kai Wang, Yong-Kek Pang**
0815	**Arrival of Guests**
0830	**Arrival of VIPs** Mr Zhou Bin (Cultural Consular of the Malaysian China Embassy)
0835 – 0845	National Anthem of Malaysia National Anthem of China
0845 – 0855	**Welcome Speech** *– Yong-Kek Pang (Malaysia)* *– Chun Xue Bai (China)*
0855 – 0915	**Speech and Opening of China-Malaysia Friendship Symposium** *– Zhou Bin*
0915 – 0920	**Signing of Memorandum of Agreement** for collaboration of APSR and Chinese Society of e-Health to develop platforms for e-health **Presentation of Memorabilia** to Mr Zhou Bin by Prof Chun Xue Bai and Assoc Prof Yong-Kek Pang
0920 – 0930	**Slideshow of 40 years of Bilateral ties between Malaysia and China**
0930 – 1000	Coffee Break
1000 – 1015	**History of respiratory speciality in Malaysia** *– Abdul Razak Muttalif (Malaysia)*
1015 – 1030	**e-Health in China** *– Chun Xue Bai (China)*
1030 – 1045	**Lung Cancer in Malaysia** *– Chong-Kin Liam (Malaysia)*

1045 – 1100 **Challenges in precision medicine for lung cancer**
– Yue Hong Wang (China)

1100 – 1115 **Study on the molecular mechanism of COPD to lung cancer based on a microfluidic chip mimicking the microenvironment of tumor in vivo**
– Qi Wang (China)

1115 – 1130 **Regimens of enhancing response in lung cancer immunotherapy**
– Pingli Wang (China)

1130 – 1138 **New strategies to overcome HGF-mediated resistance to EGFR-TKIs in EGFR mutant NSCLC cells**
– Qian Chen (China)

1138 – 1146 **IGFBP5 promotes tumorigenicity and enhances stem-cell-like properties to lung cancer cells through Wnt signaling pathway**
– You Zhou (China)

1146 – 1154 **Respiratory endoscopy earns its specific spot in exploring immune evasion mechanism of lung cancer**
– Cheng Chen (China)

1154 – 1204 **Cancer-associated fibroblasts promote tumour progression in lung cancer by activation of H3K18ac**
– Xia Xu (China)

1204 – 1212 **The potential mechanism of SPP1 related extra cellular matrix remolding in lung adenocarcinoma development**
– Yong Zhang (China)

Day 2

4th December 2015

0700 – 1700

Registration

Venue **CENTRE CORE LEVEL 3**

0700 – 0800

Sunrise Session – ATS 2015 Discovery Series

0700 – 0800 **Past, Present and Future of Bronchoscopy** (Video Presentation)
– Atul C. Mehta
Venue: Conference Hall 1

0700 – 0800 **Asthma: The Emergence of Molecular Phenotyping and Its Impact on Therapy** (Video Presentation)
– Sally E. Wenzel
Venue: Conference Hall 3

0800 – 0815

Opening Remarks

Venue **CONFERENCE HALL 2**
– Roslina A Manap
– Michiaki Mishima

0815 – 0900

Presidential Lecture

Venue **CONFERENCE HALL 2**
Chairperson Roslina A Manap

COPD is a Systemic Disease - Importance of Comorbid Diseases
– Michiaki Mishima (Japan)

0900 – 1030

Session 1A: Asthma (1)

Venue	**CONFERENCE HALL 2**
Chairpersons	Kittipong Maneechotesuwan, Richard Li-Cher Loh

0900 – 0930	**Antibody treatment in asthma**
	– Elisabeth Bel (Netherlands)
0930 – 1000	**Anti-neutrophil treatment in asthma**
	– Peter Barnes (UK)
1000 – 1030	**Bronchial thermoplasty in asthma**
	– Gerard Cox (Canada)

Session 1B: Chest Imaging

Venue	**CONFERENCE HALL 3**
Chairpersons	Masashi Takahashi, Umadevi A Muthukumaru

0900 – 0930	**CT for infectious lung diseases**
	– Abdul Samad Sakijan (Malaysia)
0930 – 1000	**CT for COPD**
	– Nguyen Van Tho (Vietnam)
1000 – 1030	**CT for rare lung diseases**
	– Masashi Takahashi (Japan)

Session 1C: Cell & Molecular Biology

Venue	**CONFERENCE HALL 1**
Chairpersons	Takahide Nagase, Roslan Harun

0900 – 0930	**Recent progress of cellular and molecular biology in respiratory diseases**
	– Takahide Nagase (Japan)
0930 – 1000	**Generation of airway epithelial cells from iPS cells**
	– Isao Ito (Japan)
1000 – 1030	**Role of microbiomes in lung diseases**
	– Philips Hansbro (Australia)

1030 – 1100 Coffee Break

1100 – 1230

Session 2A: COPD

Venue	**CONFERENCE HALL 2**
Chairpersons	Diahn-Warng Perng, Tengku Saifudin Tengku Ismail

1100 – 1130	**Non-smoking COPD versus neutrophilic asthma: similarities and differences** *– Jorgen Vestbo (UK)*
1130 – 1200	**Asthma-COPD overlap syndrome: biomarker, phenotype or endotype-driven treatment** *– Diahn-Warng Perng (Taiwan)*
1200 – 1230	**Steroid insensitivity and target therapy for COPD: What do we need and where are we now** *– Peter Barnes (UK)*

Session 2B: Clinical Respiratory Medicine (Grand Round)

Venue	**CONFERENCE HALL 3**
Chairpersons	Hilmi Lockman, Abdul Razak Muttalif

1100 – 1130	**Case 1**: *Abdul Razak Muttalif (Malaysia)*
1130 – 1200	**Case 2**: *Sanjeev K. Mehta (India)*
1200 – 1230	**Case 3**: *Sanjay H. Chotirmall (Singapore)*

Session 2C: Environmental & Occupational Health & Epidemiology

Venue	**CONFERENCE HALL 1**
Chairpersons	Soon-Hee Jung, Mat Zuki Mat Jaeb

1100 – 1130	**E-cigarettes – The good, the bad and the ugly** *– Hayden McRobbie (New Zealand)*
1130 – 1200	**Asbestos ban and asbestos-related diseases in Asia** *– Soon-Hee Jung (South Korea)*
1200 – 1230	**Effect of inorganic dust on the lung** *– Takashi Nakano (Japan)*

1230 – 1330 Industry-sponsored Lunch Symposia

Heritage meets Innovation in the Management of COPD

Venue	**CONFERENCE HALL 2**
Chairpersons	Jamalul Azizi bin Abdul Rahaman
Sponsored by	Boehringer Ingelheim (Malaysia) Sdn Bhd

Burden of COPD in South East Asia & South Korea
– Lim Seong Yong

Heritage meets Innovation in the Management of COPD
– Peter MA Calverly

Bronchodilators - A breath of fresh air into the current thinking for COPD

Venue	**CONFERENCE HALL 1**
Chairpersons	Diahn-Warng Perng
Sponsored by	Novartis

The right treatment for the right patient: Does the use of inhaled corticosteroids in COPD reflect this?
– Klaus Rabe

Bronchodilators: A cornerstone of COPD treatment
– Christine Jenkins

Populations to patients : Personalised medicine in COPD

Venue	**CONFERENCE HALL 3**
Chairpersons	Richard Li-Cher Loh, Neil Snowise
Sponsored by	GlaxoSmithKline Pharmaceutical

Epidemiology of COPD in Asia Pacific: Understanding current disease and future risks
– Kourtney Davis

The evidence base for treatment decisions in COPD
– Paul Jones

Individualised treatment: where we are and where to go next?
– Chris Lai

1400 – 1530

Session 3A: Lung Cancer (1)

Venue	**CONFERENCE HALL 2**
Chairpersons	Kwun M Fong, Chong-Kin Liam

1400 – 1430	**What's new in the next revision of lung cancer staging** – *Kwun M Fong (Australia)*
1430 – 1500	**Molecular testing in advanced NSCLC** – *David Chi Leung Lam (Hong Kong)*
1500 – 1530	**Management of resistance to EGFR-TKIs and ALK-inhibitors** – *Chong-Kin Liam (Malaysia)*

Session 3B: Respiratory Infection (non-TB)

Venue	**CONFERENCE HALL 3**
Chairpersons	Sanjay H. Chotirmall, Fauzi Md Anshar

1400 – 1430	**Pulmonary aspergillosis** – *Sanjay H. Chotirmall (Singapore)*
1430 – 1500	**Antibiotic resistance in pneumonia - Asia Pacific perspective** – *Yuan-Lin Song (China)*
1500 – 1530	**Pulmonary melioidosis** – *Soon-Hin How (Malaysia)*

Session 3C: Respiratory Neurobiology and Sleep (1)

Venue	**CONFERENCE HALL 1**
Chairpersons	Kazuo Chin, Ahmad Izuanuddin Ismail

1400 – 1430	**OSA and COPD (Overlap syndrome) in Asian and Western countries** – *Patrick Gerard Moral (Philippines)*
1430 – 1500	**Obesity Hypoventilation Syndromes in Asian and Western countries** – *Kazuo Chin (Japan)*
1500 – 1530	**Recent advances in OSA and Sleep Medicine** – *Atul Malhotra (USA)*

1530 – 1600 Coffee Break

1600 – 1730

Session 4A: Lung Cancer (2)

Venue	**CONFERENCE HALL 2**
Chairpersons	David Chi Leung Lam, Chong-Kin Liam
1600 – 1630	**Evolving role of immunotherapy for NSCLC** – *Ross Soo (Singapore)*
1630 – 1700	**Small cell lung cancer: the evolving role of radiotherapy** – *Yoichi Nakanishi (Japan)*
1700 – 1730	**Update on Asian lung cancer screening: randomised and non-randomised studies** – *David Chi Leung Lam (Hong Kong)*

Session 4B: Interstitial Lung Disease (1)

Venue	**CONFERENCE HALL 3**
Chairpersons	Masahito Ebina, Helmy Haja Mydin
1600 – 1630	**Difficult-to-diagnose interstitial lung disease: the role of the multidisciplinary team** – *John Simpson (UK)*
1630 – 1700	**The role of CT scan in ILD - Historical Changes and Future Direction** – *Masashi Takahashi (Japan)*
1700 – 1730	**Advances in the treatment of IPF** – *Takashi Ogura (Japan)*

Session 4C: Pleural Diseases

Venue	**CONFERENCE HALL 1**
Chairpersons	Pyng Lee, Hilmi Lockman
1600 – 1630	**Unusual pleural diseases** – *Anantham Devanand (Singapore)*
1630 – 1700	**Management of complicated parapneumonic effusion and empyema thoracis** – *Pyng Lee (Singapore)*
1700 – 1730	**Current management of malignant mesothelioma** – *Thirugnanam Agasthian (Singapore)*

New perspectives for mucoactive drugs in COPD treatment: The RESTORE study

Venue	**CONFERENCE HALL 2**
Chairperson	Tengku Saifudin Tengku Ismail
Sponsored by	Edmond Pharma – Recipharm Group

Efficacy and safety of erdosteine in COPD: results of a 12-month prospective, multinational study
– Roberto W Dal Negro

Asthma and COPD Overlap Syndrome (ACOS) – An India Perspective

Venue	**CONFERENCE HALL 1**
Chairperson	Chong-Kin Liam
Sponsored by	Cipla Malaysia Sdn Bhd / Cipla Limited

Asthma COPD Overlap Syndrome – the Indian Perspective
– Harjit Dumra

Day 3

5th December 2015

0700 – 1700

Registration

Venue **CENTRE CORE LEVEL 3**

0700 – 0800

Sunrise Session

0700 – 0800 **Pulmonary Rehabilitation and field walking tests in COPD**
– Alejandro Casas (Colombia)
Venue: Conference Hall 1

0700 – 0800 **Paediatric Grand Round**
Venue: Conference Hall 3

0815 – 0900

Memorial Lecture: Michiyoshi Harasawa Research Award

Venue **CONFERENCE HALL 2**

– Hiroshi Kimura (Japan)

0900 – 1030

Session 5A: Respiratory Neurobiology and Sleep (2)

Venue	**CONFERENCE HALL 2**
Chairpersons	Kazuo Chin, Ahmad Izuanuddin Ismail
0900 – 0930	**The upper airway function in OSA** *– Peter Eastwood (Australia)*
0930 – 1000	**Relationship between OSA and gastroesophageal reflux** *– Patrick Gerard Moral (Philippines)*
1000 – 1030	**Cerebro-cardiovascular diseases in the Korean OSA cohort study** *– Chol Shin (South Korea)*

Session 5B: Paediatric Lung Diseases (1)

Venue	**CONFERENCE HALL 3**
Chairpersons	Albert Li, Anna Marie Nathan
0900 – 0930	**Acute management of childhood asthma – an update** *– Albert Li (Hong Kong)*
0930 – 1000	**Is intermittent use of inhaled corticosteroids a feasible option in children?** *– Anne Goh (Singapore)*
1000 – 1030	**Longer term outcome of childhood asthma – what do the longitudinal studies tell us?** *– Peter Le Souef (Australia)*

Session 5C: Bronchoscopy & Interventional Pulmonology

Venue	**CONFERENCE HALL 1**
Chairpersons	Takashi Ishida, K Kannan a/l Sivaraman Kannan
0900 – 0930	**Recent advances in diagnostic bronchoscopy** *– Lonny Yarmus (USA)*
0930 – 1000	**Complications of airway stenting** *– Philip Eng (Singapore)*
1000 – 1030	**Bronchoscopic management of tracheobronchomalacia/EDAC** *– Jamsak Tscheikuna (Thailand)*

1030 – 1100 Coffee Break

1100 – 1230

Session 6A: Asthma (2)

Venue	**CONFERENCE HALL 2**
Chairpersons	Kittipong Maneechotesuwan, Richard Li-Cher Loh

1100 – 1130	**Identifying therapeutic phenotypes of severe asthma** *– Kittipong Maneechotesuwan (Thailand)*
1130 – 1200	**What are the current and future treatment options for refractory asthma?** *– Omar Sharif Usmani (UK)*
1200 – 1230	**Role of biomarkers in severe asthma** *– Dave Singh (UK)*

Session 6B: Paediatric Lung Diseases (2)

Venue	**CONFERENCE HALL 3**
Chairpersons	Azizi Omar, Patrick Chan

1100 – 1130	**Update and controversies in the management of childhood pneumonia** *– Anne Chang (Australia)*
1130 – 1200	**Viruses in paediatric respiratory disease** *– Peter Le Souef (Australia)*
1200 – 1230	**Bronchiectasis: is it a reversible disease?** *Anne Chang (Australia)*

Session 6C: Pulmonary Circulation (1)

Venue	**CONFERENCE HALL 1**
Chairperson	Hiroshi Kimura, Ashari Yunus

1100 – 1130	**Screening and diagnosis of pulmonary hypertension** *– Aizai Azan Abd Rahim (Malaysia)*
1130 – 1200	**Mechanism and management of hypoxia-induced pulmonary hypertension** *– Sooronbaev Talant (Kyrgyzstan)*
1200 – 1230	**Novel therapy for acute pulmonary embolism** *– Heng-Joo Ng (Singapore)*

Beyond ICS/LABA: The Role of Tiotropium Respimat in Asthma

Venue	**CONFERENCE HALL 1**
Chairpersons	Abdul Razak bin Abdul Muttalif
Sponsored by	Boehringer Ingelheim (Malaysia) Sdn Bhd

Identifying the Symptomatic Patients on ICS/LABA
– Nicola Hanania

Treating Symptomatic Patients Beyond ICS/LABA: The Role of Tiotropium Respimat in Asthma
– Dina Diaz

Conversations in IgE-mediated severe asthma – Past, present and future

Venue	**CONFERENCE HALL 2**
Chairpersons	Peter Barnes
Sponsored by	Novartis

The evolving understanding of IgE in asthma
– Oscar Palomares

IgE in the real world: Applying knowledge for better patient outcomes
– David Price
– Jo Douglass

The Key Role of Small Airways in Asthma and COPD

Venue	**CONFERENCE HALL 3**
Chairpersons	Roslina A Manap
Sponsored by	Chiesi and Orient EuroPharma

An overview on Asthma and COPD in the Asian Pacific Counties
– Yong-Kek Pang

The clinical benefit of treating Asthma and COPD by reaching the small airways
– Dave Singh

1400 – 1530

Session 7A: Young Investigators Session

Venue	**CONFERENCE HALL 3**
Chairpersons	Peter Eastwood, Jane Bourke

E-cigarette and Conventional Cigarette Pro-Virulent Effects on Airway Colonizer and PathogenStaphylococcus aureus
– Laura E. Crotty Alexander (USA)

Performance of TB Diagnostic Committee (TBDC) in Certifying Disease Activity Among Smear Negative Retreatment Cases in District IV of Manila, Philippines from January 2013 to June 2014
– Caroline Bernadette Olanka King Kay (Philippines)

Cancer and OSA: Macrophages as Protagonists or Bystanders?
– Isaac Almendros (Spain)

Regression equations to estimate the two-minute walk distance (2MWD) in Malaysian adults aged 40 to 75 years
– Fatim Tahirah Mirza Mohd Tahir Beg (Australia)

MPR2 Upregulation via in situ Gene Delivery or via Engineered Endothelial Progenitor Cells Alleviates Pulmonary Arterial Hypertension in a Rat Model
– Rebecca L Harper (Australia)

1400 – 1530

Session 7B: Clinical Allergy & Immunology

Venue	**CONFERENCE HALL 2**
Chairpersons	Shu Hashimoto, Jessie de Bruyne

1400 – 1430	**Epithelial biology in the pathogenesis of airway inflammation** *– Yasuhiro Gon (Japan)*
1430 – 1500	**Recent advances in the pathogenesis of asthma** *– Elisabeth Bel (Netherlands)*
1500 – 1530	**The lung's response to air pollution** *– Ian Yang (Australia)*

Session 7C: Pulmonary Circulation (2)

Venue	**CONFERENCE HALL 1**
Chairperson	Hiroshi Kimura, Ashari Yunus

1400 – 1430	**The journey to PAH and PH-related respiratory diseases** *– Hiroshi Kimura (Japan)*
1430 – 1500	**Treatment update on CTEPH** *– Nobuhiro Tanabe (Japan)*
1500 – 1530	**Treatment update on PAH** *– Masayuki Hanaoka (Japan)*

1530 – 1600 Coffee Break

1600 – 1700

The Great Debate - Inhaled corticosteroids have a limited role in the management of COPD

Venue	**CONFERENCE HALL 2**
Chairperson	Diahn-Warng Perng, Tengku Saifudin Tengku Ismail
	Jorgen Vestbo (Pro) versus Peter Barnes (Con)

1730 – 1830 Industry-sponsored Evening Symposia

Exploring the bronchial tree in asthma: Probing deep into the small airways

Venue	**CONFERENCE HALL 2**
Chairperson	Aziah Ahmad Mahayiddin
Sponsored by	Mundipharma

Bypassing the oropharynx
– Omar S Usmani

Considerations for the Selection of ICS/LABA in the Treatment of Asthma
– Chul-Gyu Yoo

Treating the underlying inflammation to achieve asthma control

Venue	**CONFERENCE HALL 1**
Chairperson	Richard Li-Cher Loh
Sponsored by	AstraZeneca

Treating the underlying inflammation to achieve asthma control
– Christine Jenkins

1900 – 2100

Gala Dinner

Venue	**BANQUET HALL, LEVEL 3**

Day 4

6th December 2015

0700 – 1330

Registration

Venue **CENTRE CORE LEVEL 3**

0700 – 0800

Sunrise Session – ATS Discovery Series

0700 – 0800 **Mechanical Ventilation: From Vesalius to Ventilator-Induced Lung Injury** (Video Presentation)
– Arthur S. Slutsky
Venue: Conference Hall 1

0700 – 0800 **Two Billion and Counting: Reinvigorating the Battle Against Our Old Foe, TB** (Video Presentation)
– Trevor Mundel
Venue: Conference Hall 3

0815 – 0900

Memorial Lecture: Ann Janet Woolcock Research Award

Venue **CONFERENCE HALL 2**

Translating advances in research to improved health outcomes for patients with respiratory disease
– Ian Yang (Australia)

0900 – 1030

Session 8A: Tuberculosis (1)

Venue	**CONFERENCE HALL 2**
Chairperson	Charles Y. Yu, Abdul Razak Muttalif

0900 – 0930	**TB in Asia Pacific** *– Nobuyuki Nishikiori (WHO West Pacific Region)*
0930 – 1000	**Public-Private Mix in TB Control: Critical appraisal of global evidence and local applications** *– Charles Y. Yu (Philippines)*
1000 – 1030	**TB and migrants** *– Nobuyuki Nishikiori (WHO West Pacific Region)*

Session 8B: Respiratory Structure & Function

Venue	**CONFERENCE HALL 1**
Chairperson	Peter Eastwood, Yong-Kek Pang

0900 – 0930	**Role of handheld spirometer in the screening, diagnosis and monitoring of airflow limitation** *– Le Thi Tuyet Lan (Vietnam)*
0930 – 1000	**Diffusion capacity – pitfalls and interpretation** *– Peter Eastwood (Australia)*
1000 – 1030	**Tips and tricks to achieve best lung deposition with different inhaler devices** *– Yuko Komase (Japan)*

Session 8C: Clinical Respiratory Medicine (2)

Venue	**CONFERENCE HALL 3**
Chairperson	David S Hui, Hilmi Lockman

1400 – 1430	**Epidemiology and clinical data on MERS CoV** *– David S Hui (Hong Kong)*
1430 – 1500	**From SARS to MERS - What have we learned?** *– David S Hui (Hong Kong)*
1500 – 1530	**Emerging Infections: How can we improve preparedness?** *– Leo Lit Man Poon (Hong Kong)*

1030 – 1100 Coffee Break

1100 – 1200 Industry-sponsored Symposia

Idiopathic Pulmonary Fibrosis - Recent perspective and future challenges

Venue	**CONFERENCE HALL 2**
Chairpersons	Ganesh Raghu, Chong-Kin Liam
Sponsored by	Boehringer Ingelheim (Malaysia) Sdn Bhd

Lessons learned from recent IPF trials
– Ganesh Raghu
Acute exacerbations – a remaining challenge in IPF management
– Dong Soon Kim
Slowing disease Progression in IPF: the INPULSIS trials
– Ulrich Costabel

Pneumonia Prevention in Adults: Current Perspectives

Venue	**CONFERENCE HALL 1**
Chairperson	Termizy Hassan Mashat
Sponsored by	Pfizer

The CAPiTAStudy: Assessing the Public Health Impact
– Rontgene Solante
Preventing Pneumonia in Hajj Pilgrims
– Abdul Razak Bin Abdul Muttalif

1200 – 1330

Session 9A: Tuberculosis (2)

Venue	**CONFERENCE HALL 2**
Chairpersons	Erlina Burhan, Abdul Razak Muttalif
1200 – 1230	**New TB diagnostic tests** – *Somsak Rienthong (Thailand)*
1230 – 1300	**Challenges of MDR TB management** – *Harjit Dumra (India)*
1300 – 1330	**Diagnosis and treatment dilemma of nontuberculous mycobacterial infection** – *Erlina Burhan (Indonesia)*

Session 9B: Interstitial Lung Diseases (2)

Venue	**CONFERENCE HALL 3**
Chairpersons	Masahito Ebina, Helmy Haja Mydin
1200 – 1230	**Prevalence and incidence of ILD in the Asia-Pacific region** – *Yoshikazu Inoue (Japan)*
1230 – 1300	**Pathogenesis, diagnosis and treatment of co-existing emphysema and ILD** – *Masahito Ebina (Japan)*
1300 – 1330	**The ABC of hypersensitivity pneumonitis – from the acute to chronic stages** – *Tengku Saifudin Tengku Ismail (Malaysia)*

Session 9C: Critical Care Medicine

Venue	**CONFERENCE HALL 1**
Chairpersons	Yoshiki Ishii, Jyi-Lin Wong
1200 – 1230	**Diagnosis of ventilator-associated pneumonia – difficult but do-able** – *John Simpson (UK)*
1230 – 1300	**Significance of the Berlin definition and current status of ARDS treatment** – *Yoshiki Ishii (Japan)*
1300 – 1330	**Early mobilisation of the ventilated patient: from evidence to practice** – *Li-Ling Tai (Malaysia)*

1330 – 1400 Closing Ceremony

Venue **Conference Hall 2**

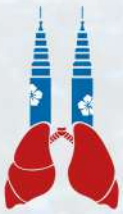
Speaker Abstracts

Thursday, 3rd December

Friday, 4th December

Saturday, 5th December

Sunday, 6th December



APSR 2015

Congress of the Asian Pacific Society of Respirology

Day 1: Thursday, 3rd December 2015

Session: **Smoking Cessation**
Time: **0930 – 1000**
Venue: **Meeting Room 306**

Smoking cessation therapies

– Nurhayati Mohd Marzuki (Malaysia)

Tobacco is the only causative agent that leads to all non-communicable diseases. The effect of tobacco smoking is beyond the user alone. It is estimated 1 billion people will die in 21th century due to tobacco use.

In 2005, a World Health Organisation treaty, the Framework Convention of Tobacco Control was signed by 40 parties. In the past decade, the number of signatories has risen to 180, covering 90% of the world population.

Article 14 FCTC states: “Demand reduction measures concerning tobacco dependence and cessation”. It translate into Offer Help to Quit Tobacco Use as part of MPOWER strategy.

The call to action in Tobacco Atlas 2015 stated: “Governments should subsidise all aspects of individual- and group-level cessation while simultaneously employing strong population-based cessation strategies.”

There are multiple strategies employed for smoking cessation. It has been proven that combination of medical therapy and counselling strategy leads to the higher quit rate.

The duration of session, number of sessions, number of health personnel involved and type of medications used whether single or in combination affect smoking cessation success.

Day 1: Thursday, 3rd December 2015

Session: **Smoking Cessation**
Time: **1030 – 1100**
Venue: **Meeting Room 306**

The challenges of implementing a smoking cessation service

– Suthat Rungruanghiranya (Thailand)

Although Thailand has been quite successful in limiting the number of new smokers, there are still more than 11 million current smokers nationwide. Despite of several strong campaign efforts, the smoking rate remains disappointingly plateau. One of the most important reasons could be attributed by the lack of effective systematic, treatment system. Without establishing the good treatment strategies, the number of Thai smokers would be unlikely to drop any further.To successfully implement the effective smoking cessation services, several challenges must be overcome. Firstly, healthcare personnel (HCP)'s attitudes, knowledge, and beliefs has to be improved. For the young generation of HCP, smoking cessation module has been added to the curriculum of all HCP.Meanwhile, the national clinical practice guideline was developed for the first time in the country. Instead of using awidely-accepted complicated American-based 5A scheme, a simplified 3-step Thai "Sabai (to feel comfortable)" protocol, including ask, treat, and follow-up has been developed and recommended in order to avoid the language barrier and complexity of 5A scheme. This simplified "Sabai" protocol was then implemented nationwide.

To strengthen the system, a network of qualified smoking cessation clinics has been developed. Starting from only 5 clinics in Bangkok, the number went up to 330 clinics nationwide in 5 years. All clinicsare required to use the abovementioned "Sabai" protocoland build up their own community-based cessation system, led by trained health volunteers.This system was also supported by another community-based campaign, 1 health-volunteer for 1 quitter. Monks and other religious leaders were also trained to help local smokers in their communities. Smokers who are unable to quit using the community-based system will receive additional aids from the clinics. Annual awards have also been provided to the clinics that have good clinical performance. Mobile applications were also made and distributed in order to strengthen the service. Thus far, the success rate at 6 months of our clinic network reaches 45%. More than 50,000 smokers have been treated through our network.

In conclusion, our smoking cessation system in Thailand is getting stronger, although a lot more is still needed to be done. Our system is on its way towards the complete community-based cessation.

Day 1: Thursday, 3rd December 2015

Session: **Interstitial Lung Disease (ESAP)**
Time: **1430 – 1530**
Venue: **Meeting Room 306**

HRCT of the ILD – Basic Anatomy, Essential Patterns and Pearls for Interpretation

– *Masashi Takahashi (Japan)*

1. Normal Anatomy of the Peripheral Lung

Starting from the trachea, the airway reaches the level of the lobular bronchus with a diameter of 1mm after branching dichotomously 9 to 14 times. The lobular bronchus supplies 3 to 5 terminal bronchioles and the area supplied by each terminal bronchiole is called an "acinus". Therefore, the secondary lobule consists of 3 to 5 acini. Intralobular bronchioles arise at an interval of 1 to 2 mm while the pre-lobular bronchus arises at intervals of 0.5 to 1.0 cm. The secondary lobule is surrounded by the peri-lobular structures including interlobular septum and pleura. It should be noted that large bronchovascular bundle or pulmonary vein can be recognized as perilobular structures because many secondary lobules are adjacent to these extralobular structures.The airway is usually accompanied by the pulmonary artery, even at the level of the secondary pulmonary lobule. The area around the tip of the terminal bronchiole is called the "centrilobular portion" or "centriacinar portion". The pulmonary vein runs between the pulmonary segment and subsequently connects to the interlobular septum. The distance from the centrilobular portion to the peripheral structures including the interlobular septum, pulmonary vein, and pleura is constant at approximately 2.5mm.

Alveolar interstitium is a matrix among the basement membrane of pneumocytes and capillary. Type I pneumocyte has a role of gas exchange and Type II secretes surfactant to prevent alveolar collapse. Because the thickness of alveolar interstitium is about 0.4μm and is under the resolution of CT , it is impossible to be visualized directly even by state-of-art HRCT technology. However once the interstitial pneumonitis occur and thickening of alveolar interstitium becomes obvious, this structure can be recognized with increased density of the lung on CT.

There is another type of pulmonary interstitium which is called lymphatic interstitium. This type of interstitium is found at interlobular septum, pleura, pulmonary vein and bronchovascular bundle. Figuratively speaking, these structures can be recognized as “framework of the lung”. These structures have rich networks of lymphatic channels and many pathological processes involve this interstitium including granulomatous disease, neoplastic change as well as exudative or edematous changes.

2. Basic HRCT Findings of ILD

Increased lung density

The essential pathology of interstitial pneumonitis(IP) is an inflammatory thickening of alveolar interstitium and sometimes with exudative collection in the alveolar lumen. As noted above, it is impossible to visualize this structure directly even by state-of-art HRCT technology. However once the interstitial pneumonitis occur and thickening of alveolar interstitium becomes obvious, this structure can be recognized with increased density of the lung on CT. The degree of this change is correlating to the ratio of residual gas of alveolar lumen and interstitial structures reflecting from a faint GGO to dense consolidation.

Architectural distortion

Some types of IP show architectural distortion of the involved lung due to increased fibrotic change with progression of the disease. The well-known architectural distortion is honeycomb and traction bronchiectasis. These changes are hallmarks for making diagnosis of irreversible fibrotic process of the IP such as UIP. Honeycombing is defined as the cystic spaces with clustered and share walls, predominantly subpleural surface in several layers.In UIP, the distribution of the honeycombing is exclusively subpleural portion of lower lung field.

Other associated CT patterns of IP

Thin walled cysts are frequently observed in the CT images of LIP. In some cases of DIP, thin walled cysts also can be recognized.

Centrilobular nodules or GGO can be observed in the CT images with COP, RB-ILD, Chronic HP and LIP.

Cross sectional distribution of the opacity

Most of the idiopathic ILD has a tendency to show subpleural distribution. However, in some cases of IP with CVD tends to show peri-bronchovascular distribution. Inner zone of the lung is sometimes affected in the cases with acute IP such as AIP, CVD associated pneumonia or drug induced pneumonia.

Day 1: Thursday, 3rd December 2015

Session: **Bronchoscopy and Interventional techniques**
Time: **0810 – 0840**
Venue: **Meeting Room 303**

Biopsy of the peripheral lung nodule - The role of Navigational Bronchoscopy

– Chung-Ming Chu (Hong Kong)

Solitary pulmonary nodule (SPN) is a common medical problem. The key concern is it being an early asymptomatic lung cancer [1-2]. Small SPNs are increasingly detected by CT scan, often done for other purposes, or as screening for lung cancer. The burden of SPN is higher in Asia, and the probability of it being malignant is less predictable because tuberculosis is endemic in Asia and a significant proportion of lung cancer patients in Asia are non-smoker [3].

Because of the size, some smaller SPN cannot be biopsied with conventional bronchoscopic biopsy. Transthoracic needle aspiration under CT guidance may not be suitable for deeply seated SPNs because of pneumothorax risk. Conventionally, SPNs which cannot be biopsied can be followed by serial CT scans or resected by surgical means. Neither method is ideal because there are false negatives (delayed diagnosis of lung cancer), or false positives (unnecessary surgery for benign pathologies). Both approaches are also resource intensive.

Electromagnetic navigation bronchoscopy (ENB) can improve transbronchial biopsy yield by 70 – 90%, obviating serial CT monitoring or unnecessary surgical resection in many cases [4,5]. It extends the current capability of bronchoscopists and localization techniques (fluoroscopy, endoscopic ultrasound) to reach and biopsy small SPNs. ENB consists of 2 components: (i) a software to construct a virtual bronchoscopy (VB) image that allows biopsy pathway planning using available CT thorax data; (2) an electromagnetic field generator and sensors to guide the bronchoscope to reach the target(s). The American College of Chest Physicians (Grade 1C evidence) recommends that in patients with peripheral lung lesions difficult to reach with conventional bronchoscopy, electromagnetic navigation guidance is recommended if the equipment and the expertise are available [1].

In this presentation, the theoretical and technical aspects of ENB and related techniques will be discussed.

References:

1. Detterbeck FC, et al. Executive summary. Diagnosis and management of lung cancer, 3rd ed: American College of Chest Physicians evidence-based clinical practice guidelines. Chest 2013;143:7S – 37S.
2. Gould MK, et al. Evaluation of individuals with pulmonary nodules: when is it lung cancer? Chest 2013;143:e93S – e120S.
3. Bai CX, Choi CM, Chu CM, et al. Evaluation of pulmonary nodules: clinical practice consensus guidelines for Asia. (Submitted for review)
4. Eberhardt R, et al. Multimodality bronchoscopic diagnosis of peripheral lung lesions. A randomized controlled trial. Am J Respir Crit Care Med 2007;176:36-41.
5. Eberhardt R, et al. Electromagnetic navigation diagnostic bronchoscopy in peripheral lung lesions. Chest 2007;131:1800-1805.

Day 1: Thursday, 3rd December 2015

Session: **Bronchoscopy and Interventional techniques**
Time: **0910 – 0940**
Venue: **Meeting Room 303**

EBUS TBNA - Tips to improve the yield

– Takehiro Izumo (Japan)

Endobronchial ultrasound guidedtransbronchial needle aspiration (EBUS-TBNA) is a widely used minimally invasive procedure that has been shown to have a high sensitivity and diagnostic yield for detecting metastasis to hilar and mediastinal lymph nodes (LNs). Here are some tips for an efficient EBUS-TBNA:(1) Puncture precisely between cartilages (Outer sheath method); (2) Move the needle slider quickly when you push forward into the lesionthen pull it back slowly to scrape off the tissues; (3) Avoid puncturing necrotic areas as much as possible. In particular, (1) is important because pieces of cartilage tend to get stuck inside the needle if you insert it forcefully and carelessly, resulting in failure to collect tissue samples. The Outer Sheath Method is recommended to confirm the space between cartilages. After confirming the target site on ultrasound, move the bronchoscope back and forth while pressing the outer sheath against the mucous membrane of trachea/ bronchus. The ultrasonogram will shift upward and become clearer when the outer sheath has been lodged in between cartilages, after which wedge the outer sheath by flexing the scope at a maximum upward angle. This is the art of doing a smooth EBUS-TBNA to obtain tissue samples.

Recently, elastography, a new ultrasonography-associated technology that measures tissue compressibility, was introduced. In principle, pathophysiological processes, such as malignancy,make tissues less deformable or stiff. Elastography, is a new non-invasive EBUS modality that is hypothesized to predict mediastinal and hilar nodal metastasis based on hardness of tissue. Based on our results, we propose a simple EBUS elastography classification that could predict with 96.7% accuracy the presence or absence of mediastinal and hilar nodal metastasis. Type 1 (predominantly non-blue) indicates a benign pathology; Type 2 (part blue, part non-blue) is equivocal; and Type 3 (predominantly blue) indicates malignancy. EBUS elastography is a useful tool with very high sensitivity, specificity and accuracy for differential diagnosis of mediastinal and hilar LNs.

In this lecture and hands-on session, our arts of EBUS-TBNA and new technologywill be presented.

Day 1: Thursday, 3rd December 2015

Session: **Bronchoscopy and Interventional techniques**
Time: **0940 – 1010**
Venue: **Meeting Room 303**

Airway debulking - Which is the best option?

– Jamalul Azizi Abdul Rahaman (Malaysia)

Central airway (trachea and mainstem bronchi) tumours are a common cause of airway obstruction with the most common tumour being bronchogenic carcinoma. Besides advanced primary lung tumours, central airway obstruction (CAO) can also be due to a variety of malignant causes such as tracheal tumours (primary or secondary), lymphoma and non-malignant causes such as papillomas, hamartomas, foreign body and systemic diseases.

The clinical presentation varies from slowly progressive cough and dyspnoea to rapidly developing respiratory distress. Therapeutic bronchoscopy is usually reserved for patients with inoperable disease or in those with emergent airway obstruction. It is also utilised for debulking of tumours prior to surgery to stabilise the patient prior to surgery. A close collaboration among interventional pulmonologists, radiologists, anaesthesiologists and thoracic surgeons is essential for optimal outcome in these challenging situations. Immediate goals of the therapy are to secure the airway and to restore the patency of the airway lumen.

Several therapeutic bronchoscopy techniques are available using flexible or rigid bronchoscope to perform airway debulking. The choice of technique depends on the underlying cause, type of obstruction, severity of CAO, availability of instruments and expertise.

Therapeutic bronchoscopy for airway debulking of endobronchial tumours can be carried out using either rigid or flexible bronchoscope or both and the methods include rigid bronchoscopic coring (and removal with biopsy forceps), laser photoresection, electrocautery, argon plasma coagulation (APC), microdebrider and cryorecanalization. A combination of these procedures is usually successful in rapid palliation of symptoms and in selected cases paves the way for further treatments such as external beam radiation, chemotherapy and surgery.

Rigid bronchoscopy facilitates the management of tracheobronchial tumours by restoring ventilation to collapsed lung, allowing stent placement and removal of benign tumours (typical bronchial carcinoid, endobronchial hamartoma) with a curative intent. Mechanical debulking using rigid bronchoscope by itself is an effective method to achieve rapid recanalization of central airways.

In the absence of large randomized controlled trials in this field to guide the therapy, the choice of therapy also depends on the availability of trained personnel and equipment at the facility, personal preference and institution-specific protocols. In many instances, a combination of procedures is employed to achieve optimal results.

Day 1: Thursday, 3rd December 2015

Session: **Thoracic Ultrasound & Pleuroscopy**
Time: **0830 – 0930**
Venue: **Meeting Room 304**

Thoracic ultrasound imaging - Introduction and normal anatomy

– Anushya Vijayanathan (Malaysia)

Ultrasound is a diagnostic modality that first gave the world a glimpse of the "insides" of the human body. Since its inception in the early 20th century, diagnostic ultrasound has progressed in leaps and bounds in tandem with the age of global technology. We have seen the image quality and resolution change over the years from lines and dots to almost picture perfect three and four dimensional images. Many mathematical equations and physics principles are responsible for this miracle.

Ultrasound will always be a popular diagnostic imaging modality due to its lack of ionizing radiation, portability and patient comfort. It is an asset to be able to perform an ultrasound examination and perform it well. Due to its large dependency on the operator, proper training and knowledge is essential to ensure accurate diagnosis.

Thoracic ultrasound has many advantages over other imaging modalities, especially for the pleura. These include, the absence of radiation, portability and real-time imaging.It is more sensitive in the detection of pleural fluid from consolidation in a "white out" chest radiograph. Although CT scan is the imaging modality of choice for the chest, there are advantages with ultrasound in differentiation of pleural fluid, pleural masses and thickening. Its portability also makes ultrasound a popular choice in the imaging of an ill patient in the Intensive care setting.

The most revolutionary role of ultrasound in medicine is its ability as a guide to the targeted placement of needles and tubes.

The use of ultrasound guidance for thoracentesis has remarkably reduced the complication rate of this procedure. Similarly, ultrasound guided biopsies have increased the yield of the tissue and the success of the procedure.

In conclusion, ultrasound is a safe, reliable, cost effective diagnostic tool, but the operator has to ensure that he or she has had adequate training before embarking on this journey of diagnostic and therapeutic ultrasound.

Day 1: Thursday, 3rd December 2015

Session: **Thoracic Ultrasound & Pleuroscopy**
Time: **0930 – 1000**
Venue: **Meeting Room 304**

Medical Pleuroscopy - Introduction

– Anantham Devanand (Singapore)

Pleuroscopy has been described as a 'window' to the pleural space that facilitates both diagnostic and therapeutic interventions. Both rigid and semi-rigid equipment are available. Pleuroscopy is different from Video-Assisted Thoracoscopic Surgery (VATS) because of the absence of general anesthesia, single lung ventilation or multiple ports. Instead pleuroscopy is often performed in an endoscopy suite under moderate sedation and local anesthesia. In addition, pleuroscopy is primarily a procedure that is focused on the parietal pleura and any interventions on the lung are strictly reserved for expert practitioners who have the necessary expertise and equipment. The three basic principles of pleuroscopy are pleural drainage, induction of a pneumothorax and the use of an endoscope.

Anatomic considerations include surface anatomy and identification of the 'safe triangle' on the thorax to avoid inadvertent laceration of vessels or nerves. The location of the diaphragm in the lateral decubitus position is also important to ensure safe entry into the pleural space. The use of bedside ultrasound has made it possible to target smaller or loculated pleural effusions. Physiological considerations include ventilation-perfusion mismatching in the lateral decubitus position and this can be worsened when a pneumothorax is induced on the non-dependent side.

Indications for pleuroscopy include the work-up of pleural effusions of unknown origin. The diagnostic yield for malignant and tuberculous effusions is >90%. False negatives in the diagnosis of malignant effusions are due to pleural adhesions limiting inspection of pleural space, early mesothelioma that can present as pleuritis and operator inexperience resulting in non-representative biopsies. False negatives can be avoided by taking multiple biopsies systematically, removing necrotic fibrin before sampling, performing deep biopsies to assess chest wall infiltration, performing adhesiolysis to visualize the entire pleural cavity and communicating with the pathologist. Furthermore, non-diagnostic pleuroscopy i.e. where the pathology result is non-specific pleuritis, needs to be followed up because of the <20% chance of recurrence of pleural effusion and <10% risk of malignancy, which is usually malignant mesothelioma.

Therapeutic indications include pleurodesis via talc poudrage for both malignant effusion and pneumothorax recurrence prevention. Possible advantages of pleuroscopic talc poudrage over bedside talc slurry include identification of entrapped lung, complete drainage of pleural cavity under visual guidance and optimal placement of chest drains. In addition, pleuroscopic talc insufflation can be followed by inspection to ensure all pleural surfaces are coated with an even distribution of talc.

The fibrinopurulent stage of empyema will be the ideal window of opportunity for intervention using pleuroscopy with adhesiolysis, drainage and optimal chest tube placement under direct vision. Patients who have progressed to the organizational stage often will require decortication by a thoracic surgeon. Additional benefits of a pleuroscopic approach include exclusion of malignancy, pleural biopsies for microbiological diagnosis and saline irrigation of the pleural cavity.

Advanced procedures that are strictly reserved for experts include resection of blebs and bullae, lung biopsy for the diagnosis of parenchymal lung disease, sympathectomy for hyperhidrosis management and creation of a pericardial window.

The absolute contraindications for pleuroscopy are a lack of a pleural space, type 2 respiratory failure not attributable to the effusion, positive pressure ventilation and uncorrectable coagulopathy. Relative contraindications are ongoing sepsis (besides empyema), uncontrolled cough, unstable hemodynamics, inability to lie in the lateral decubitus position for at least 1 hour, pulmonary arterial hypertension, superior vena cava obstruction, morbid obesity and chest wall deformity. We can reduce complications by postponing pleuroscopy if the patient is coughing, checking arterial blood gases, having continuous ECG monitoring, oxygenating all patients during the procedure, avoiding biopsy in the lung fissures or mediastinum and keeping the chest tube until the lung re-expands.

Day 1: Thursday, 3rd December 2015

Session: **Thoracic Ultrasound & Pleuroscopy**
Time: **1100 – 1130**
Venue: **Meeting Room 304**

Rigid Pleuroscopy - How I do it

– Anantham Devanand (Singapore)

The benefits of rigid pleuroscopy include larger biopsy specimens and more efficient adhesiolysis is a complex pleural space. With the use of electrocautery, bloodless lung biopsies are also possible. The trade-off is that the rigid scope may be unfamiliar to pulmonologists who are experienced in flexible bronchoscopy.

The pre-procedure checklist includes valid informed consent, exclusion of contraindications, routine laboratory tests such as PT/PTT, platelet counts and serum creatinine, prophylactic antibiotics (iv Cefazolin 2g within 60 minutes of skin incision) and marking the side of procedure. Conscious or moderate sedation is used with spontaneous breathing. Risks include apnoea, aspiration, as well as hemodynamic instability from drugs, vagal stimulation and excessive pleural fluid evacuation.

The endoscopy room is set up with the endoscopist, scope, patient and monitor in the correct position to avoid disorientation. Two sterile trolleys are set up: a scope trolley and a chest tube insertion trolley with the addition of the trochar.

Trochar insertion starts with local anesthesia infiltration of the 4 layers of the chest wall: epidermis, thoracic muscle aponeurosis, intercostal muscles and parietal pleura. A single port technique is less painful but multiple ports are needed for adhesiolysis. Inadvertent lung perforation can cause pulmonary hemorrhage, empyema or pneumothorax. This is prevented by not introducing the trochar when you cannot aspirate pleural fluid, using ultrasound to mark the entry site and by performing extended pleuroscopy using an open technique.

Pleuroscopy starts with inspection of the pleural cavity. The lung fissures and the diaphragm are used for orientation. Pleuroscopy for the diagnosis of malignant pleural effusions facilitates assessment of intrapleural tumor burden by inspection of the visceral pleura (to identify lung entrapment), diaphragmatic pleura, chest wall parietal pleura and pericardium. During pleural biopsy with forceps, performing the biopsy over ribs avoids intercostal neurovascular bundle damage.

Intra-procedure complications include vagal syncope at the time of pleural entry, pain at trochar insertion site, cough when pleural fluid is suctioned out and bleeding post biopsy. A chest tube is retained post procedure until daily drainage is < 150 ml; or there is lung re-expansion and radiological evidence of complete drainage. Early complications include wound site discomfort, wound infections, air-leak, subcutaneous emphysema, fever, increased oxygen requirements post pleurodesis, sub-optimal chest tube placement, bleeding, pneumonia and pulmonary embolism. Talc pneumonitis is reduced by using graded talc of > 10 microns. Late complications include failed pleurodesis, empyema, pleurocutaneous fistula and tumor seeding at the pleuroscopy entry site.

Day 1: Thursday, 3rd December 2015

Session: **Respiratory Neurobiology & Sleep**
Time: **0830 – 0920**
Venue: **Meeting Room 302**

Updates on Sleep-Related Breathing Disorders in International Classification of Sleep disorders 3rd Edition (ICSD – 3)

– Kazuo Chin (Japan)

International Classification of Sleep Disorders (3rd Edition) was published in 2014. There are 17 sleep related breathing disorders (SRBDs) and 2 isolated symptoms and normal variants. Eighteen SRBDs were categorized into 4 groups: obstructive sleep apnea disorders, central sleep apnea syndromes, sleep related hypoventilation disorders and sleep related hypoxemia disorders. Isolated symptoms and normal variants were snoring and catathrenia. We will note a number of significant content changes from the 2nd edition. The diagnostic criteria for obstructive sleep apnea in adults have been changed. Within the Central Sleep Apnea Syndromes, a diagnosis of treatment-emergent central sleep apnea now appears. This term is preferred to the widely used term complex sleep apnea. Assigning a diagnosis of a sleep related hypoventilation disorder requires demonstration of elevated $PaCO_2$ (by direct measure of arterial blood gas or, more commonly, by proxy measures such as end-tidal or transcutaneous CO_2 determination). The new diagnosis of sleep related hypoxemia disorder should be employed when there is sustained drop in SaO_2 but $PaCO_2$ has not been measured. Obesity hypoventilation is categorized in one of SRBDs, while it was in sleep related hypoventilation/hypoxemia due to neuromuscular and chest wall disorders in the 2nd version. Thus, we will discuss the contents of ICSD-3 in this section.

Day 1: Thursday, 3rd December 2015

Session: **Respiratory Neurobiology & Sleep**
Time: **0920 – 1010**
Venue: **Meeting Room 302**

Polysomnographic assessment of SDB (OSA, CSA, Sleep-Related Hypoventilation in ICSD – 3)

– Naricha Chirakalwasan (Thailand)

Polysomnography can be divided into 4 types.[1]

Type 1: full attended polysomnography (≥ 7 channels) conducted in alaboratory setting
Type 2: full unattended polysomnography (≥ 7 channels)
Type 3: limited channel devices (usually using 4–7 channels)
Type 4: 1 or 2 channels usually using oximetry as 1 of theparameters

Generally type 1 is a preferred test since it will provide most accurate information and it is also allowed conversion to PAP titration study if needed (split-night polysomnography). Typical montage includesstandard EEG including frontal leads (F1, F2), central leads (C3, C4), occipital leads (O1, O2),reference leads at mastoids (M1, M2), electromyography, and electrooculography. Oxygen Saturation(SpO2) was measured with a finger probe. Air flow was measured by 2 methods: nasal pressure transducer and oronasal thermal sensor. The thoracic and abdominal respiratory movements were monitored by respiratory inductance plethysmography. Carbon dioxide monitoring in the form of end-tidal CO2 (ETCO2) or transcutaneous CO2 (TcCO2) may be utilized when hypoventilation is suspected.

Respiratory events can be divided to 3 types. [2]

1. Apnea: score a respiratory event as an apnea when BOTH of the following criteria are met:

a. There is a drop in the peak signal excursion by ≥90% of pre-event baseline using an oronasal thermal sensor (diagnostic study), PAP device flow (titration study) or an alternative apnea sensor (diagnostic study)

b. The duration of the ≥90% drop in sensor signal is ≥10 seconds.

1.1 Obstructive apnea:score an apnea as obstructive if it meets apnea criteria and is associated with continued or increased inspiratory effort throughout the entire period of absent airflow.

1.2 Central apnea:score an apnea as central if it meets apnea criteria and is associated with absent inspiratory effort throughout the entire period of absent airflow

1.3 Mixed apnea: score an apnea as mixed if it meets apnea criteria and is associated with absent inspiratory effort in the initial portion of the event, followed by resumption of inspiratory effort in the second portion of the event

2. Hypopnea: score a respiratory event as a hypopnea if ALL of the following criteria are met:

a. The peak signal excursions drop by ≥30% of pre-event baseline using nasal pressure (diagnostic study), PAP device flow (titration study), or an alternative hypopnea sensor (diagnostic study).

b. The duration of the ≥30% drop in signal excursion is ≥10 seconds

c. There is a ≥3% oxygen desaturation from pre-event baseline or the event is associated with an arousal

3. Respiratory Effort-Related Arousal (RERA): Score a respiratory event as RERA if there is a sequence of breaths lasting ≥10 seconds characterized by increasing respiratory effort or by flattening of the inspiratory portion of the nasal pressure (diagnostic study) or PAP device flow (titration study) waveform leading to arousal from sleep when the sequence of breaths does not meet criteria for an apnea or hypopnea Hypoventilationcan be diagnosed using arterial PCO2, transcutaneous CO2 (TcCo2) or end-tidal CO2 (ETCO2). Generally TcCo2 or ETCO2 are most feasible. If electing to score hypoventilation, score a respiratory event as hypoventilation during sleep if EITHER of the below occur:[2]

a. There is an increase in the arterial PCO2 (or surrogate) to a value >55 mmHg for ≥10 minutes.

b. There is ≥10 mmHg increase in arterial PCO2 (or surrogate) during sleep (in comparison to an awake supine value) to a value exceeding 50 mmHg for ≥10 minutes

References

1. Practice parameters for the use of portable recording in the assessment of obstructive sleep apnea. Standards of Practice Committee of the American Sleep Disorders Association. Sleep 1994;17:372-7.
2. Berry RB BR, Gamaldo CE, Harding SM, Lloyd RM, Marcus CL and Vaughn BV for the American Academy of Sleep Medicine. The AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology and Technical Specifications, Version 2.0.2. www.aasmnet.org, Darien, Illinois: American Academy of Sleep Medicine, 2013.

Day 2: Friday, 4th December 2015

Session: **Presidential Lecture**
Time: **0815 – 0900**
Venue: **Conference Hall 2**

COPD is a Systemic Disease- Importance of Comorbid Diseases

– Michiaki Mishima (Japan)

COPD has recently been recognised as “a systemic disease”. It is clinically important to realise that COPD and its comorbid diseases often conspire to adversely influence patients condition as well as their long-term prognosis. I will introduce topics including our studies about the relationship between COPD and comorbid diseases. I will also refer to the induced pluripotent stem (iPS) cell study which may be useful for understanding of COPD as a systemic disease.

Terada et al. reported that the relative risk for GERD symptoms in COPD patients compared with healthy subjects was 2.15, and the risk for the exacerbation in patients with GERD symptoms compared to those without was 1.93 (1). *Tabata et al.* reported that airflow limitation in smokers was associated with arterial stiffness in the results of large scaled cohort study (2). *Ogawa et al.* showed that body mass index (BMI) correlated with LAA% (percent ratio of low attenuation area to whole lung area in X-ray CT: index of emphysema) (3). *Ohara et al.* reported that the LAA% had a significant negative correlation with bone mineral density (BMD) assessed by X-ray CT (4). *Haruna et al.* reported that emphysematous change had strong association with mortality and may predict respiratory mortality in COPD patients (5). *Tanabe et al.* demonstrated that exacerbations were involved in emphysema progression in patients with COPD (6).

Induced pluripotent stem (iPS) cells, developed by Shinya Yamanaka in Kyoto University who won the Novel Prize in 2012, have a potential to differentiate into any cells in the body. Since alveolar type I cells differentiate from alveolar type II (AT2) cells, the key cells for alveolar tissue has been appointed to be AT2 cells. *Gotoh S et al.* developed a novel method to isolate alveolar type AT2 cells from induced iPS cells by way of endodermal lineage (7). Recently, we successfully obtained airway epithelial cells with cilia from iPS cells. These methods may be useful to understand COPD as a systemic disease, and enhance new drug development.

References:

(1) *Thorax* 63; 951-5: 2008, (2) *Atherosclerosis* 232; 59-64: 2014, (3) *Thorax* 64; 20-5: 2009,

(4) *Chest* 134; 1244-9: 2008, (5) *Chest* 138; 635-40: 2010, (6) *Am J Respir Crit Care Med* 183; 1653-9: 2011, (7) *Stem Cell Reports* 3; 394-403: 2014.

Day 2: Friday, 4th December 2015

Session: **Session 1A-2: Asthma**
Time: **0930 – 1000**
Venue: **Conference Hall 2**

Anti-neutrophil treatment in asthma

– Peter Barnes (UK)

Some patients with asthma have an increased number of neutrophils in induced sputum and appear to be less responsive to corticosteroids than the majority of asthmatics who have an eosinophilic (T2) pattern of inflammation. These neutrophilic patients (non-T2) may have more severe disease or may be cigarette smokers and may be difficult to control with conventional therapy. There is a need to develop more effective therapies for these patients by targeting the neutrophilic inflammation, but there is still little known about the mechanisms of neutrophilia in severe asthma. High doses of corticosteroids prolong the survival of neutrophils so may contribute towards the neutrophilia in severe asthma. Neutrophil chemotactic factors, such as leukotrieneB$_4$ (from macrophages) and the chemokines CXCL1 and CXCL8 (from epithelial cells and macrophages) may be increased in severe and smoking asthma. LTB$_4$ receptor antagonists have proved to be ineffective in asthma, although not tested in the right population of patients. CXCL1 and CXCL8 activate the chemokine receptor CXCR2 on neutrophils and small molecule antagonists are now developed. However so far these antagonists have not been effective in treating patients with neutrophilic asthma, probably as several neutrophil chemotactic factors may be involved. Neutrophilic inflammation in the lungs may be driven by Th17 and ILC3 cells, which secrete IL-17, which in turn releases neutrophil chemoattractants from airway epithelial cells. A monoclonal antibody that blocks the IL-17 receptor (brodalumab) is ineffective in treating patients with severe asthma, although patients with increased neutrophilic inflammation were not selected. TNF-α is also associated with neutrophilic inflammation and is increased in severe asthma, although blocking TNF-α with a monoclonal antibody was not effective in patients with severe asthma and increased pneumonias. Similarly blocking IL-1β, also associated with neutrophilic inflammation has so far been ineffective. Existing therapies may target neutrophilic inflammation and the long-acting β_2-agonist formoterol reduces sputum neutrophilic in asthma patients by reducing CXCL8 release. The most promising treatments for neutrophilic asthma are macrolide antibiotics, which appear to work by inhibiting the proinflammatory transcription factor NF-κB, or possibly by an antibacterial effect. Antibiotic resistance is a problems but non-antibiotic macrolides are in development. Statins may also be effective in smoking asthmatic and have been shown to reduce the imbalance between Th17 and regulatory T cells. New treatments for neutrophilic asthma include p38 MAP kinase inhibitors and JAK inhibitors, but these may need to be delivered by inhalation to avoid side effects. More treatments for non-T2 asthma are needed.

Day 2: Friday, 4th December 2015

Session: **Session 1A-3: Asthma**
Time: **1000 – 1030**
Venue: **Conference Hall 2**

Bronchial thermoplasty in asthma

– Gerard Cox (Canada)

Asthma is a serious public health problem. It is one of the top five chronic diseases globally, which includes heart disease, stroke, cancer, and diabetes. Some patients with severe asthma, who are on the highest doses of standard of care medications (inhaled corticosteroids and long acting bronchodilators) taken daily, may still experience frequent and life-threatening asthma attacks. The increased burden of severe asthma can lead to a substandard quality of life for these patients, and few treatment options exist to adequately control their disease.

In the United States, asthma affects almost 25 million Americans, five to ten percent of whom are estimated to have severe asthma. U.S. government data reported, in one year:

- 12.8 million people experiencing asthma attacks
- 1.75 million emergency room visits
- 456,000 hospitalizations
- 3,447 asthma-related deaths

Source: Centers for Disease Control and Prevention National Center for Health Statistics,National Health Statistics Report: Asthma Prevalence, Health Care Use, and Mortality: United States, 2005–2009: http://www.cdc.gov/nchs/data/nhsr/nhsr032.pdf

Severe asthma can have a dramatic impact on a person's quality of life:

- In 2008, affected adults missed an average of five days of work due to asthma.1
- On average, a person will spend four days in the hospital each time they are admitted for asthma treatment.2
- Patients who are severely affected by asthma are four times as likely to experience negative outcomes, such as emergency room visits and use of oral steroids.3
- Patients with severe asthma experience frequent symptoms such as coughing, wheezing,
- shortness of breath, chest tightness, mucus production, and exacerbations (asthma attacks)
- caused by a narrowing of the lungs' airways.4

Clinical Results – *At Year One*

The AIR2 Trial evaluated the safety and effectiveness of the Alair™ Bronchial Thermoplasty System in patients with severe asthma. At one year, patients with severe asthma treated with BT, showed significant improvement compared to sham controlled patients.

- **32 percent fewer** severe asthma attacks
- **84 percent fewer** emergency room visits for respiratory symptoms
- **66 percent fewer** days lost from work, school, and other activities due to asthma symptoms

In addition,

- **79 percent** of patients treated with Bronchial Thermoplasty (BT), delivered by the Alair™ System, saw a **significant improvement** in their asthma-related quality of life.

Source: Castro M, et al, for the AIR2 Trial Study Group. Effectiveness and safety of Bronchial Thermoplasty in the treatment of severe asthma: a multicenter, randomized, double-blind, sham-controlled clinical trial. Am J RespirCrit Care Med. 2010;181:116-124

Long-Lasting Clinical Results – *Out to Five Years*

In September 2013, the article, "Benefits of Bronchial Thermoplasty Persist Out to 5 Years in Patients with Severe Asthma," by Michael Wechsler and colleagues appeared in the *Journal ofAllergy and Clinical Immunology. In Press*

The AIR2 Trial 5-Year Extension Study was conducted to evaluate the sustained effectivenessof BT beyond 1 year, and the long-term safety of BT out to five years in BT-treated patients fromthe AIR2 Trial.

Safety & Effectiveness Maintained Out to Five Years

- The reduction in severe asthma attacks was maintained out to five years.
- The reduction in emergency room visits for respiratory symptoms was maintained out to five years.
- ·No increase in hospitalizations, asthma symptoms, or respiratory adverse events over the course of 5 years

Source: Wechsler M, et al. for the AIR2 Study Group. Benefits of Bronchial Thermoplasty Persist Out to 5 Years in Patients with Severe Asthma.Journal of Allergy and Clinical Immunology. In Press

Scientific Evidence

Experience with Bronchial Thermoplasty has been described in more than 15 peer reviewed publications including the *New England Journal of Medicine*, *Journal of Allergy and ClinicalImmunology, CHEST*, *Annals of Allergy, Asthma and Immunology* and the *American Journal ofRespiratory and Critical Care Medicine*.

Day 2: Friday, 4th December 2015

Session: **Session 1B-2: Chest Imaging**
Time: **0930 – 1000**
Venue: **Conference Hall 3**

CT for COPD

– Nguyen Van Tho (Vietnam)

Computed tomography (CT) has been used to evaluate pulmonary structural changes in chronic obstructive pulmonary disease (COPD). These changes include emphysema, air-trapping, bronchial wall thickening, bronchiectasis, etc. Traditionally, physicians evaluate these changes visually by identifying the abnormality and estimating the severity. However, the traditional method is subject to intra-observer and inter-observer variation. Quantitative CT—a computer-based method for objective evaluation of CT datasets—has been developed to complement the traditional method. By using CT image processing software, these structural changes can be measured objectively and robustly. Some measurements derived from the quantitative CT include the extent of emphysema (a surrogate for parenchymal destruction), the severity of air-trapping (for small-airway obstruction), and large-airway dimensions (for airway remodeling); the CT measurements are determined for whole-lung, single-lung, and individual lung lobes; and they are related to lung function. As a result, quantitative CT has been used to complement routine tests to shed light on the lung structure-function relation and to evaluate the heterogeneity of COPD. Because the CT measurements are significantly associated with clinically relevant outcomes (clinical symptoms, exacerbations, hospitalizations, disease progression, and mortality), they have been used as biomarkers in ongoing large cohorts to investigate the pathogenesis, therapeutics, and genetics of COPD. However, to apply quantitativeCT routinely in clinical settings, we may consider using low-dose CT, standardizing CT scanning protocols, and establishing reference values for CT measurements. Some important findings, potential applications, and major challenges of quantitativeCT in COPD will be highlighted in this presentation.

Day 2: Friday, 4th December 2015

Session: **Session 1B-3: Chest Imaging**
Time: **1000 – 1030**
Venue: **Conference Hall 3**

CT for rare lung diseases

– Masashi Takahashi (Japan)

1. Why should we know the CT features of rare lung disease?
In daily clinical practice, it is extremely important to know the clinical and imaging features of common diseases. However the knowledge of rare lung disease enable us to enrich the differential diagnosis to reduce the confusion when facing the very unusual imaging findings. Some of the rare lung disease show the pathognomonic CT appearance and those knowledge conduce us to the final diagnosis more easily.

2. The list of rare lung disease
In this lecture, CT findings as well as some clinical features of the following diseases will be summarized.

A. Genetic disorders
-LAM(lymphangioleiomyomatosis)、MMPH(Multifocal micronodularpneumocyte hyperplasia)
-Birt-Hogg-Dube syndrome
-Alveolar microlithiasis
-Ehlers-Danlos syndrome

B.Inflammatorydisorders
-Relapsing polychondritis
-GPA (Granulomatous polyangiitis)
-EGPA (Eosinophilic granulomatois with polyangiitis)
-MPO-ANCA related ILD

C. Neoplastic or neoplasm-like disorders
-PTTM (Pulmonary tumor thrombotic microangiopathy)
-MALT Lymphoma (Mucosa associated lymphoid tissue lymphoma
-IVL (Intravascular lymphoma)
-MCD (MulticentricCastleman's disease)
-PAL (Pyothorax associated lymphoma)
-MTX related lymphoproliferative disorder)
-LCH (Langerhans cell histiocytosis)
-Erdheim-Chester disease
-IgG4 related pleuro-pulmonary disease

D. Other
-DIP (Desquamative interstitial pneumonia)
-Amyloidosis
-PAP (Pulmonary alveolar proteinosis)
-Chronic expanding hematoma

Day 2: Friday, 4th December 2015

Session: **Session 1C-1: Cell and Molecular Biology**
Time: **0900 – 0930**
Venue: **Conference Hall 1**

Recent progress of cellular and molecular biology in respiratory diseases

– Takahide Nagase (Japan)

In respiratory diseases, there are various inflammatory disorders to which very few pharmaceutical agents are currently effective. For example, ARDS is an acute lung injury and the mortality rate for ARDS ranges from 40-70% despite of intensive care using currently available drugs. Idiopathic pulmonary fibrosis (IPF) is a progressive and fatal disorder of the lung parenchyma, while few useful drugs are currently available to treat IPF. However, their mechanisms still remain to be elucidated. COPD is one of the leading causes of death worldwide, while molecular mechanism underlying COPD is still little known. COPD with emphysema has been considered to be an accelerated involutional disease in aged smokers. However, based on the fact that only a limited proportion

Day 2: Friday, 4th December 2015

Session: **Session 1C-2: Cell and Molecular Biology**
Time: **0930 – 1000**
Venue: **Conference Hall 3**

Generation of airway epithelial cells from iPS cells

– Isao Ito (Japan)

Lungepithelial cells are damaged in a large numbers of lung diseases such as chronic obstructive lung disease, idiopathic pulmonary fibrosis and acute lung injury. The epithelial damage leads to respiratory failure and death. Generation of the epithelial cells from human induced pluripotent stem cells (iPSCs) *in vitro* would be applied in modeling of such lung diseases, drug screening and regeneration of the lung. The alveoli consists of type I alveolar epithelial cells (AECs) which have a role in gas exchange, and type IIAECs which produce surfactant proteins. To differentiate the lung epithelial cells from human iPSCs, complicated stepwise induction through definitive endoderm and ventral anterior foregut endoderm (VAFE) cells have been tried worldwide. In the induction, we have identified carboxypeptidase M (CPM) as a surface markerfor NKX2.1-positive VAF cells and successfully isolated the cells. Further, applying three-dimensional culture method with fetal lung fibroblasts, CPM+ cells isolated from VAFE cells formed spheroid structures.A portion of the spheroid cells were proven to differentiate into surfactant protein C-producing cells that are consideredas type II AECs. In addition, aquaporin 5, a marker of type I AECs, was identified in some of the cells. *In vivo*, CPM+ cells are identified in fetal lung epithelium in mouse and human. Thus, CPM+ cells are considered to be a progenitor cells of the lung. Our findings could contribute further understanding of development of thelungand clinical application of regenerative medicine in the future. We would like to discuss further application of lung epithelial cells differentiated from human iPSCs in respiratory investigations.

Day 2: Friday, 4th December 2015

Session: **Session 1C-3: Cell and Molecular Biology**
Time: **1000 – 1930**
Venue: **Conference Hall 1**

Role of microbiomes in lung diseases

– Philips Hansbro (Australia)

Recent technical advances have enabled the assessment of entire microbiomes in tissues. This had led to the elucidation of their roles in health and disease. Until recently the lower respiratory tract was thought to be sterile but microbiome studies have shown this not to be the case and that there is a core lung microbiome. Alterations in the microbiome indicate and may be causal in disease. In dysbiosis commensals are displaced by pathgens that drive inflammation and inflammatory diseases including the in the respiratory tract. It is now established thatthere is infectious and inflammatory cross talk between the lung and gut and so changes in gut microbiomes may also be involved in respiratory disease potentially through the induction of systemic inflammation. The current state of the field in asthma and COPD will be assessed and new data from our lab on the role of changes in the gut microbiome in COPD will be presented.

Day 2: Friday, 4th December 2015

Session: **Session 2A-1: COPD**
Time: **1100 – 1130**
Venue: **Conference Hall 1**

Non-smoking COPD versus neutrophilic asthma: similarities and differences

– Jorgen Vestbo (UK)

Asthma and COPD are both heterogeneous diseases characterised by either permanent or variable airflow limitation. According to GOLD, a clinical diagnosis of COPD requires a recognised exposure. When this is not cigarette smoking, biomass exposure seems the most likely but other causes such as outdoor air pollution or occupational exposures to dusts and gases are also of relevance. In patients where the exposure is not obvious (non-smoking COPD) and important differential diagnosis is often chronic asthma, in particular neutrophilic asthma. Little guidance exists as to the differentiation and often these patients end of having labels of both asthma and COPD, the asthma COPD overlap syndrome (ACOS).

However, ACOS should be regarded as a last descriptive option as a thorough history of childhood respiratory illness, exposures during childhood, adolescence and adulthood as well as a proper history combined with serial spirometries (or serial measurements of PEF) will often provide a single diagnosis. Few good studies have described the underlying pathobiology of non-smoking COPD compared to neutrophilic asthma. It was previously believed that independent of type and origin of asthma, one would always find a thickened basement membrane which would not be found in COPD, but subsequent studies have not been able to prove this.

Most importantly, treatment of these disease entities should not be based on disease labels – and evidence-based treatment is rarely available anyway. Treatable traits should be identified and be the basis of intervention.

Day 2: Friday, 4th December 2015

Session: **Session 2A-2: COPD**
Time: **1130 – 1200**
Venue: **Conference Hall 1**

Asthma-COPD overlap syndrome: biomarker, phenotype or endotype-driven treatment

– Diahn-Warng Perng (Taiwan)

According to GINA and GOLD joint consensus statement, “Asthma- COPD overlap syndrome(ACOS) is characterized by persistent airflow limitation with several features usually associated with asthma and several features usually associated with COPD. ACOS is therefore identified in clinical practice by the features that is shares with both asthma and COPD.” The biomarker, phenotype or endotype that can specifically represent the character of ACOS and therefore guide the treatment remains unclear and require further investigation. Regarding the commencement of initial therapy, ICS plays a pivotal rolein the management of ACOS. Consensus statement also recommends that “if there are features of asthma, do not treat with a LABA without ICS.” Current evidence shows that blood or sputum eosinophils, bronchial reversibility or IL-5 may be associated with treatment for COPD with eosinophilic inflammation or ACOS. We still need more studies to understand the clinical and physiological characteristics and underlying mechanisms which may help better recognition and guide appropriate treatment for ACOS.

Day 2: Friday, 4th December 2015

Session: **Session 2A-3: COPD**
Time: **1200 – 1230**
Venue: **Conference Hall 1**

Steroid insensitivity and target therapy for COPD: What do we need and where are we now?

– Peter Barnes (UK)

Most patients with COPD have a very poor response to corticosteroids, even with high doses and high doses of inhaled corticosteroids do not reduce COPD disease progression or mortality. The inflammation in COPD lungs is corticosteroid-resistant. The nuclear enzyme histone deacetylase-2 (HDAC2) is required for corticosteroids, via glucocorticoid receptor activation, to switch off activated inflammatory genes. In COPD patients there is an increase in oxidative stress in the airways, which accounts for the reduction in HDAC2 as a result of two mechanisms. Oxidative and nitrative stress form peroxynitrite, which nitrates tyrosine residues on HDAC2 leading to its ubiquitination and degradation.HDAC2 is also reduced by activation of phosphoinositide-3-kinase-δ (PI3Kδ) which leads to phosphorylation and inactivation of HDAC2. Low concentrations of theophylline and nortriptyline and macrolides restore the low levels of HDAC2 levels in COPD macrophages to normal and reverse steroid resistance*in vitro* and *in vivo* by selectively blocking PI3Kδ. These studies may lead to new therapeutic approaches, with the use of low dose theophylline, new macolidesand in the future PI3Kδ-selective inhibitors to reverse corticosteroid resistance. We have recently described a new mechanism of steroid resistance which involves the activation of PI3K and mammalian target of rapamycin (mTOR), leading to activation of the MAP kinase c-Jun, with phosphorylation of the glucocorticoid receptor, resulting in impaired nuclear translocation. Rapamycin reverses this mechanism of steroid resistance. In the future treatments that reverse corticosteroid resistance may improve the management COPD and more effectively suppress the underlying inflammation.

Day 2: Friday, 4th December 2015

Session: **Session 2B-3: Clinical Respiratory Medicine**
Time: **1200 – 1230**
Venue: **Conference Hall 3**

Case 3 - Clinical Respiratory Medicine

– Sanjay H. Chotirmall (Singapore)

A forty-nine year old female presented to the hospital emergency room complaining of progressive dyspnoea for months. This had acutely worsened over the preceding weeks and progressed rapidly prompting the current presentation. Her background medical history was only significant for Lofgren's syndrome and neurosarcoidosis affecting the facial nerve, both occurring approximately two decades prior to the current presentation. She had not been exposed to corticosteroids for over a decade. Despite aggressive treatment, she developed progressively worsening respiratory symptoms and a myopathy necessitating intensive care unit admission and ventilator support. She was eventually diagnosed with a myositisspectrum interstitial lung disease ('anti-synthetase syndrome') and aggressively treated with immunosuppression. She responded well and eventually weaned successfully. This talk will present details of the case, its presentation, management and therapeutics employed. It will then discuss the group of diseases termed 'idiopathic inflammatory myopathies' and how they affect the lung including their diagnosis, presentation and treatment with focus on the 'anti-synthetase syndrome'.

Day 2: Friday, 4th December 2015

Session: **Session 2C-1: Environmental & Occupational Health & Epidemiology**
Time: **1100 – 1130**
Venue: **Conference Hall 3**

E-cigarettes – The good, the bad and the ugly

– Hayden McRobbie (New Zealand)

Electronic cigarettes (EC) are electronic devises the heat a solution of propylene glycol and/or glycerol creating an aerosol which users inhale. The solutions typically contain flavouring and nicotine. EC use has increased substantially in countries where they are freely available, mostly in smokers who want to stop or reduce their cigarette consumption.

EC have polarised the tobacco control sector with some believing that these devices will contribute towards a reduction in health risks caused by smoking, by assisting people either to quit or reduce smoking. Others however have concerns that EC may impact adversely on individual and population health, perpetuating nicotine addiction, re-normalising smoking behaviour, promoting dual use, and fearing that they might be a gateway to smoking tobacco.

This presentation will present data on use of EC, a summary of what is known about nicotine delivery and toxicant exposure, the effectiveness of EC in helping people stop and reduce smoking, and outline the concerns and how these might be managed.

Conflict of interest statement: In the last 5 years Hayden McRobbie has received research funds and consultancy fees from manufacturers of smoking cessation medicines. He has no links with any tobacco or e-cigarette manufacturers.

Day 2: Friday, 4th December 2015

Session: **Session 2C-2: Environmental & Occupational Health & Epidemiology**
Time: **1130 – 1200**
Venue: **Conference Hall 3**

Asbestos ban and asbestos-related diseases in Asia

– Soon-Hee Jung (Korea)

In comparison with the reduction or eliminationof asbestos use in developed countries due to health risk awareness, Asia has increased its global share of asbestos use. The proportion of global asbestos use attributed to Asia has increased from 14% (1920-1970) to 33% (1971-2000) and to 64% (2001-2007), making it the largest consumer of asbestos in the world. Also, three out of the top five asbestos-producingcountries are in Asia (Russia, China andKazakhstan). The country that uses the most asbestos is China (29%), followed by India (17%), Kazakhstan (7%), Indonesia(5%), Uzbekistan (5%), Thailand (4%), Vietnam (4%),Sri Lanka (2%) and Iran (1%).Although some countries such asJapan, Korea and Singapore have banned the use of asbestos, there are numerous countries in Asia thatcontinue to mine, import and use asbestos, particularlyChina. Asbestos use in Asia has increased markedly since 1970. Asbestos cement is very easily used for residential homes in rural areas in many Asian countries, whileasbestos-containing materials (ACM, any material containing more than 1% asbestos) are found in most buildings, potentially making the construction business the main source of asbestos-related diseases (ARDs) in Asia. In fact, 12,882 ARD deaths have beenrecorded cumulatively, which is equivalent to12.5% of the cumulative number of ARD deaths inthe world during the same period (national population data from 1920–2008obtained from the WHO).The level of development of different Asian countries varies. Numerous factors ranging frompolitical andeconomic to the lack of understanding of asbestos andARD management in Asia may explain the increase in asbestos consumption. To prevent and minimize the risks of ARDs and occupational/environmental exposure to asbestos in Asia, therefore, systematic approaches to monitor ACMs and proper management of ACMs are highly required and should be effectively implemented through careful surveillanceof ARDs, training in the recognition and diagnosis of ARDs, and collaboration among government and non-government groups of Asian countries.

Day 2: Friday, 4th December 2015

Session: **Session 2C-3: Environmental & Occupational Health & Epidemiology**
Time: **1200 – 1230**
Venue: **Conference Hall 3**

Effect of inorganic dust on the lung

– Takashi Nakano (Japan)

The effects of inhaled hazardous particles in polluted air are of great public interest with regard to health. Most dust particles that are inhaled are filtered out through a series of airway defense mechanisms; however, some fibrous particles such as asbestos fibers can evade elimination and smaller particles succeed in passing through the defense mechanism and reach small airways, where Club cells containing cytochrome P-450 enzymes and alveolar macrophages play a role in the defense mechanism. Club cells engulf airborne toxins and break them down using their cytochrome P-450. Lung diseases due to the inhalation of a wide variety of organic dusts are characterized by immunologic reactions to specific antigens contained in the dust (extrinsic allergic alveolitis). On the other hand, the inhalation of inorganic dust, crystalline silica, coal mine dust, and asbestos causes classic forms of pneumoconiosis, i.e., pulmonary fibrosis related to a toxic effect of the inhaled substances and aggregates of particle-laden macrophages. In contrast to the resolution of organic dust-related pathophysiology after cessation of its exposure, cessation of inorganic dust exposure does not lead to the resolution of the pathophysiology because of the activities of particle-laden macrophages. Diseases caused by the inhalation of inorganic dust are strongly associated with work environments, particularly with occupations that lead to the production of airborne particles. Crystalline silica in the form of quartz and cristobalite dust causes lung cancer in humans (IARC Group-1). Inhalation of crystalline silica during the use of commercial products containing quartz is considered the primary route of exposure for the non-occupationally exposed (i.e. general) population. Volcanoes spew ash and inorganic dust including particulate matter into the air of the widespread residential area surrounding it. Mt. Sakurajima in Japan is one of the most active volcanoes in the world, the ash from which contains up to 7 wt% of cristobalite. Increased mortality associated with lung cancer and COPDs have been shown in the vicinity of Mt. Sakurajima, which has the largest amount of ashfall. Continued inhalation of inorganic dust can produce a wide range of pulmonary diseases including airway disorders, parenchymal diseases, and cancer. Several factors influence the effects of inhaled inorganic particles. Particle size is usually a critical factor that determines where the particle is deposited in the respiratory tract. In general, fumes of metal oxides are smaller in size than dust particles. Physicochemical features are also important because some substances, when in the particle form, can destroy the cilia that are used for the removal of particles in lungs.

The effects of inorganic dust on lungs will be discussed in the meeting.

Day 2: Friday, 4th December 2015

Session: **Session 3A-2: Lung Cancer**
Time: **1430 – 1500**
Venue: **Conference Hall 2**

Molecular testing in advanced NSCLC

– David Chi Leung Lam (Hong Kong)

Lung cancer is the leading cause of global cancer mortality, partly attributable to late presentation and indeed late detection after it has spread distantly without a reliable biomarker for early detection. The identification of biomarkers will support the practice of personalized as well as precision medicine to improve clinical outcomes and this will be closely tied to the understanding of lung cancer biology and molecular progression of lung neoplasms. In the past decade, the management of advanced lung cancer has undergone a revolutionary change, from the very limited chemotherapy options to the current biomarker detection to guide targeted treatment decision. The ultimate goal of personalized therapy would be to improve clinical therapeutic efficacy and treatment outcome.

Lung adenocarcinoma is the most common type, accounting for 70% of lung cancer. Different lung tumor could have different types of oncogenic driver mutations, modulating different oncogenic signaling pathways. EGFR gene mutation is present in up to 50% of lung adenocarcinoma from the Asian population. Epidermal growth factor receptor tyrosine kinase inhibitor (EGFR-TKI) should be used as primary treatment of lung cancer bearing favorable EGFR mutations, namely 15-base pair deletion at exon 19, missense mutations of L858R and L861Q at exon 21. The presence of different oncogenic driver gene mutations in individual lung tumor is mutually exclusive to each other.

First- and Second-generation EGFR-tyrosine kinase inhibitor (TKI) has greatly improved the treatment of EGFR-mutant lung cancer. However, the majority of patients who respond initially will eventually experience treatment failure with disease progress. For these patients, alternative treatment strategies are urgently needed. In patients who develop acquired resistance to TKI, 50% of them showed a second T790M mutation at exon 20, while 16% showed MET amplification. Third-generation EGFR-TKI with specific target to T790M mutation has been developed and recently approved by US FDA for treatment of resistant tumors.
ALK gene rearrangements are not common in lung cancer, less than 7%, but once detected, upfront ALK-inhibitor is available with good therapeutic efficacy. There are current interests in alternative targets like c-MET as well as ROS1 as new markers or therapeutic targets in lung cancer. With recent US FDA approval of anti-PD1 antibodies for the treatment of advanced stage non-small cell lung cancer, there are also keen interests to explore the relevance of PD-1 and PD-L1 as a biomarker of treatment response.

Molecular testing of lung cancer biomarkers will not be limited to testing in tumor tissues. Biomarker detection based on circulating tumor DNA or cell free tumor DNA using different genomic platforms or molecular biology techniques is feasible and is under validation before possible clinical diagnostic application.

Day 2: Friday, 4th December 2015

Session: **Session 3A-3: Lung Cancer**
Time: **1500 – 1530**
Venue: **Conference Hall 2**

Management of resistance to EGFR-TKIs and ALK-inhibitors

– Chong-Kin Liam (Malaysia)

Molecular targeted treatment with tyrosine kinase inhibitors (TKIs) of oncogenic drivers particularly epidermal growth factor receptor (EGFR) mutations (with gefitinib, erlotinib or afatinib) and anaplastic lymphoma kinase (ALK) rearrangements (with crizotinib) has resulted in dramatic improvements in the survival of defined subsets of patients with advanced non-small cell lung cancer. However, despite dramatic initial responses acquired resistance invariably develops with clinical disease progression after a median of 9 to 12 months.

T790M mutation in exon 20 of the EGFR gene is the most common acquired resistance mechanism which develops in 50% to 60% of patients treated with first- generation EGFR TKIs. Other mechanisms of acquired resistance include activation of alternate signaling pathways by both adaptive mutations that develop outside the EGFR kinase domain and mutation-independent mechanisms including *MET* oncogene amplification (5-10%), *HER-2* amplification (10%), *PI3K* mutations (5%) and transformation to small cell lung cancer (SCLC) (5-10%). Treatment approaches for patients experiencing progressive disease on frontline EGFR TKI therapy should be individualised according to the characteristics of disease progression and mechanism of acquired resistance. If progression is indolent and asymptomatic, continuing the EGFR TKI with close monitoring is an option. Palliative radiotherapy followed by EGFR TKI continuation for central nervous system (CNS)-only progression or other local ablative treatment may be considered if there is localised symptomatic progression. A repeat biopsy at the time of disease progression may provide insight into the mechanism of resistance and therefore the selection of optimal treatment strategies. Third-generation EGFR TKIs such as osimertinib (AZD929) and rociletinib (CO-1686) being more *T790M* selective, clinically more potent and less toxic than the first- and second-generation EGFR TKIs have shown encouraging treatment responses and disease control in patients with progressive disease on frontline first- and second-generation EGFR TKIs. Molecular targeted agents already exist or are being developed against the other pathways implicated in acquired resistance, such as *MET*, *HER2*, and *PIK3CA*. Cytotoxic chemotherapy is the treatment option if novel molecular targeted treatment is not available, the mechanism of acquired resistance is unknown or when there is SCLC transformation.

The same principles apply in the treatment approach for patients experiencing progressive disease on frontline ALK TKI therapy. In carefully selected patients with an isolated site of recurrence that can be treated with local therapy or those with mild and asymptomatic oligometastatic progression, crizotinib may be continued with close monitoring after initial evidence of disease progression. Second-generation ALK inhibitors such as ceritinib and alectinib, being more potent and have the potential to overcome acquired resistance to crizotinib and are active against brain metastases are the treatments of choice when symptomatic progressive disease is not amenable to local treatment. In cases where a second-generation ALK inhibitor is not available or fails to control the disease, chemotherapy is the next treatment of choice.

Day 2: Friday, 4th December 2015

Session: **Session 3B-1: Respiratory Infection (non-TB)**
Time: **1400 – 1430**
Venue: **Conference Hall 3**

Pulmonary aspergillosis

– *Sanjay H. Chotirmall* (Singapore)

Aspergillus moulds are ubiquitous and spores are inhaled in large numbers daily. Removed by intact anatomical barriers and an effective immune response, disease occurrence is dictated by the state of the host immune system and the virulence of the infecting fungal strain. Clinical consequences range from acute invasive disease in the immunocompromised to chronic manifestations in those immunocompetent. An excessive immune response occurs in allergic manifestations such as ABPA following Aspergillus exposure (1). This talk introduces the spectrum of disease observed in relation to Aspergillus illustrated by clinical cases and radiology. Appropriate diagnostic and treatment strategies will be discussed including recently published work by our group assessing CD203c use in determining the sensitized and ABPA clinical states. Interactions between Aspergillus species and the host immune system are bi-directional. The fungus elicits an immune response resulting in clearance while concurrently releasing immunoevasive virulence factors. Aspergillus virulence is multifactorial and under polygenetic control. Strategies used are multi-faceted including fungal structure, capacity for growth, adaptation to stressful conditions, the ability to damage host cells and evade immune-recognition. Prior research from our group will be presented including the role of gliotoxin, a potent immune-evasive mycotoxin in Aspergillusassociated disease. Immunosuppressive roles of gliotoxin include the inhibition of phagocytosis, T-cell proliferation, mast cell activation and cytotoxicity. Additionally, it inhibits superoxide production and reduces epithelial ciliary movement leading to dysfunction. In the context of Aspergillus colonization in cystic fibrosis, our group has performed a significant body of work and are now extending this to non-CF settings (1-4). We have shown that the Vitamin D receptor (VDR), a key component of an immunemodulating pathway is down-regulated by gliotoxin. Treatment with itraconazole decreases bronchoalveolar lavage (BAL) gliotoxin concentrations, restoring VDR expression, diminishing systemic Th2 cytokines IL-5 and IL-13 with concomitant improvement in clinical and radiological patient outcomes (5). Understanding Aspergillus-associated disease is critical to early diagnosis and subsequent initiation of therapy in instances of pulmonary aspergillosis where to date the morbidity and mortality burden remains high.

Day 2: Friday, 4th December 2015

Session: **Session 3C-2: Respiratory Neurobiology and Sleep**
Time: **1430 – 1500**
Venue: **Conference Hall 1**

Obesity hypoventilation syndromes in Asians and Western countries

– Kazuo Chin (Japan)

In accordance with the continuing increases in rates of obesity, the incidence of obesity hypoventilation syndrome (OHS) has also increased. OHS is defined as the combination of obesity (body mass index (BMI) ≥ 30 kg/m^2) and chronic daytime hypercapnia (arterial carbon dioxide pressure ($PaCO_2$) ≥ 45 mmHg).[1,2] In addition, the definition of OHS in previous reports included hypoxemia (arterial oxygen pressure (PaO_2) < 70 mmHg).[3,4] It was reported that the prevalence of OHS in patients with obese obstructive sleep apnoea (OSA) was 10-20%[4-6] and that 0.3-0.4% of the general population might have OHS.[7,8] OHS is an entity distinct from OSA or simple obesity and is associated with greater morbidity and mortality than either.[9] Because of cephalometric differences, OHS may occur at a lower BMI in Asians than in Western individuals, as does OSA.[10-12] Investigated were 981 participants consecutively hospitalized for suspected OSA. At least 90% of them were from urban areas, including 162 with obese OSA (BMI ≥ 30 kg/m^2 and AHI ≥ 5/h).The prevalence of OHS (BMI 36.7 ± 4.9 kg/m^2) in OSA and that in obese OSA were 2.3% and 12.3%, respectively. With 12.3 ± 4.6 months of CPAP treatment, more than 60% of OHS patients no longer had hypercapnia. The prevalence of OHS in OSA in Japan was 2.3%. The mean BMI of patients with OHS in Japan was lower than that in Western countries (36.7 kg/m^2 vs. 44.0 kg/m^2).We will discuss similar and/or different points in patients with OHS between Asians and Western countries.

Day 2: Friday, 4th December 2015

Session: **Session 4A-2: Lung Cancer**
Time: **1630 – 1700**
Venue: **Conference Hall 1**

Small cell lung cancer: the evolving role of radiotherapy

– Yoichi Nakanishi (Japan)

For over 20 years radiotherapy have been recognized tobe one of important treatment modalities to small-cell lung cancer (SCLC), since SCLC is extremely sensitive to radiotherapy. Recently, new evidences regarding with radiotherapy are accumulating followed byimprovement of radiation devices and progress of radiation biology.

SCLC is clinically classified into limited-disease (LD) and extended-disease (ED). Generally, LD is a disease which is confined to the hemithorax of origin, the mediastinum, or the supraclavicular lymph nodes. Except very early disease stage (T1-2N0 M0), combination with chemotherapy and radiotherapy is a standard treatment modality for patients with LD-SCLC, and the goal of treatmentwith chemotherapy plus thoracic radiotherapy is to achieve a cure. Based on randomized trials,early, concurrent radiotherapy is recommended along withchemotherapy for patients with LD-SCLC. Intracranial metastases occur in more than half of patients with SCLC.A meta-analysisby Auperinet al. has shown that prophylactic cranial irradiation(PCI) significantly elongates overall survivalin patients achieving complete response.Since late neurologic sequelaehave been attributed to PCI, fractions of2.5Gyx10after the completion of chemotherapy are recommended today.

Prognosis of ED-SCLC is poor. A combination of platinum plus etoposide is accepted as a first choice in western countries, while a combination of platinum plus irinotecan is recommended in Japan. There would be genetic difference pharmacologically. Although chemotherapy alone was so far recommended in patients with ED-SCLC, recent randomized trial has shown that thoracic irradiation significantly prolongs overall survival of patients who responded chemotherapy. On the other hand, clinical usefulness of PCI to ED-SCLC patients who responded chemotherapy is under debate.

Radiotherapy is a trenchant blade to SCLC. On the other hand, acute and chronic toxicities can not ignored. The indication of radiotherapy should be judged in consideration of risk-benefit balance.

There is no conflict of interest in this presentation.

Day 2: Friday, 4th December 2015

Session: **Session 4A-3: Lung Cancer**
Time: **1700 – 1730**
Venue: **Conference Hall 1**

Update on Asian lung cancer screening: randomised and non-randomised studies

– David Chi Leung Lam (Hong Kong)

The landmark US National Lung Cancer Screening study (NLST) demonstrated low-dose CT screening can reduce lung cancer mortality by 20% compared with conventional radiography. The mortality benefit observed from annual screening of a high-risk cohort with low-dose CT in the NLST was associated with increased detection of early stage lung cancers and reduction in the number of late-stage lung cancers diagnoses. Similar large scale lung cancer screening trials including the NELSON, DANISH and ITALUNG all are suggesting that lung cancer screening are supported with scientific evidence yet the targeted population should be well defined according to local population characteristics. However, the high false positive rates are regarded as potential burden of medical, physical and psychological aspects to both physicians and participating subjects.

The next scientific question is whether the performance of low-dose CT or relevant screening procedures could be improved with better selection of high-risk individuals and the refinement of definitions of positive and negative screens. Determining appropriate screening intervals based on risk stratification is another practical issue. Other lung-cancer risk-prediction model from the Prostate, Lung, Colorectal, and Ovarian (PLCO) Cancer Screening Trial, the Pan-Canadian Early Detection of Lung Cancer Study (Pan-Can), and other early lung cancer detection trial are on-going.

Lung Cancer screening trials had been performed in Asia but there was in general a lack of endeavor in the region after the low screening detection rate in previous reported series in China, Japan and Korea. This could partly reflect issues on targeted screening population and could partly represent the issue of the different characteristics of Asian population and their susceptibility to develop lung cancer. All these would warrant designated screening trials adapted to the local lung cancer epidemiology in this Asian region.

Day 2: Friday, 4th December 2015

Session: **Session 4B-2: Interstitial Lung Disease**
Time: **1630 – 1700**
Venue: **Conference Hall 3**

The role of CT scan in interstitial lung diseases

– Masashi Takahashi (Japan)

1. The potential difficulty for approaching the diagnosis of ILD

The difficulty to diagnose ILD is associated with the following three facts. First, the symptoms of the patient with ILD are usually very nonspecific such as cough, sputum, chest pain, dyspnea on effort or finger clubbing. Second, although the histological confirmation is still extremely important to establish the final diagnosis of ILD, the tiny sample does not necessary reflect the entire pathological event which occurred in heterogeneous fashion throughout the lung. The overall distribution of disease and the uniformity of histologic findings cannot be judged from the microscopic and localized information obtained from lung biopsy. Third, radiological evaluation especially HRCT plays an important role for assessing ILD as HRCT can noninvasively evaluate what is going on in the lung entirely, however the each of the HRCT findings are nonspecific and its resolution does not reach the level of microscopy.

2.Importance of multidisciplinary discussion

Due to those problems, ATS/ERS guideline for IIP in 2002 proposed the dynamic interaction between pathologists, radiologists and pulmonologists to accurately diagnose IIPs. It is recommended that the final diagnosis should be rendered only after the pulmonologist, radiologist, and pathologist have reviewed all of the clinical,radiological,and pathological data obtained from the patient (CRP diagnosis). This method was acceded to the update guideline for IIPs2013 as a multidisciplinary approach. In ATS/ERS/JRS/ALAT statement for IPF 2011 also emphasize this integrated approach as using terms “multidisciplinary discussion:MDD). Flaherty et al. studied the kappa statistic for intraobserver agreement among expert clinicians evaluating ILD and showed that the kappa significantly improved as more clinical,radiologic and pathologic information was added,suggesting that clinicians had become more confident of their diagnoses with this process(AJRCCM 2004;170:904-910.).

3. Role of HRCT within MDD

It has been reported that the interobserver agreement for diagnosing ILD is better in HRCT than histopathological diagnosis. Confident rate of the diagnosis is also much higher in HRCT than histopathology. From these data, the primacy of histopathological diagnosis of ILD is deleted and the shift to the MDD was appeared. Among MDD, the role of HRCT can be summarized as follows;

a. HRCT can boost the diagnostic accuracy of ILD.

Diagnostic accuracy of HRCT depends largely on the presence of a likely cause and pretest probability and with these clinical information, the differential diagnosis of HRCT can be narrowed. If the biopsy specimen is insufficient to make correct pathological diagnosis, typical HRCT patternsconduct us toward the correct diagnosis.

b. Pathognomonic HRCT findings make a truly solo diagnosis.

Very specific and identical HRCT findings to one disease are powerful tools for making diagnosis themselves. For example, the diagnosis of alveolar proteinosis is relatively easy when crazy-paving pattern is observed on HRCT. Clinical information of BAL findings will promote this diagnosis.

c. An evaluation of the disease reversibility can be achieved.
If the homogenous ground opacity without honeycombing or traction bronchiectasis is demonstrated, it is speculated that the disease will be reversible with an appropriate treatment. On the other hand, if there are HRCT signs suggesting fibrosis including honeycombing, irregular traction bronchiectasis or marked reticular pattern, it is speculated that the disease is irreversible.

d. An evaluation of the prognosis is possible.
Extent of fibrosis on HRCT such as honeycombing or reticulation has been consistently linked mortality in IPF/UIP. It is also demonstrated that HRCT pattern is closely associated with mortality in acute exacerbation of IPF.

Day 2: Friday, 4th December 2015

Session: **Session 4B-3: Interstitial Lung Disease**
Time: **1700 – 1730**
Venue: **Conference Hall 3**

Advances in the treatment of IPF

– Takashi Ogura (Japan)

Idiopathic pulmonary fibrosis (IPF) is specific form of chronic, progressive, fibrosing interstitial pneumonia of unknown cause, limited to the lungs. The management generally includes some combination of supportive care, use of antifibrotic agents (pirfenidone, nintedanib), identification and treatment of comorbidities, and referral for lung transplant evaluation.

No medication has been found to cure IPF, but two antifibrotic agents, nintedanib and pirfenidone, appear to slow disease progression. For IPF patients who live in an area where either pirfenidone or nintedanib is available, we recommend initiating therapy with the available agents.However.The clinical course and prognosis of this disease may be variable and unpredictabl. Treatment of this disease is tailored to the disease severity and desires of the individual patient.

As pirfenidone was first licensed in 2008 in Japan, we experiences many patients who was treated with pirfenidone. Now that nintedanib, in 2015, has become available as a second treatment option.

The objective of this presentation is to review advances in the treatment of IPF and demonstrate our experiences in the real world.

Day 2: Friday, 4th December 2015

Session: **Session 4C-1: Pleural Diseases**
Time: **1600 – 1630**
Venue: **Conference Hall 1**

Unusual pleural diseases

– Anantham Devanand (Singapore)

The estimated prevalence of pleural effusion is 320 to 400 cases per 100,000 people in industrialized countries and the distribution of causes is related to the prevalence of underlying diseases. Congestive heart failure, bacterial pneumonia, tuberculosis, malignancy and pulmonary embolus account for most cases. This interactive case based discussion will focus on pleural effusions in young adult females. The differential diagnosis, diagnostic workup and management will be elaborated upon. The concept of a dissociated exudative pleural effusion i.e. exudate on protein and not lactate dehydrogenase criteria will be introduced. The determinants of pleural fluid protein are plasma protein concentration, protein reflection coefficient, capillary 'leakiness', solvent filtration and fluid bulk flow via lymphatics. In addition, the value of pleural manometry and the measurement of pleural elastance (pressure change divided by volume change) will be explored.

Day 3: Saturday, 5th December 2015

Session: **Session 5A-1: Respiratory Neurobiology and Sleep (2)**
Time: **0900 – 0930**
Venue: **Conference Hall 2**

The upper airway function in OSA

– Peter Eastwood (Australia)

Sleep poses several challenges to maintenance of effective airflow through the human upper airway. Sleep onset is accompanied by physiological changes that act to worsen airway patency, including a decreasedpharyngeal dilator muscle tone, decreased sensitivity of protective pharyngeal mechanoreceptor reflexes and decreased lung volume, the latter ofwhich acts to lessen the caudal forces transmitted via the trachea that 'stiffen' the pharyngeal walls and protect it from collapse.In individuals with an anatomically predisposed airway, such as those with a narrow pharyngeal airway and retrognathia, these changes result in upper airway narrowing (hypopneas) and collapse (apneas) during sleep (obstructive sleep apnea, OSA). Anatomical predisposition can also be modified over the course of a night as a consequence of changesin body position, head posture and jaw position.Emerging evidence supports an important role for non-anatomical factors in modifying upper airway behaviour during sleep. These include OSA-related increases in sensitivity of ventilatory control mechanisms that act to increase breathing instability,decreasesin the capacity of pharyngeal muscle activity to improve airway patency,and premature waking from an obstructive event (a low respiratory arousal threshold) resulting in disruptions to sleep continuity and decreased chance for pharyngeal muscle activity to restore airway patency during sleep. Careful consideration of the complex, multifactorial nature of OSA is required to optimise treatment of upper airway obstruction.

Day 3: Saturday, 5th December 2015

Session: **Session 5B-2: Paediatric Lung Diseases (1)**
Time: **0930 – 1000**
Venue: **Conference Hall 3**

Is intermittent use of inhaled corticosteroids a feasible option in children?

– Anne Goh (Singapore)

Current guidelines recommend the daily use of inhaled corticosteroids in children with persistent asthma. However, parents and physicians alike have concerns about putting young children on daily doses of inhaled corticosteroids, hence the attraction of intermittent use of inhaled corticosteroids for these children. What is the evidence for this?

Studies that have compared the use of daily vs intermittent use of inhaled corticosteroids in persistent asthma have consistently found that daily use of corticosteroids is better at achieving good asthma control compared to intermittent use which in turn is better than placebo. However, obviously there are some children who will be fine on intermittent treatment. Furthermore, is there a role of intermittent use of corticosteroids in children with intermittent wheezing?

Some of the difficulty is the correct identification of phenotypes for wheezing in children. Treatment can then be targeted at the group who will benefit from either treatment without the side effects of unnecessary or over treatment.

Day 3: Saturday, 5th December 2015

Session: **Session 5C-2: Bronchoscopy & Interventional Pulmonology**
Time: **0930 – 1000**
Venue: **Conference Hall 3**

Complications of Airway Stenting

– Philip Eng (Singapore)

Complications from airway stenting are relatively uncommon and can largely be prevented. An accurate diagnosis, thorough understanding of the patient's anatomy, meticulous techniques, experience supplemented by a spirit of self-critique can help to a large extent. I will illustrate some of these issues in this presentation.

Day 3: Saturday, 5th December 2015

Session: **Session 6A-1: Asthma (2)**
Time: **1100 – 1130**
Venue: **Conference Hall 2**

Identifying therapeutic phenotypes of severe asthma

– Kittipong Maneechotesuwan (Thailand)

Our understanding of asthma have advanced over time from a single disease to a complex of various phenotypes, with varied natural history, physiologies and responses to treatment. Early therapies treated most patients with asthma similarly, with bronchodilatorsand corticosteroids, but these therapies had varying degrees ofsuccess.Although Th2 inflammation is widely known seen in asthmatic airways, biologic therapy targeted towards these type 2 pathways were unsuccessful inall patients. Clinical approaches, both biased and laterunbiased/statistical approaches to large asthma patient cohorts have identified clinically relevant phenotypes that are determined by age of onset of disease and the presence of eosinophils. The paralleled molecular approaches to phenotyping developed an understanding that not all patients share a type 2inflammatory pattern.A subgroup of patients with severe asthma have refractory disease, with responses to nonspecific anti-inflammatory drugs such as inhaled corticosteroids that are dependent on the presence and type of airway inflammation. Severe asthma phenotypesis possibly driven by complicated/mixed Th2 and non-Th2 inflammation.Patients with severe asthma are characterized and identified by molecular and clinical characteristics together with the use of sputum, peripheral blood, and exhaled biomarkers. However, there are still some limitations in the use of these biomarkers, resulting from stability of the types of inflammation.

Day 3: Saturday, 5th December 2015

Session: **Session 6A-2: Asthma (2)**
Time: **1130 – 1200**
Venue: **Conference Hall 2**

What are the current and future treatment options for refractory asthma?

– Omar Sharif Usmani (UK)

Asthma is an umbrella term that covers a heterogeneous population each with a different phenotype and hence one drug cannot treat all and there is a need for personalised therapy. Refractory asthma (RA) is not very responsive to current standard therapy used for asthma, where there is relative corticosteroid insensitivity, and a real need for disease modifying agents. Drug development in patients with RA has concentrated its efforts on agents that inhibit specific biological targets, but so far these not been wholly effective in clinical trials, although there are 'responder' patients that have been identified. These agents can be broadly classified into broad spectrum anti-inflammatory drugs, inhibitors of lipid mediators, inhibitors of signal transduction pathways, anti-allergy treatments, immunotherapy, and cytokine modulators (Barnes, Sem Resp Crit Care Med 2012). The majority of agents developed have been cytokine modulators focused on the Th-2 pathways; particularly IL-5 and IL-4/IL-13. Use of Th-2 cytokine modulators in clinical trials has also allowed the exploration of Th-2 biomarkers such as sputum eosoiniphilia, blood eosinophilia, serum periostin, and exhaled nitric oxide, and CCL-17 that have allowed patient stratification into sub-group 'responder' patients. However, RA only accounts for ~10-15% of the asthmatic population, and expensive biologics targeting a single mediator or receptor may only be effective in even fewer patients as they can only be used in highly selected specific asthma phenotypes. What about the remaining 85% of the asthmatic population who remain poorly controlled despite optimal current therapy (Demoly, Eur Resp Rev 2012)? We should equally strive to improve the majority of our asthmatic patients. Clinical and research efforts should be focused on utilising effective drug delivery from inhaler devices, considering treating small airways (Usmani, Curr Opinion Pulm Med 2015), using long-acting muscarinic antagonists, considering amaintenance and reliever treatment approach (MART), treating comorbidities, and recognising our 'real-life' clinic population may often be excluded from clinical trials, such as smoking asthmatics.

Day 3: Saturday, 5th December 2015

Session: **Session 6A-3: Asthma (2)**
Time: **1200 – 1230**
Venue: **Conference Hall 2**

Role of biomarkers in severe asthma

– Dave Singh (UK)

We are increasingly recognising that severe asthma is composed of distinct subsets of patients with distinct clinical and inflammatory characteristics. The search for biomarkers to define patient subtypes, and to predict treatment responses, is critical for the future of personalised medicine in these patients. We also recognise the concept of endotypes, which are patient subgroups defined by biological mechanisms. Targeted endotype driven therapy can be facilitated by the development of robust biomarkers. This lecture will provide a state of the art review of severe asthma biomarkers in this rapidly evolving field.

Day 3: Saturday, 5th December 2015

Session: **Session 6B-1: Paediatric Lung Diseases (2)**
Time: **1100 – 1130**
Venue: **Conference Hall 3**

Update and controversies in the management of childhood pneumonia

– Anne Chang (Australia)

Pneumonia is the greatest contributor to childhood mortality and morbidity in resource-poor regions, while in high-income countries it is one of the most common reasons for clinic attendance and hospitalization in this age group. Furthermore, pneumonia in children increases the risk of developing chronic pulmonary disorders in later adult life.

While substantial advances in managing childhood pneumonia have been made, many issues remain, some of which will be highlighted in the talk.

Multiple studies are required as many factors that influence outcomes, such as etiology, patient characteristics and prevention strategies can vary between and within countries and regions. Also, outside of vaccine studies, most randomized controlled trials (RCTs) on pneumonia have been based in resource-poor countries where the primary aim is usually prevention of mortality.

Few RCTs have focused on medium to long-term outcomes or prevention. To advance the management of pneumonia, different tiers of primary outcomes are required, where in resource-rich countries medium to long-term sequelae should also be included and not just the length of hospitalization and readmission rates.

There are few longitudinal studies of pneumonia in otherwise healthy children that extend into adulthood. Birth cohort, longitudinal, case-control and retrospective studies have reported restrictive and obstructive lung function deficits, asthma, bronchiectasis and chronic obstructive pulmonary disease. Most studies though were limited by incomplete follow-up, some reliance upon parental recall, risk of diagnostic misclassification and potential confounders, such as nutrition, social deprivation and pre-existing small airways or lungs

Day 3: Saturday, 5th December 2015

Session: **Session 6B-3: Paediatric Lung Diseases (2)**
Time: **1200 – 1230**
Venue: **Conference Hall 3**

Bronchiectasis: is it a reversible disease?

– Anne Chang (Australia)

A paradigm presenting a spectrum related to airway bacteria, with associated degradation and inflammation products causing airway damage if untreated, entails protracted bacterial bronchitis (at the mild end) to irreversible airway dilatation with cystic formation as determined by HRCT (at the severe end of the spectrum). Increasing evidence suggest that progression of airway damage can be limited by intensive treatment, even in those predestined to have bronchiectasis (eg immune deficiency). Treatment is aimed at achieving a cure in those at the milder end of the spectrum to limiting further deterioration in those with severe 'irreversible' radiological bronchiectasis.

The airway inflammation in children with bronchiectasis is predominantly neutrophilic with evidence of neutrophilic activation such as elevated neutrophil elastase, MMP-9 and interleukin 8. This mechanism is also present in pre-bronchiectatic conditions like protracted bacterial bronchitis.

The natural history of bronchiectasis and mortality has altered with improvements in health and the environment suggests that with the implementation of other preventative factors, the progression of bronchiectasis could be ameliorated in the majority of children. Further most adults with bronchiectasis have had symptoms since childhood. Also, there is evidence demonstrating:

The effect that exacerbations and/or delayed treatment is associated with lung function decline

Children at risk of bronchiectasis can have normal lungs with early diagnosis and appropriate management, and

Appropriate treatment reduces exacerbations of bronchiectasis.

Clinical bronchiectasis is reversible only if it is in the early stages and thus attention to early diagnosis and intensive treatment is important.

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Day 3: Saturday, 5th December 2015

Session: **Session 6C-1: Pulmonary Circulation (1)**
Time: **1100 – 1130**
Venue: **Conference Hall 1**

Screening and diagnosis of pulmonary hypertension

– Aizai Azan Abd Rahim (Malaysia)

Pulmonary hypertension (PH) is a chronic and progressive disease which is life limiting. It is defined as an increase in mean pulmonary arterial pressure (mPAP) > 25 mmHg at rest as assessed by right heart catheterization as the gold standard. The upper limit of normal mPAP at rest is 20 mmHg. The clinical significance of a mPAP of between 21 to 24 mmHg remains controversial but patients who present within this range of mPAP should be followed up carefully especially those at risk of developing PH such as those with family history of PH, connective tissue disease, sickle cell anemia, portal hypertension, HIV and congenital heart disease.

Patients commonly present with progressive shortness of breath, which, as right ventricular function deteriorates, progresses to chest pain, fatigue, palpitations, dizziness, syncope, right ventricular failure and eventually premature death. Often, a high index of clinical suspicion is necessary due to the gradual nature of the development of symptoms. Early signs are often overlooked, which may delay diagnosis, often by many years. Furthermore, the presentation of PH may sometimes be modified by diseases, which cause or are associated with PH. This can ultimately have a damaging effect on disease prognosis, including early, preventable mortality by delaying initiation of therapy with proven PH specific treatment.

Much progress has since been made in the development and refinement of PH classification, available investigation tools and treatment options as reflected by the various international clinical practice guidelines, which will be discussed during the presentation. This will provide healthcare professionals with greater choice and scope for optimizing the management of patients with PH.

Day 3: Saturday, 5th December 2015

Session: **Session 6C-2: Pulmonary Circulation (1)**
Time: **1130 – 1200**
Venue: **Conference Hall 1**

Mechanism and management of hypoxia-induced pulmonary hypertension

– *Talant* **Sooronbaev** *(Kyrgyzstan)*

New pathophysiological and molecular aspects of hypoxia-induced pulmonary hypertension (HIPH) is associated with sleep apnea, even when adjusted for age and body mass index. Combined intermittent and chronic hypoxia due to sleep apnea and residence at high altitude may predispose to pulmonary hypertension. We have found high expression of PDE-5 in pulmonary arterioles at the patients with HIPH. PDE5 immunostaining is localised to muscularised distal arterioles in alveolar ducts and alveolar walls. Another interesting finding was that increased ET-1 levels play an important role in development of HIPH. Obtained the first evidence of a modest role of Rho-kinase in the maintenance of pulmonary artery pressure. We have a some evidence that HIPH is a genetically determined disease.

A serious problem related to the question of how to identifypatients with HIPH - especially for early detection. Evaluation of clinical symptoms is important, but difficult, because a lot of patients with HIPH to have mild or moderate pulmonary hypertension and without symptoms**.** Combination of ECG and echocardiography may be useful for screening high altitude pulmonary hypertension, especially in resource-limited settings. We observed good correlation between mean*P*pa estimated by Doppler echo and mean*P*pa measured invasively by right heart catheterization.

Not so much data on the treatment of HIPH. Clinical trails in Kyrgyzstan for the treatment of HIPH during last 10 yers have shown the effectiveness and prospects for the use of sildenafil (PDE-5 ingibitor), bosentan (receptor antagonist of ET-1) and fasudil (ROCK inhibitor).

Day 3: Saturday, 5th December 2015

Session: **Session 7A: Young Investigators Session**
Time: **1400 – 1530**
Venue: **Conference Hall 3**

Performance of TB Diagnostic Committee (TBDC) in Certifying Disease Activity Among Smear Negative Retreatment Cases in District IV of Manila, Philippines from January 2013 to June 2014

– Caroline Bernadette Olanka King Kay (Philippines)

Introduction: The TB Diagnostic Committee (TBDC) was established in the country to certify disease activity among presumptive smear negative pulmonary TB (PTB) cases and avoid unnecessary treatment. However, there are no available studies to evaluate the accuracy of TBDC decisions. Recent policy of requiring retreatment cases to undergo bacteriologic confirmation, through Xpert MTB/RIF or culture, now provides this opportunity.

Objectives: To determine the performance of the University of Santo Tomas Hospital (USTH) TBDC in certifying disease activity among smear negative retreatment cases in District IV of Manila, Philippines from January 2013 to June 2014, using Xpert MTB/RIF or culture as the standard. Secondly, to establish possible associations between symptoms and chest radiograph findings, which TBDC members correlate with disease activity, and bacteriologic confirmation through Xpert MTB/RIF or culture.

Methodology: Presumptive PTB cases from District IV of Manila, Philippines, from January 2013 to June 2014, with negative DSSM but deemed active by the TBDC, were listed and cross-tabulated with results of corresponding Xpert MTB/RIF or culture, symptoms and chest radiograph findings. Data analysis using descriptive statistics and Pearson Product Moment Correlation were done.

Results: Of the 952 cases evaluated during the study period, 257 (27.00%) were assessed to have active PTB. However, only 143 (55.64%) had available Xpert MTB/RIF or culture results. Twenty nine (20.28%) cases were bacteriologically confirmed. Symptoms highly correlated with disease activity were cough and sputum production. Ill defined infiltrates on chest radiograph had moderate correlation with disease activity.

Conclusion: There is limited data to definitively correlate TBDC disease activity assessment with bacteriologic confirmation. But this study shows that overdiagnosis is still likely despite screening by an expert panel. A prospective study is recommended to determine specificity and negative predictive value. In the absence of means for bacteriologic confirmation or expert opinion, cough and sputum production, together with ill defined infiltrates on chest radiograph can help primary physicians in deciding disease activity in smear negative TB.

Day 3: Saturday, 5th December 2015

Session: **Session 7A: Young Investigators Session**
Time: **1400 – 1530**
Venue: **Conference Hall 3**

Regression equations to estimate the two-minute walk distance (2MWD) in Malaysian adults aged 40 to 75 years

– Fatim Tahirah Mirza Mohd Tahir Beg (Australia)

Introduction: There is increased interest in using the two-minute walk test (2MWT) to assess functional exercise capacity. However, the distance achieved during this test (i.e. 2MWD) may be difficult to interpret in the absence of normative data derived from a local population. Regression equations to estimate the 2MWD exist for North American and Brazilian populations.

Aims: To develop regression equations to estimate the 2MWD in Malaysian adults and compare the 2MWD measured in this sample with the 2MWD estimated using existing equations.

Methods: Eighty-seven adults (43 males) aged 40 to 75 years performed two 2MWTs using a standardised protocol. Heart rate (HR) was recorded every 30s during the test.

Results: The better of two 2MWDs was 200±34m with males walking 33±6m further than females ($p<0.001$). The 2MWD was associated with age and change in HR (ΔHR) during the test; both $r>0.43$, $p<0.001$. Stepwise regression analysis showed that gender, age and ΔHR were independent contributors to the 2MWD. The following equation explained 73% of the variance in 2MWD (m): 196 - (1.1◊age, yr) + (1.0◊ΔHR) + (31.2◊gender [males=1, females=0]). When ΔHR was excluded from the equation, age and gender explained 47% of the variance in 2MWD (m): 279 - (1.7◊age, yr) + (35.9◊gender [males=1, females=0]). Regression equation derived from North American sample underestimates 2MWD in Malaysian adults. Bland and Altman plots showed the presence of proportional error between the 2MWD measured in this sample and the 2MWD estimated using both existing equations.

Conclusions: This is the first study to report 2MWD for Malaysian adults. The equations will facilitate interpretation of the 2MWD in clinical populations in Malaysia and other Asian countries with similar cultural backgrounds. Disparity between the measured 2MWD with that estimated using both of the existing equations highlights the importance of using regression equations derived in a local sample.

Day 3: Saturday, 5th December 2015

Session: **Session 7A: Young Investigators Session**
Time: **1400 – 1530**
Venue: **Conference Hall 3**

MPR2 Upregulation via in situ Gene Delivery or via Engineered Endothelial Progenitor Cells Alleviates Pulmonary Arterial Hypertension in a Rat Model

– Rebecca L Harper (Australia)

Introduction:
Reduced expression of the bone morphogenetic protein reception type-2 (BMPR2) is causally linked to familial, idiopathic and secondary forms of PAH. Thus we proposed that upregulation of BMPR2 may be therapeutic. As proof of concept, we previously attenuated PAH in animal models using BMPR2 targeted gene delivery using Adenoviral (Ad) vectors, but further understanding of the cell signalling mechanisms involved is required to progress this approach to the clinic. There are also shortcomings with viral vector approaches that need to be overcome. Endothelial progenitor cells (EPCs) are important for angiogenesis and tissue repair and have been shown to have altered function and abundance in patients with PAH. Manipulating these cells may be an alternate means to upregulate BMPR2 in lungs affected by PAH, thereby avoiding some of the limitations of viral gene delivery techniques and enabling easier clinical translation.

Aim:
To assess changes in PAH and the relevant pathways involved (Smad and non- Smad) following 1) targeted gene delivery of BMPR2 to pulmonary vascular endothelium and 2) intravascular delivery of EPCs transduced to overexpress BMPR2.

Methods:
1). In situ Ad vector transduction: A pulmonary endothelial- targeted Ad vector was used to deliver the BMPR2 gene to the pulmonary vascular endothelium of rats (n=8) with monocrotaline (MCT) - induced PAH. PAH amelioration was assessed through the Fulton Index at day 2 and hemodynamic measurements at day 10. Readings were taken using catheters inserted into the left carotid artery and right subclavian vein of each rat. Right ventricular systolic pressure (RVSP), mean pulmonary arterial pressure (mPAP), cardiac output using the thermodilution technique, mean systemic arterial pressure (mSAP), heart rate and temperature were all obtained. Fulton Index was measured as the right ventricle/ left ventricle + septum (RV/LV+S).Whole lungs were then removed, protein was extracted and analysed via western blot (n=6).

2). Ex-vivo EPC transduction then injection: Bone marrow cells were extracted from the femur of donor rats and cultured in selective endothelial medium (EGM-2MV, Lonza). After 6 days, cells were characterised as EPCs with flow cytometry as CD31+, CD106+, CD34dim and CD45dim, and through microscopy looking for EC morphology. Successful transfection of these cells was shown by western blot analysis of BMPR2 expression. Rats (n=8) were injected with MCT, and then at day 10, rats were intravenously injected with EPCs only, AdBMPR2 transfected EPCs, or uninjected. After a further 8-10 days, PAH was assessed via hemodynamic measurements as above.

Results:
1). Compared to rats that received control vector, BMPR2 transduction significantly ($p<0.05$) reduced Fulton Index at day 2, by 24.9% and day10 by 25.7%. RVSP and mean pulmonary

artery pressure (mPAP) at day 10 were reduced by 44.5% and 42.7% respectively. Extracted lungs given BMPR2 had a 2-fold increase in Smad1/5/8 activation, a 2-fold increase in PI3K and a 2-fold decrease in activated p38 MAPK at 2 days post BMPR2 treatment, but no changes were seen between the groups at 10 days. Other relevant non-Smad signalling proteins such as MEK1/2, RASGRP3, and ERK1/2 were shown to be increased in diseased rats compared to non-PAH controls, but BMPR2 transduction did not significantly alter this. RAS expression in the BMPR2 treated group is significantly increased compared to the MCT only treated group at 10 days.

2). After 8-10 days, PAH was shown to be attenuated in the BMPR2-transduced EPC group compared to rats that received untransfected EPCs, with significant ($p<0.05$) reduction in the Fulton Index by 16.3%, RVSP by 33.7% and mPAP by 15.7%

Conclusion:
BMPR2 upregulation ameliorates PAH via a number of pathways. This may be accomplished using direct in situ transduction, or via the novel use of modified EPCs. The mechanism by which EPCs exert their effect is yet to be determined but may involve the release of microvesicles. Use of cells may avoid certain adverse effects of viral vectors and be more amenable to clinical translation. Both treatment methods allow investigation of BMPR2 induced changes in Smad and non-Smad pathways.

Grant Support: NHMRC, NHF, RAH, Lions Research Foundation, Australian Postgraduate Award.

Day 3: Saturday, 5th December 2015

Session: **Session 7B-3: Clinical Allergy & Immunology**
Time: **1500 – 1530**
Venue: **Conference Hall 2**

The lung's response to air pollution

– Ian Yang (Australia)

Adverse health effects from outdoor air pollutants remain important, despite improvement in air quality in the past few decades. In the Asia-Pacific region, burning of biomass fuels are a major source of indoor air pollution. The exact mechanisms of lung injury from exposure to air pollutants are not yet fully understood. Using physiologically relevant model such as cells cultured at an air-liquid interface (ALI) can provide new insights into cellular response to air pollution. Respiratory cell line and animal models demonstrate distinct gene expression signatures in the transcriptome, arising from exposure to particulate matter or ozone. Particulate matter and other environmental toxins alter expression of microRNAs, which are short non-coding RNAs that regulate gene expression.Studying the genome (e.g. single nucleotide polymorphisms, SNPs), epigenome (e.g. methylation of genes), transcriptome (mRNA expression) and microRNAome (microRNA expression) has the potential to improve our understanding of the adverse effects of air pollutants. While it is clearly important to contain rising levels of air pollution, strategies also need to be developed to minimise the damaging effects of air pollutant exposure on the lung, especially for patients with chronic lung disease and for people at-risk of future lung disease. Future studies should be directed at testing potentially useful interventions against the adverse health effects of air pollution exposure.

Day 3: Saturday, 5th December 2015

Session: **Session 7C-1: Pulmonary Circulation (2)**
Time: **1400 – 1430**
Venue: **Conference Hall 1**

The journey to PAH and PH-related respiratory diseases

– Hiroshi Kimura (Japan)

The significance of prostacyclin, nitric oxide, and endothelin pathways has been well established in the pathogenesis and treatment strategies for pulmonary arterial hypertension (PAH). Assessing the Spectrum of Pulmonary Hypertension Identified at Referral Center (ASPIRE) registry demonstrated that patients with pulmonary hypertension (PH) due to respiratory diseases (group 3) exhibited the worst prognosis among all PH groups. However, the treatment strategies have not yet been established for group 3. The treatment algorithm of PAH was not recommended to apply to group 3 patients in the 2015 ESC/ERS guidelines. Despite that the pathogenesis of PAH is multifactorial and complicated, the prognosis and therapeutic outcomes of patients with PAH are assessed based on average of values in large database of cohort. Similar to the patients with PAH, the group 3 patients respond individually to various therapeutic strategies. Recently, a multi-institutional retrospective cohort study demonstrated the remarkable clinical significance of PAH-specific pharmacotherapy in patients with severe PH (mean PAP >= 35mmHg) associated with respiratory diseases (R-PH) and revealed the determinants of prognosis1). Clinical data of patients with four major respiratory diseases, including chronic obstructive pulmonary disease (COPD), combined pulmonary fibrosis with emphysema (CPFE), interstitial pneumonia (IP), IP associated with connective tissue disease (CTD-IP) with normal pulmonary arterial wedge pressure were retrospectively analyzed regarding clinical characteristics, treatment and prognosis. PAH-specific therapy was used for the treatment of 81% of patients. It was surprisingly found that the severe R-PH patients with the treatment of phosphodiesterase-5 inhibitors (PDE-5I) had significantly better survival from the date of diagnosis compared to those without the treatment of PDE-5I. This was particularly noted for patients with IP, CTD-IP and CPFE. Multivariate analysis revealed that the treatment with PDE-5I was an independent positive prognostic factor in severe R-PH patients. Future prospective clinical trial is expected to verify the present findings. Conflict of Interest: Actelion Pharmaceuticals Japan.

Day 3: Saturday, 5th December 2015

Session: **Session 7C-2: Pulmonary Circulation (2)**
Time: **1430 – 1500**
Venue: **Conference Hall 1**

Treatment update on CTEPH

– Nobuhiro Tanabe (Japan)

Chronic thromboembolic pulmonary hypertension (CTEPH) is a form of pulmonary hypertension that is characterized by the presence of organized thrombi in the proximal pulmonary arteries. The development of pulmonary hypertension in this disease is caused not only by the occlusion of proximal pulmonary arteries, but distal pulmonary vascular remodeling can also contribute to elevated pulmonary arterial pressure or resistance. We biopsied lung tissues from 17 CTEPH patients, and found that the extent of pulmonary arteriopathy was positively correlated with pulmonary vascular resistance evaluated at 1 month and 1 year after pulmonary endarterectomy. Interestingly, pulmonary venopathy was also recognized in most patients. The facts that riociguat increased 6 minutes walk distance and also decreased pulmonary vascular resistance even in patients with persistent pulmonary hypertension after surgery, may support the importance of pulmonary vascular remodeling in CTEPH.

Although the 5 year survival rate in medically treated non operable patients in our center is more than 80%, pulmonary endarterectomy (PEA)could cure patients and improve QOL much more than medical treatment with75-90% of 5 year survival rate. Recentlyballoon pulmonary angioplasty (BPA) may cure even non operable patients with 3.9% of the mortality from Japanese report. Recent guideline recommends that the assessment of operability and decisions regarding other treatment strategies should be made by a multidisciplinary team of experts.Interventional BPA may be considered inpatients who are technicallynon-operable or carry an unfavourablerisk:benefit ratio for PEA.

Finally it must be emphasized that this disease is treatable once patients with dyspnea on exercise have been accurately diagnosed with CTEPH.

Day 3: Saturday, 5th December 2015

Session: **Session 7C-3: Pulmonary Circulation (2)**
Time: **1500 – 1530**
Venue: **Conference Hall 1**

Treatment update on PAH

– Masayuki Hanaoka (Japan)

Pulmonary arterial hypertension (PAH) is a disease with the major lesions in pulmonary artery, resulting by sustained elevation of pulmonary vascular resistance due to pulmonary artery remodeling that is basically caused by hyperplasia of vascular endothelial cells and proliferation of smooth muscle cells. The PAH is defined by that the mean pulmonary artery pressure (mPAP) is over 25 mmHg plus pulmonary artery wedge pressure (PAWP) lower than 15 mmHg on the examination of right heart catheterization at rest. Right heart failure and respiratory failure are exhibited with progress of the disease, and the prognosis is poor with the mortality rate of 8-15% in idiopathic PAH/heritable PAH after one year from diagnosis. The 6minutes walking distance, electrocardiograms and cardiac ultrasound, plasma BNP/NT-proBNP values are followed during treatment, and the right heart catheterization should be performed on the requirement of therapeutic adjustment and disease situation. Regarding treatment, the specific PAH therapeutic agents are recommended after substantially administration of supportive therapy, such as, supervised rehabilitation, influenza and pneumococcal vaccinations, long-term oxygen therapy, and anticoagulant therapy. Specific PAH therapeutic agentsprecisely effect on pulmonary circulatory systeminvolving with vasodilation through endothelin pathway, nitric oxide pathway, and prostacyclin pathway correspondingly. Guiding by WHO pulmonary hypertension functional classification, it is fundamental to choose the agents depending on the severity of PAH, however, cooperation with specialists who are familiar with PAH is desirable during clinical practice. In recent years, new drugs such asmacitentan of the endothelin receptor antagonist, riociguat of the soluble guanylate cyclase stimulator, are in pharmacy market, which extends the treatment options. Moreover, the new evidences regarding the therapeutic strategy, for instance,the effectiveness of the initial combination therapy of ambrisentanwith tadalafil, are getting accumulated. This symposium will present an overview of the current state of the up-to-date advances concerning the diagnosis and treatment of PAH.

Day 4: Sunday, 6th December 2015

Session: **Session 8A-2: Tuberculosis (1)**
Time: **0930 – 1000**
Venue: **Conference Hall 2**

Public-Private Mix in TB Control: Critical appraisal of global evidence and local applications

– Charles Yu (Philippines)

The objectives of this talk are to critically review the evidence on the impact of PPM DOTS on tuberculosis including MDR TB and give a brief historical background. Much of the initial work in PPM DOTS was published in 2004 involving the work done in India, Vietnam, Kenya, Nepal and pilot programs in the Philippines through PhilCAT. In 2007, criticisms were raised that PPM-DOTS was being massively advocated and scaled up with less than robust evidence. More recently, in 2015, a systematic review by Lei, Tang and colleagues extensively reviewed 48 PPM programs worldwide and concluded that PPM was a promising strategy but was affected by contextual characteristics and needed integrated collaborative mechanisms, substantial financial support and continuous material input. A critical appraisal of this systematic review is provided, local experiences, best practices and lessons learned in running a private PPM facility will be shared.

Day 4: Sunday, 6th December 2015

Session: **Session 8B-3: Respiratory Structure & Function**
Time: **1000 – 1030**
Venue: **Conference Hall 1**

Tips and tricks to achieve best lung deposition with different inhaler devices

– Yuko Komase (Japan)

Asthma and chronic obstructive pulmonary disease (COPD) are primarily treated with inhaled drugs. As an enormous number of devices have become available in recent years, both medical professionals and patients have difficulty making full use of all options. A compound formulation comprised of the long-acting muscarinic antagonist (LAMA), a long acting β2-agonist (LABA), and inhaled corticosteroid (ICS) is scheduled to be launched in the near future. As delivery devices, metered dose inhalers (MDIs), dry powder inhalers (DPIs), and soft mist inhalers (SMIs) are currently available. In order to enhance efficacy, attention must be paid to the following issues. 1) Particle size of the inhaled drug: It is easier for smaller particles to reach the peripheral airways. Drugs with an appropriate particle size need to be selected in consideration of the patient's disease pathology. 2) Inlet flow. DPIs require a certain minimum level of inlet flow. 3) Devices that patients can easily use for inhalation: Choose devices that patients can operate easily and use for inhalation on a daily basis without difficulty. 3) Choose similar devices or compound drug formulations when several types of drugs need to be inhaled. 4) Patient adherence: Even if appropriate drugs are selected, good treatment cannot be guaranteed unless patients inhale them correctly. Motivation to perform inhalation therapy correctly can be enhanced if patients use inhaled drugs after thoroughly understanding the pathology of their disease and the necessity of drug treatment. 5) Always pay attention to the inhalation status of patients. It is important that all medical professionals supporting patients cooperate medically and support the treatment of patients. Determining treatment policy with patients is a practice now referred to as "concordance."

Day 4: Sunday, 6th December 2015

Session: **Session 9A-1: Tuberculosis (2)**
Time: **1200 – 1230**
Venue: **Conference Hall 2**

New TB diagnostic tests

– Somsak Rienthong (Thailand)

Tuberculosis is diagnosed by finding *Mycobacterium tuberculosis* bacteria in a clinical specimen taken from the patient by smear microscopy, culture together with ID and continues to DST for detection of MDRTB. Laboratory plays a critical part in this issue and provide clinician with invaluable information that is used to diagnose and guide treatment to the patient. Since quality test results require good quality specimens, the accurate, rapid microbiological diagnosis of TB and other mycobacterial infections begins with proper specimen collection and rapid transport to the laboratory. The patient must be provided with clearly presented and fully understood instructions for sputum collection to ensure the collection of the best possible specimen. In the majority,the diagnosis of TB relies primarily on the identification of AFB in sputum smears using a conventional light microscope. Acid-fast microscopy is easy and rapid, but it is low sensitivity and does not confirm a diagnosis of TB because some acid-fast-bacilli are not *M. tuberculosis*. Inspite of the high specificity, the sensitivity of the direct smear has beenreported to vary from 20 to 80%dependent on staff that has beenwell trained so that sufficient time is spent on preparing, staining,and reading each smear, with a well-functioning EQAprogram in place. Although new technologies are developed but microscopy will likely remain the primary tool for thelaboratory diagnosis of TB in many countries. Theimplementation of fluorescence microscopy has been hamperedin the past because traditional fluorescence microscopeswith short-lived mercury vapor lamps have been too costly for many setting. The current availability of newer,less-expensive microscopes with light-emitting diodes (LEDs)that can generate both light and fluorescence wavelengthsshould allow expanded feasibility studies under field conditions to determine the practicality of a more extensive implementation of fluorescence microscopy, including the need for training staff and the availability of LED microscopes and fluorescent stains.In addition, the WHO has recommended that conventional fluorescence microscopy be replaced by LED microscopy in all settings where fluorescent microscopy is now used and that LED microscopy be phased in as an alternative for conventional Ziehl-Neelsen (ZN) microscopy in both high and lowvolume laboratories. Also, countries implementing LED microscopy should introduction of adapted systems for internal quality control and external quality assessment.

The presence of acid-fast-bacilli (AFB) on a **sputum smear** microscopy or other specimen often indicates TB disease. Therefore, a **culture** is done on all initial samples to confirm the diagnosis. Culture examinations should be completed on all specimens, regardless of AFB smear results. Before culture in the laboratory, clinical specimens from nonsterile body sites such as sputum must be subjected to a pre-treatment involving digestion, homogenization, decontamination,and concentration. However, the efficacy of these procedures is highly influenced by the time of exposure to the reagent used for decontamination, the toxicity of that reagent, the efficiency of centrifugation, and the killing effect of heat build-up duringcentrifugation. There is evidence that even the mildest decontamination methods, such as the widely used *N*-acetyl-L-cysteine, NALC–NaOHmethod, can kill about 33% of the mycobacteria in a clinical specimen, while more overzealous methods can kill up to 70% of the mycobacteria.Culture is more sensitive than AFB smear microscopy for the detection of *M. tuberculosis*: while microscopy requires approximately 5,000 to 10,000 AFB/ml of sputum for detection, culture can detect as few as 10 to 100 viable bacteria/ml. Both solid and liquid method, Bactec MGIT 960 – 320, can be used for cultivation.While liquid media are preferred for the rapid initial isolation of mycobacteria. LJ medium can be prepared locally, has good buffer capacity, has a shelf-life of several months when refrigerated, supports the growth of most mycobacterial species, and contains malachite green in the medium to inhibit the growth of most contaminants. However, there are disadvantages such asLJ medium can vary from

batch to batch in its ability to support the growth of TB depending on the quality of the eggs used, contaminants are not completely eliminated but only suppressed by the malachite green and can overgrow *M. tuberculosis* during subsequent testing. In spite of these drawbacks, there are strains of the MTBC that will grow better or only on solid media, and thus, the CDC-recommended gold standard for the detection of TB is to inoculate at least one tube each of solid and liquid media. Lateral flow ImmunochromatographyAssays (ICA), SD – Bioline for instance, has been usedfor the rapid MTB identification, with in 15 mins,instead of biochemical techniques such as Niacin test, Nitrate reduction test and Catalase test which it does take a month for the result. Now, molecular techniques, has been introduced and as the newest technique for ID of MTBC.

For all patients, the initial *M. tuberculosis* isolate should be tested for drug resistance. It is crucial to identify drug resistance as early as possible to ensure effective treatment. So,AFB diagnosis is just the beginning of the long to cure. Drug susceptibility patterns should be repeated for patients who do not respond adequately to treatment or who have positive culture results despite 3 months of therapy. Both phenotypic and genotypic test can be used for detection of DS – TB and DR - TB from direct specimen or from isolated culture. Phenotypic DST can see the ability of organism can grow on the media contain drug at critical concentration and genotypic DST can detect point mutation gene of essential drugs both First line drug (FLD)and second line drug (SLD).To ensure the accuracy of susceptibility testing results, it is good practice to confirm all drug resistances either by a second method or by a second laboratory. However, quality control tests should be performed in every batch of test with*M. tuberculosis*H37Rv or another well characterized strain that is susceptible to all standard antituberculosisagents and strain that knownresistance pattern except resistance to H and R.

Molecular techniques that could reduce turnaround times in the laboratory for diagnosing TB and detection of drug resistance are being developed and evaluated in many countries. LPAs for the rapid diagnosis of TB and /or detection of RIF resistance and M/XDRTB are currently available on the market such as GenotypeMTBDRplus and Genotype MTBDR*sl*.These techniques have already approved tools for detection of drug resistance-associated genetic mutations directly from the sputum of smear-positive patients or from the isolated culture.RT-PCR based assay and single provider,Xpert MTB/RIF, is a fully automated system that allows a relatively untrained operator to perform sample processing, DNA amplification, and detection of *M. tuberculosis* and screening for rifampicin resistance in less than 2 hours and only minutes of hands-on time..Results can be available while patient waits in clinic. The only manual step, adding sample treatment reagent to the specimen cupbefore loading the cartridge, kills over 99.9% of TB bacilli in the specimen. The test detects MTB in essentially all smear-positive samples and the majority ofsmear-negative samples. Xpert MTB/RIF is real an important innovation for detecting TB in its early stages and hence reducing the transmission in the community.The presence of non-tuberculous mycobacteria does not confound testing. The cartridges are stable at room temperature. Now, thesame device can be used for HIV viral load detection, Flu virus and others diseases.A reliable source of electricity is needed as well as a room that can be secured outside working hours. All buffers and reagents are included in the cartridge package.With the introduction of Xpert MTB/RIF and other technologies, early M/XDRTB diagnosis is becoming possible.

Non-molecular technique TB-LAM Ag, Lipoarabinomannan (LAM), was identified as a promising target for antigen detection for TBdiagnosis due to its temperature stability. LAM-based assays are currently being developed by a number of commercial companies,Alere company USA, and preliminary results indicate their potential applicability in the rapid diagnosis of TB by detecting LAM in urine, especially in TB-HIV patient.LAM-based assays are included in the WHO TBdiagnosis re-tooling programme.MTB antigen detection provides direct evidence of TB. Such as LAM, 65Kd, 14 Kd antigenswere widely used. It is very quick and easy to perform. Main limitation is low sensitivity. It does not rule out TB in patients with poor antibody response.

Day 4: Sunday, 6th December 2015

Session: **Session 9A-3: Tuberculosis (2)**
Time: **1300 – 1330**
Venue: **Conference Hall 2**

Diagnosis and treatment dilemma of nontuberculous mycobacterial infection

– Erlina Burhan (Indonesia)

The incidence/prevalence of Nontuberculosis Mycobacterium(NTM) infection appears to be increasing nowadays. There are at least 140-150 species identified with variety of pathogenicity. The most common organisms involved in human infection are *M. kansasii, M. avium, M. intracellulare, M. chelonae* and *M. abscessus*. The symptoms caused by NTM infection can vary from no symptoms to severe cough, fatigue, and weight loss.

Diagnosis of NTM lung disease is often delayed, because the symptoms are similar to other lung diseases like COPD, bronchitis or bronchiectasis. Failure to recognize NTM infection lead to misdiagnosis chronic pulmonary Tuberculosis.However, once NTM infection is suspected, diagnosis is not difficult. ATS /IDSA criteria required clinical and microbiological or histological finding to diagnose NTM. However, NTM should be identified to the species level. Methods of rapid species identification include; commercial DNA probes (MAC, *M. kansasii*, and *M. gordonae*), high-performance liquid chromatography (HPLC). For some NTM isolates, especially rapidly growing mycobacterial isolates (*M. fortuitum*, *M abscessus*, and *M. chelonae*), other identification techniques may be necessary (extended antibiotic *in vitro* susceptibility testing, DNA sequencing).

Treatment of non tuberculosis mycobacterium (NTM) infection of the lung is dependent upon the species of the infecting organism. Treatment may be difficult because NTM bacteria may be resistant to many common types of antibiotics. For some patients, the same drugs used to treat tuberculosis (TB) will be recommended. To avoid becoming resistant to medications, several types of antibiotics can be taken at the same time. These drugs may cause side effects so that monitoring should be taking place closely during treatment regimen. The length of treatment varies, depending on the severity of the disease. Sometimes response to treatment is slow and often incomplete.*M. kansasii* is easier to treat and often can be killed with only three anti-TB medications. On the other hand, organisms such as *M. avium*, *M. chelonae* and *M. abscessus* are among the most stubborn germs. They are more difficult to treat. Three to five medications may be needed. Surgery also may be an option of treatment, if the disease is localized.

Key words: Diagnosis, Nontuberculosis Mycobacterium, treatment

Day 4: Sunday, 6th December 2015

Session: **Session 9B-1: Interstitial Lung Diseases (2)**
Time: **1200 – 1230**
Venue: **Conference Hall 3**

Prevalence and incidence of ILD in the Asia-Pacific region

– Yoshikazu Inoue (Japan)

Epidemiological studies of interstitial lung diseases (ILDs) are fundamentally important to understand the pathogenesis and to develop the strategies for treatment. However data on incidence, prevalence, and mortality of these diseases vary across studies or countries because of different criterion and/or method. So establishment of standardized international diagnostic criteria is the key step for the epidemiological studies.

Among ILDs, idiopathic pulmonary fibrosis (IPF) is one of the major important ILDs with the worst prognosis. Before 2000, not only the diagnostic criteria, but also the name differed between the countries such as IPF, cryptogenic fibrosing alveolitis, and chronic type of idiopathic interstitial pneumonia. After the publications of the international consensus statement in 2000 (ATS/ERS, Am J Respir Clit Care Med, 2002, 165: 277) and the guideline in 2011 (Raghu et al. Am J Respir Clit Care Med, 2011, 183: 788), many important epidemiological studies and global clinical trials have been conducted about IPF. On the other hand, the results of the global IPF mega-studies, which used modified criteria, have influenced the current diagnostic criteria (e.g. IPF, acute exacerbation).

Most of epidemiological studies have been published from USA and Europe, but a few from Asia-Pacific region including Japan, Korea, and Taiwan.

In this symposium, the diagnostic criteria, epidemiological data including incidence, prevalence from Asian-Pacific countries will be discussed comparing USA and European data. In addition to IPF, epidemiological data about other ILDs, such as pulmonary alveolar proteinsis, sarcoidosis, etc. will be shown.

Day 4: Sunday, 6th December 2015

Session: **Session 9B-3: Interstitial Lung Diseases (2)**
Time: **1300 – 1330**
Venue: **Conference Hall 3**

The ABC of hypersensitivity pneumonitis – from the acute to chronic stages

– Tengku Saifudin Tengku Ismail (Malaysia)

Hypersensitivity Pneumonitis (HP) is a form of diffuse granulomatous ILD caused by exposure to a wide variety of inhaled organic particles. In susceptible individuals, these antigens provoke an exaggerated immune response of the small airways and lung parenchyma. An intriguing question is why only a few exposed individuals develop the disease.

HP represents a diagnostic challenge because of the absence of any unique features that distinguish it from other ILDs. The diagnosis of HP relies on a high level of clinical suspicion, the recognition of antecedent antigen exposure, and a constellation of clinical, radiologic, laboratory, and pathologic findings. In 40% of histological proven cases of HP, the offending agent is not identified.

HP may present as acute, subacute or chronic disease but frequently the clinical features overlap. In acute exacerbation, an accelerated respiratory deterioration with the presence of new bilateral ground glass opacities on HRCT in patients with chronic HP is present.

Early recognition of the disease and prevention of long-term antigen exposure are necessary to avoid progression to irreversible fibrosis, as sustained antigen inhalation is associated with adverse outcome. Prednisolone is the mainstay of treatment for subacute disease. In the absence of clinical response to treatment and progressive fibrosis, lung transplant should be recommended.

Day 4: Sunday, 6th December 2015

Session: **Session 9C-3: Critical Care Medicine**
Time: **1300 – 1330**
Venue: **Conference Hall 1**

Early mobilisation of the ventilated patient: from evidence to practice

– Li-Ling Tai (Malaysia)

Bed rest and sedation is assumed to be beneficial in the critically ill patients for preventing complications, conserving metabolic resources and providing comfort. However, bed rest is known to cause the deconditioning effects of critical illness, which is defined as the multiple changes in organ system physiology that are induced by inactivity and reversed by activity. These include changes in muscle fibers, inflammatory markers and metabolic parameters. Immobility is also a known risk factor for complications such as respiratory insufficiency, deep vein thrombosis and pressure ulcers.

Early mobilisation helps in providing patient comfort, promoting psychological well-being, improving respiratory function and preventing the complications of bed rest. The ultimate goals of early mobility are to promote maximal level of independence and cardiovascular fitness before hospital discharge. Current literature supports that early mobility in patients in intensive care settings including those on mechanical ventilation is feasible, safe, reduced ICU and hospital length of stays and did not increase costs.

Mobilisation techniques include active/active assisted limb exercises, active moving or turning in bed, getting out of bed to chair via mechanical lifting machines or slide boards, sitting on the edge of the bed, standing, standing transfers from bed to chair, and even walking.

The major challenges to mobilising critically ill patients are fears of accidental dislodgement of tubes and devices and concerns over the strain that mobility could have on the patient's oxygenation and haemodynamic status. Other issues include personnel and equipment resources, pain and discomfort, the time and priority of mobilisation.

There is a need for a cultural shift in the intensive care settings from prolonged bed rest to early mobilisation in the ventilated patients to decrease the morbidity of the ICU stay.

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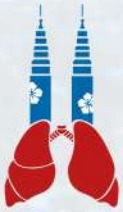

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Relvar® Ellipta® is indicated for the symptomatic treatment of adults with COPD with a FEV_1 <70% predicted normal (post-bronchodilator) with an exacerbation history despite regular bronchodilator therapy[1]

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Asthma	Adults and adolescents aged 12 years and over One inhalation of Relvar Ellipta 100/25 micrograms or 200/25 micrograms once daily. If symptoms arise in the period between doses, an inhaled, short-acting beta2-agonist should be taken for immediate relief. If patients are inadequately controlled on Relvar Ellipta 100/25 micrograms, the dose can be increased to 200/25 micrograms, which may provide additional improvement in asthma control. The maximum recommended dose is Relvar Ellipta 200/25 micrograms once daily.
COPD	Adults aged 18 years and over One inhalation of Relvar Ellipta 100/25 micrograms once daily. Relvar Ellipta 200/25 micrograms is not indicated for patients with COPD.
Elderly patients (>65 years) and Renal impairment:	No dose adjustment is required in this population
Hepatic impairment	Caution should be exercised when dosing patients with hepatic impairment who may be more at risk of systemic adverse reactions associated with corticosteroids. For patients with moderate or severe hepatic impairment the maximum dose is 100/25 micrograms.

Method of administration: Relvar Ellipta is for inhalation use only. It should be administered at the same time of the day, each day. After inhalation, patients should rinse their mouth with water without swallowing. **Contraindications:** Hypersensitivity to the active substances or to any of the excipients like lactose monohydrate and magnesium stearate. Patients with rare hereditary problems of galactose intolerance, the Lapp lactase deficiency or glucose-galactose malabsorption should not take this medicinal product.

Warnings & Precautions:

Deterioration of disease	Fluticasone furoate/vilanterol should not be used to treat acute asthma symptoms or an acute exacerbation in COPD, for which a short-acting bronchodilator is required. Increasing use of short-acting bronchodilators to relieve symptoms indicates deterioration of control and patients should be reviewed by a physician. Patients should not stop therapy with fluticasone furoate/vilanterol in asthma or COPD, without physician supervision since symptoms may recur after discontinuation.
Paradoxical bronchospasm	Paradoxical bronchospasm may occur with an immediate increase in wheezing after dosing. This should be treated immediately with a short-acting inhaled bronchodilator. Relvar Ellipta should be discontinued immediately, the patient assessed and alternative therapy instituted if necessary.
Cardiovascular effects:	Cardiovascular effects, such as cardiac arrhythmias e.g. supraventricular tachycardia and extrasystoles may be seen with sympathomimetic medicinal products including Relvar Ellipta. Therefore fluticasone furoate/vilanterol should be used with caution in patients with severe cardiovascular disease.
Systemic corticosteroid effects	Systemic effects may occur with any inhaled corticosteroid, particularly at high doses prescribed for long periods. These effects are much less likely to occur than with oral corticosteroids. Possible systemic effects include Cushing's syndrome, Cushingoid features, adrenal suppression, decrease in bone mineral density, growth retardation in children and adolescents, cataract and glaucoma and more rarely, a range of psychological or behavioural effects including psychomotor hyperactivity, sleep disorders, anxiety, depression or aggression (particularly in children). Fluticasone furoate/vilanterol should be administered with caution in patients with pulmonary tuberculosis or in patients with chronic or untreated infections.
Hyperglycaemia	There have been reports of increases in blood glucose levels in diabetic patients and this should be considered when prescribing to patients with a history of diabetes mellitus.
Pneumonia in patients with COPD	An increase in pneumonia and pneumonia resulting in hospitalization has been observed in patients with COPD receiving fluticasone furoate/vilanterol. In some incidences these pneumonia events were fatal. Physicians should remain vigilant for the possible development of pneumonia in patients with COPD as the clinical features of such infections overlap with the symptoms of COPD exacerbations. Risk factors for pneumonia in patients with COPD receiving fluticasone furoate/vilanterol include current smokers, patients with a history of prior pneumonia, patients with a body mass index <25 kg/m2 and patients with a (forced expiratory volume) FEV1<50% predicted. Relvar Ellipta 200/25 micrograms is not indicated for patients with COPD. The incidence of pneumonia in patients with asthma was common at the higher dose. The incidence of pneumonia in patients with asthma taking fluticasone furoate/vilanterol 200/25 micrograms was numerically higher compared with those receiving fluticasone furoate/vilanterol 100/25 micrograms or placebo.

Caution must be taken when administering with beta-blockers, CYP3A4 inhibitors (e.g. ketoconazole, ritonavir), P-glycoprotein inhibitors and Sympathomimetic medicinal products or in pregnancy and women who are breast feeding. (Refer to full prescribing information BEFORE prescribing to patient). **Adverse Events:** The following has been used for the classification of frequencies: very common (≥1/10); common (≥1/100 to <1/10); uncommon (≥1/1,000 to <1/100).

System organ class	Adverse reaction(s)	Frequency
Infections and infestations	Pneumonia, Upper respiratory tract infection, Bronchitis, Influenza, Candidiasis of mouth and throat	Common
Nervous system disorders	Headache	Very common
Cardiac disorders	Extrasystoles	Uncommon
Respiratory, thoracic and mediastinal disorders	Nasopharyngitis Oropharyngeal pain,Sinusitis, Pharyngitis, Rhinitis, Cough, Dysphonia	Very common Common
Gastrointestinal disorders	Abdominal pain	Common
Musculoskeletal and connective tissue disorders	Arthralgia Back pain Fractures	Common
General disorders and administration site conditions	Pyrexia	Common

Please read the full prescribing information prior to administration, available from: **GlaxoSmithKline Pharmaceutical Sdn Bhd (3277-U) Level 6, Quill 9, 112 Jalan Semangat, 46300 Petaling Jaya, Selangor Darul Ehsan, Malaysia.** Abbreviated Prescribing Information Version 01 based on EUSPCMay2014 MAL 1 Aug 2014 and EUSPC May2014 MAL 1 Aug 2014. Date of creation: 24 June 15

Relvar® Ellipta® was developed in collaboration with Theravance, Inc.

Before prescribing, please refer to the full prescribing information,which is available upon request.

Adverse events should be reported to drugsafetyinfo.my@gsk.com.

For Medical/Healthcare Professionals Only

MY/FFT/0032/15 08/16

ICS – inhaled corticosteroid; **LABA** – long acting beta$_2$-agonist.

*Patients' current or previous maintenance inhalers: Handihaler, DISKUS, MDI/HFA (COPD).[3]

References: 1. Relvar® Ellipta® 100/25 Malaysia Prescribing Information EUSPC May2014 MAL_1 August 2014. **2.** Boscia JA *et al.* Effect of Once-Daily Fluticasone Furoate/Vilanterol on 24-Hour Pulmonary Function in Patients With Chronic Obstructive Pulmonary Disease: A Randomized, Three-Way, Incomplete Block, Crossover Study. *Clin Ther.* 2012; 8:1655-66. **3.** Svedsater H *et al.* Qualitative assessment of a two-strip dry powder inhaler (ELLIPTA™) for COPD and asthma. *BMC Pulm Med.* 2013; **13:** 72.

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THE FIXED COMBINATION THAT DELIVERS GREATER EFFICACY PER µg OF STEROID IN THE TREATMENT OF ASTHMA[1-3]*

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Foster can **REACH** *small airways* [4]

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Foster can **TREAT** *small airways* [5]

BENEFIT

Foster provides greater clinical **BENEFIT** *vs. larger particle formulation* [6]

Further information on the product are available on request

1. Foster SmPC
2. Fabbri et al. Expert Opin Pharmacother 2008; 9(3): 479-490
3. Contoli et al. Allergy 2010; 65(2): 141-151
4. De Backer et al. J Aerosol Med Pulm Drug Deliv 2010; 23(3): 137-148
5. Vos W et al. Respiration 2013;86(5): 393-401
6. Huchon et al. Respir Med 2009; 103: 41-49

* when compared to conventional beclometasone dipropionate

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