2026
台灣胸腔暨重症加護醫學會
夏季會

2026 Summer Workshop of Taiwan Society of Pulmonary and Critical Care Medicine

KAOHSIUNG

June 27-28, 2026 (Sat-Sun)

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1. 2024 Jan 29;60(2):236. doi: 10.3390/medicina60020236.

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2026
台灣胸腔暨重症加護醫學會夏季會

2026 Summer Workshop of Taiwan Society of Pulmonary and Critical Care Medicine

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演講摘要

理事長序

各位會員先進大家好：

一年一度的夏季會即將隆重登場，在此謹代表理監事會，誠摯歡迎各位會員、專家學者以及各界好友共襄盛舉。今年夏季會移師南台灣，在美麗熱情的高雄市舉辦，並特別選擇於高雄萬豪酒店擴大辦理，期盼透過更完善舒適的會議空間與更豐富多元的學術內容，為所有與會者帶來兼具專業深度與交流溫度的學術盛會。

本次夏季會課程規劃涵蓋胸腔暨重症醫學各重要領域，包括呼吸道疾病、間質性肺病、肺部介入、肺感染與結核病、睡眠醫學、肺部環境與職業醫學、重症醫學及肺腫瘤等主題，內容緊扣最新臨床趨勢與國際治療發展。大會特別邀請國內外優秀專家學者進行專題演講與經驗分享，從基礎研究、臨床診療到精準醫療與跨領域整合，皆有精彩且深入的探討，相信能帶給所有會員豐富的收穫與啟發。

今年大會亦特別規劃「Connecting Horizons: TSPCCM Meets APSR」國際交流專題，邀請亞太地區重要學者蒞臨演講，針對 COPD 治療新趨勢、肺癌最新進展以及國際重要呼吸學期刊 Respirology 投稿經驗等議題進行交流，不僅展現我國胸腔暨重症醫學與國際接軌的成果，也期盼藉此深化與亞太各國之間的學術合作與友誼。

此外，大會安排多場 Satellite Symposium 與青年工作小組專題，希望促進不同世代醫師之間的交流與傳承，並鼓勵年輕醫師積極投入臨床研究與國際發表，持續為台灣胸腔暨重症醫學注入新的能量。

為增進會員間的互動與情誼，大會晚宴亦將於高雄萬豪酒店盛大舉行，在精彩學術活動之外，提供大家輕鬆交流與聯誼的機會。誠摯邀請各位會員踴躍參與，一同在港都高雄共享這場充滿知識、友誼與熱情的年度盛會。

敬請大家預留時間，期待與各位在高雄相見！
並祝大家身體健康、萬事如意！

陳育民

台灣胸腔暨重症加護醫學會
理事長

大會籌備處暨第 19 屆理、監事名單

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監事	黃崇旂	醫師／長庚醫療財團法人林口長庚紀念醫院
監事	謝俊民	醫師／奇美醫療財團法人奇美醫院

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執行秘書	洪緯欣	醫師／屏東榮民總醫院
執行秘書	張山岳	醫師／三軍總醫院

Jun 27 SAT

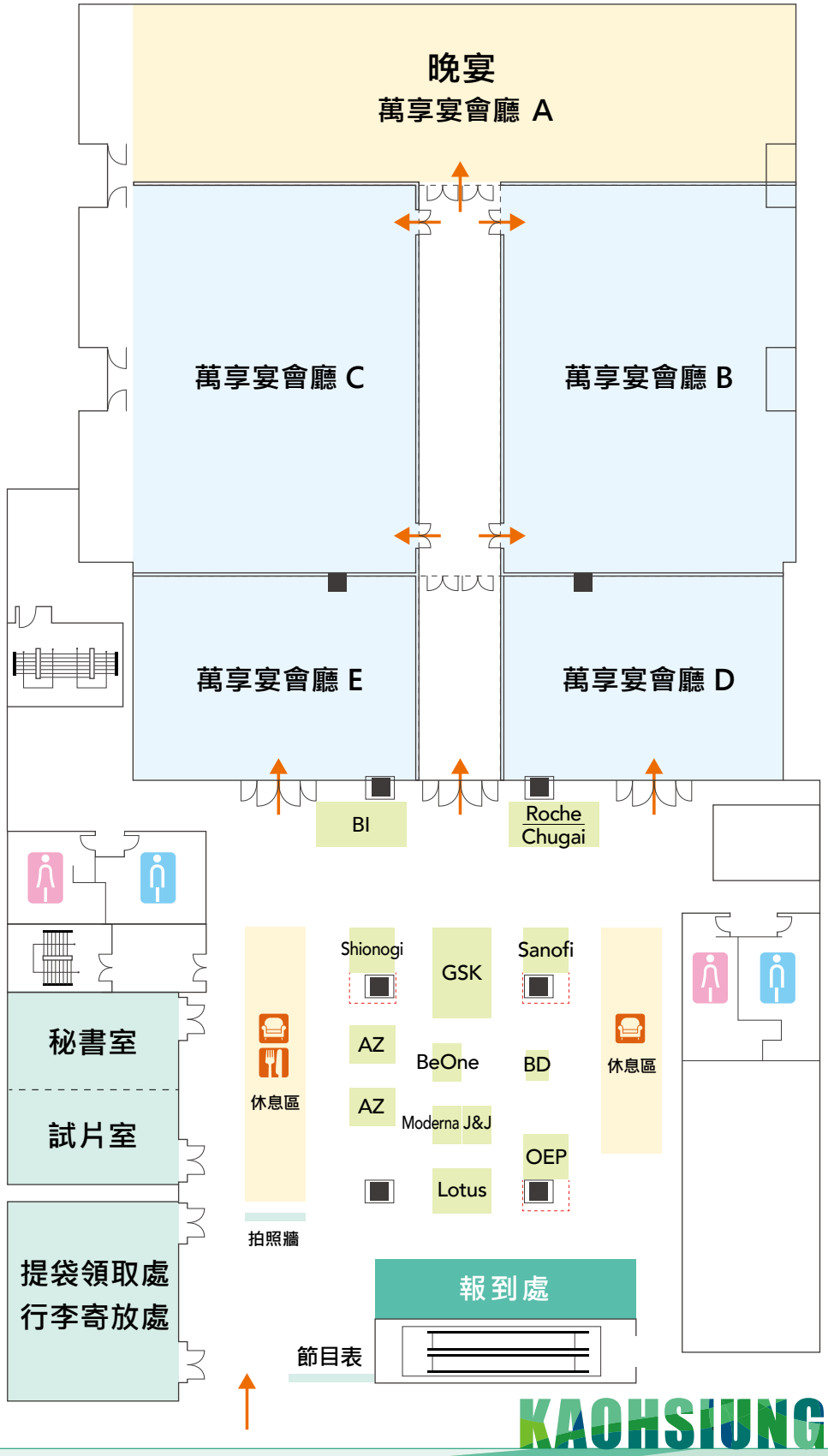
TIME	萬享宴會廳 B	萬享宴會廳 C	萬享宴會廳 D	萬享宴會廳 E
11:30	**報 到** 【請持身份證刷到】			
12:00 13:10				友華生技醫藥股份有限公司 Extrafine Triple Therapy: A New Approach to Asthma Control 講師：陳彥甫 醫師 座長：陳崇裕 醫師 Targeting Small Airways with Extrafine ICS/LABA in Asthma 講師：陳威志 醫師 座長：鄭世隆 醫師
13:20 | 14:00	呼吸道疾病 (Airway Disease) Obesity-Related Response to Biologics in Severe Asthma: How to Choose? 講師：黃俊凱 醫師 座長：陳育民 醫師	間質性肺病 (Interstitial Lung Disease) Acute Exacerbation of Interstitial Lung Disease: Early Diagnosis and Treatment 講師：林玠模 醫師 座長：林鴻銓 醫師	肺部介入 (Interventional Bronchoscopy) Advances in Mediastinal Tissue Acquisition in the Era of Interventional Pulmonology 講師：黃彥翔 醫師 座長：何肇基 醫師	青年工作小組 (Youth Committee) From 'Seeing' to 'Reaching': Practical Clinical Applications of CT Navigation and Lung Tumor Ablation 講師：張凌愷 醫師 座長：徐武輝 醫師
14:00 | 14:40	呼吸道疾病 (Airway Disease) Biological Treatment in COPD: What Is Promising? 講師：黃偉彰 醫師 座長：彭殿王 醫師	間質性肺病 (Interstitial Lung Disease) Comorbidities in Idiopathic Pulmonary Fibrosis and Progressive Pulmonary Fibrosis 講師：唐士恩 醫師 座長：杭良文 醫師	肺部介入 (Interventional Bronchoscopy) Endobronchial Microwave Ablation- A Single Center Experience 講師：施慧瑄 醫師 座長：夏德椿 醫師	青年工作小組 (Youth Committee) From Clinical Data to High-Impact Publications: Translational Strategies for Building and Publishing ILD Cohort Studies 講師：傅彬貴 醫師 座長：賴俊良 醫師
14:40 15:10	**Coffee Break**			
15:10 | 15:50	呼吸道疾病 (Airway Disease) Comparative Effectiveness of Biologic Classes in Severe Asthma 講師：吳秉宸 醫師 座長：黃崇旂 醫師	肺感染及結核病 (Tuberculosis and Infection Control) Promises and Pitfalls: Respiratory Vaccines in Patients with Chronic Lung Diseases 講師：鄭孟軒 醫師 座長：謝俊民 醫師	睡眠醫學 (Sleep Medicine) 15:10-15:30 Sleep-Related Breathing Disorders – CSA 講師：廖培雅 醫師 座長：陳寧宏 醫師 15:30-15:50 Sleep-Related Breathing Disorders – OSA 講師：孫傳硯 醫師 座長：陳寧宏 醫師	肺部環境與職業醫學 (Pulmonary Environment and Occupation Medicine) The Health Burden of Air Pollution in Taiwan 講師：蘇一峰 醫師 座長：彭忠衍 醫師
15:50 | 16:30	呼吸道疾病 (Airway Disease) COPD or Severe Asthma: from Clinical, Functional and Pathological Views 講師：吳玟叡 醫師 座長：鄭世隆 醫師	肺感染及結核病 (Tuberculosis and Infection Control) Less Is More? The Potential Role of Short-Course Tuberculosis Therapy in Taiwan 講師：樹金忠 醫師 座長：古世基 醫師	睡眠醫學 (Sleep Medicine) 15:50-16:10 Sleep-Related Hypoventilation (SRH) Disorders: Pathophysiology and Clinical Subtypes 講師：鍾心珮 醫師 座長：鍾飲文 醫師 16:10-16:30 Sleep-Related Hypoxemia: Pathophysiology and Clinical Implications 講師：鄭至宏 醫師 座長：鍾飲文 醫師	肺部環境與職業醫學 (Pulmonary Environment and Occupation Medicine) Work-Related Asthma 講師：潘奕宏 醫師 座長：林基正 醫師
16:30 17:40	台灣百靈佳殷格翰股份有限公司 Balancing Efficacy and Safety: A Holistic Approach to Adjusting Inhaled Corticosteroid Intensity 講師：黃俊凱 醫師 座長：王金洲 醫師 Acute Exacerbation in IPF 講師：陳威志 醫師 座長：陽光耀 醫師	賽諾菲股份有限公司 Precision Biologic Selection: Unlocking Higher Clinical Remission in Severe Asthma 講師：Prof. Atsuyasu Sato 座長：李岡遠 醫師 Dupilumab Across Respiratory Indications: Evidence from Clinical Trials and Real-World Practice 講師：蕭逸函 醫師 座長：簡榮彥 醫師	荷商葛蘭素史克藥廠股份有限公司台灣分公司 座長：林鴻銓 醫師 GOLD Highlights: COPD Exacerbation and RSV related CV-risk 講師：許健威 醫師 Early Optimization With Triple Therapy 講師：蕭惠元 醫師 Anti-IL5 Therapy for eosinophilic COPD 講師：莊立邦 醫師	臺灣阿斯特捷利康股份有限公司 Navigating the First-Line Challenge: Using Evidence-Led Strategies to Secure Durable Survival 講師：楊景堯 醫師 座長：施金元 醫師 Overcoming the Gap in Recurrence Risk with Adjuvant Therapy for Early-Stage EGFRm NSCLC 講師：莊政皓 醫師 座長：王金洲 醫師
18:00 21:00	**晚 宴**（萬享宴會廳 A）			

Jun 28 SUN

TIME	萬享宴會廳 B	萬享宴會廳 C	萬享宴會廳 D
08:30	**報 到** 【請持身份證刷到】		
09:00 | 09:40	鏈結國際視野 Connecting Horizons: TSPCCM Meets APSR 09:00-09:05 Opening Remarks Moderator: **Prof. Yuh-Min Chen** 09:05-09:35 A Paradigm Shift in COPD Management: Deciphering the Role of Type 2 Targeted Therapy in CoPD Speaker: **Prof. Atsuyasu Sato** Moderator: **Prof. Chul-Gyu Yoo** 09:35-09:45 Q & A	重症聯甄 (Critical Care Medicine) Resuscitation & Post–Cardiac Arrest Care: 2025 Guideline Updates 講師：**張維安** 醫師　座長：**陽光耀** 醫師	肺腫瘤 (Thoracic Oncology) Review of Small Cell Lung Cancer Treatment 講師：**江起陸** 醫師　座長：**王金洲** 醫師
09:40 | 10:20	09:45-10:15 Publishing in Respirology Speaker: **Prof. David CL Lam** Moderator: **Prof. Yoshinori Hasegawa** 10:15-10:25 Q & A	重症聯甄 (Critical Care Medicine) Hemodynamic Monitoring & Shock Management: From Guidelines to Precision Resuscitation 講師：**陳鍾岳** 醫師　座長：**林恒毅** 醫師	肺腫瘤 (Thoracic Oncology) Review of Pre/Post Operation Treatment in Lung Cancer 講師：**鄭文建** 醫師　座長：**黃明賢** 醫師
10:20 10:50	**Coffee Break**		
10:50 | 11:30	10:50–11:20 Emerging Advances in Lung Cancer Speaker: **Prof. Yung-Hung Luo** Moderator: **Prof. Yuh-Min Chen** 11:20–11:30 Panel Discussion & Q&A All Speakers/Moderators	重症聯甄 (Critical Care Medicine) Beyond Survival: Optimizing Patient-Centered Outcomes via the PADIS Framework 講師：**趙文震** 醫師　座長：**陳昌文** 醫師	肺腫瘤 (Thoracic Oncology) Review of Immunotherapy in Lung Cancer 講師：**蘇柏嵐** 醫師　座長：**施金元** 醫師
11:30 | 12:10		重症聯甄 (Critical Care Medicine) Automated closed-loop ventilation systems versus protocolized conventional ventilation in critically ill adults 講師：**郭耀文** 醫師　座長：**林孟志** 醫師	肺腫瘤 (Thoracic Oncology) Review of TKIs in Lung Cancer (Besides of EGFR & ALK) 講師：**吳寬澧** 醫師　座長：**楊政達** 醫師

	萬享宴會廳 D	萬享宴會廳 E
12:10 13:20	台灣百靈佳殷格翰股份有限公司 The Marathon of Targeted Therapy: Navigating Long-Term Care in the TKI Era 講師：**陳友木** 醫師　座長：**王金洲** 醫師 Precision Medicine in NSCLC: Navigating the Landscape of HER2 mutations 講師：**王馨儀** 醫師　座長：**何肇基** 醫師	臺灣阿斯特捷利康股份有限公司 三重優勢 共懸驅動！別讓肺傷心！ 講師：**陳家弘** 醫師　座長：**陽光耀** 醫師 TSLP in Mixed-Phenotype Severe Asthma: From Mechanism to Clinical Impact 講師：**蕭逸函** 醫師　座長：**魏裕峯** 醫師
13:20	**大 會 結 束**	

會場平面圖


晚宴
萬享宴會廳 A
萬享宴會廳 C
萬享宴會廳 B
萬享宴會廳 E
萬享宴會廳 D
BI
Roche
Chugai
秘書室
試片室
提袋領取處
行李寄放處
休息區
Shionogi
GSK
Sanofi
AZ
BeOne
BD
AZ
Moderna J&J
OEP
Lotus
休息區
拍照牆
報到處
節目表
KAOHSIUNG

交通資訊

高鐵（距離酒店約 4 公里）

- 從高鐵左營站搭乘捷運紅線至 R13 凹子底站，車程約 8 分鐘，從凹子底站 2 號出口步行約 500 公尺至高雄萬豪酒店（約 8 分鐘）
- 從高鐵左營站乘車至酒店約 15 分鐘

停車場

- B1~B2：機車停車場
- B3~B6：汽車停車場

捷運（距離酒店約 500 公尺）

- 紅線 R13 凹仔底站，從 2 號出口步行約 500 公尺至高雄萬豪酒店（約 8 分鐘）

高雄萬豪酒店接駁車

- 萬豪酒店→捷運凹子底站 (二號出口) →萬豪酒店
- 萬豪酒店發車：假日 13:00-19:30
- 循環接駁約每 30 分鐘一班，實際到站時間視車況調整

接駁車時刻表

高鐵左營站 ⟷ 高雄萬豪

預估乘車時間約 15 分鐘，實際時間可能因路況有所差異

Jun 27 SAT

高鐵左營站 發車時間				
11:00	11:30	12:00	12:30	13:00
13:30	14:00	14:30	15:00	15:30
16:00	16:30	17:00	17:30	18:00

高雄萬豪 發車時間				
13:45	14:15	14:45	15:15	15:45
16:15	16:45	17:15	17:45	18:15

晚宴（高雄萬豪 ⟶ 高鐵左營站）- 高雄萬豪 發車時間			
20:00	20:20	20:40	21:00

* 晚宴結束後接駁，滿員發車

Jun 28 SUN

高鐵左營站 發車時間				
07:30	08:00	08:30	09:00	09:30
10:00	10:30	11:00		

高雄萬豪 發車時間				
09:45	10:15	10:45	11:15	11:45
12:15	12:45	13:15	13:30	13:45

演講摘要

萬享宴會廳 B

Jun 27 SAT

13:20-14:00 P11 Obesity-Related Response to Biologics in Severe Asthma: How to Choose?
黃俊凱 醫師

14:00-14:40 P12 Biological Treatment in COPD: What Is Promising?
黃偉彰 醫師

15:10-15:50 P13 Comparative Effectiveness of Biologic Classes in Severe Asthma
吳秉宸 醫師

15:50-16:30 P14 COPD or Severe Asthma: from Clinical, Functional and Pathological Views
吳玟叡 醫師

16:30-17:40 P15 **台灣百靈佳殷格翰股份有限公司**

Balancing Efficacy and Safety: A Holistic Approach to Adjusting Inhaled Corticosteroid Intensity
黃俊凱 醫師

Acute Exacerbation in IPF
陳威志 醫師

Jun 28 SUN

09:00-09:40 P17 A Paradigm Shift in COPD Management: Deciphering the Role of Type 2 Targeted Therapy in COPD
Prof. Atsuyasu Sato

09:45-10:15 P18 Publishing in Respirology
Prof. David CL Lam

10:50–11:20 P19 Emerging Advances in Lung Cancer
Prof. Yung-Hung Luo

Jun 27 SAT

黃俊凱 醫師

台大醫學院 內科臨床講師
台大醫院 內科部胸腔科主治醫師

Obesity-Related Response to Biologics in Severe Asthma: How to Choose?

Obesity has emerged as a critical modifier of severe asthma, extending beyond a comorbidity to a distinct biological and clinical trait that reshapes disease expression. In obese individuals, the traditional paradigm of type 2 (T2) inflammation becomes less predictable. While some patients retain a T2-high profile with eosinophilic inflammation and elevated biomarkers, others exhibit attenuated T2 signals alongside features of non–T2 inflammation, including neutrophilic activity and metabolic-driven pathways. This heterogeneity reflects complex interactions between adipose tissue–derived mediators, systemic inflammation, and altered airway mechanics, all of which may influence symptom burden and treatment responsiveness.

Importantly, obesity may “mask” classical T2 signatures or create discordance between biomarkers and clinical severity, posing challenges for phenotype classification. In addition, mechanical factors such as reduced lung volumes and increased airway closure can amplify symptoms independent of airway inflammation, further complicating clinical assessment. These factors collectively suggest that conventional biomarker-driven approaches may be insufficient when applied to obese patients with severe asthma.

This presentation will explore how obesity modifies the immunologic landscape of asthma and examine the implications for biologic therapy selection. Rather than focusing solely on treatment outcomes, we will first revisit the underlying inflammatory phenotypes in obesity-associated asthma, highlight the limitations of current biomarkers, and discuss emerging concepts such as treatable traits and metabolic inflammation. The goal is to provide a conceptual framework that redefines how clinicians approach severe asthma in the context of obesity, setting the stage for a more nuanced and individualized therapeutic strategy.

Jun 27 SAT

黃偉彰 醫師

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Biological Treatment in COPD: What Is Promising?

Chronic obstructive pulmonary disease (COPD) remains a leading cause of morbidity and mortality worldwide, with a substantial proportion of patients continuing to experience exacerbations despite optimized inhaled therapies. This highlights a critical unmet need for more targeted and effective treatment strategies. Advances in the understanding of COPD heterogeneity and underlying inflammatory endotypes have opened new avenues for biological therapies, marking a potential shift toward precision medicine in COPD management.

This lecture will focus on emerging biological treatments that target key inflammatory pathways implicated in COPD. In particular, therapies directed against type 2 inflammation have shown promising results in selected patient populations. Monoclonal antibodies targeting interleukin-5 (IL-5) and its receptor (e.g., mepolizumab and benralizumab) have demonstrated modest reductions in exacerbation rates among patients with eosinophilic COPD. More recently, anti–IL-4/IL-13 therapy (dupilumab) has shown significant clinical benefits, including reductions in exacerbations and improvements in lung function, especially in patients with elevated blood eosinophil counts. In addition, upstream epithelial cytokine inhibitors, such as anti-thymic stromal lymphopoietin (TSLP) therapies, represent a promising strategy with potential efficacy across broader COPD phenotypes.

The presentation will critically review current clinical trial evidence, discuss the role of biomarkers—particularly blood eosinophils—in patient selection, and address safety considerations. Finally, it will explore key challenges in implementing biologics in COPD, including defining optimal candidates, cost-effectiveness, and the need for real-world evidence. Collectively, these advances signal a paradigm shift toward more personalized and mechanism-based treatment approaches in COPD.

Jun 27 SAT

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Comparative Effectiveness of Biologic Classes in Severe Asthma

Severe asthma continues to represent a major clinical and economic burden despite continuous advances in inhaled therapies. The landscape of severe asthma management has been revolutionized by targeted biologics addressing Type 2 (T2) and T2-low inflammation, including anti-IgE, anti-IL-5/5Ra, anti-IL-4/13, and upstream anti-TSLP therapies. However, with an ever-expanding therapeutic armamentarium in 2026, identifying the most clinically effective biologic for individual patients has become a critical and complex challenge for pulmonologists and allergists.

This presentation aims to evaluate the comparative effectiveness of distinct biologic classes in treating severe uncontrolled asthma. The discussion will focus on comparing their impact on primary clinical outcomes, including the reduction of annualized asthma exacerbation rates (AAER), improvement in forced expiratory volume in one second (FEV1), symptom control, and oral corticosteroid (OCS) sparing effects.

- Biomarker-Driven Efficacy: Analyzing how baseline biomarkers—specifically blood eosinophil counts, fractional exhaled nitric oxide (FeNO), and IgE levels—predict class-specific therapeutic responses.
- Clinical and Real-World Comparisons: Synthesizing data from recent network meta-analyses and real-world evidence (RWE) to contrast the efficacy of anti-IL-5/5Ra agents with anti-IL-4/13 agents in highly eosinophilic populations.
- Role of Upstream Targets: Evaluating the unique positioning of anti-TSLP therapies across a broader spectrum of phenotypes, particularly in patients with overlapping T2-low or complex inflammatory profiles.
- Impact of Comorbidities: Exploring how the presence of conditions like chronic rhinosinusitis with nasal polyps (CRSwNP) or atopic dermatitis shifts the comparative effectiveness and dictates class preference.

Current evidence strongly indicates that there is no universal "best" biologic; comparative effectiveness is intrinsically tied to the patient's specific endotype and clinical profile. As severe asthma care fully transitions into the era of precision medicine, utilizing composite biomarker profiles and considering concomitant T2 conditions are essential for optimizing initial biologic selection, maximizing therapeutic outcomes, and guiding effective drug-switching strategies.

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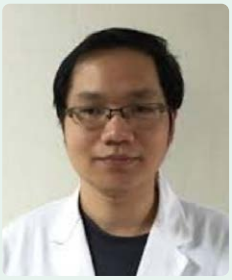

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COPD or Severe Asthma: from Clinical, Functional and Pathological Views

Although COPD and asthma remain the dominant clinical constructs used to interpret airflow obstruction, growing evidence indicates that similar functional abnormalities may reflect heterogeneous and overlapping structural and biological processes not adequately captured by traditional disease labels. Discordance between clinical manifestations, functional impairment, and structural or pathological abnormalities is increasingly recognized as a defining feature of obstructive airway disease, rather than merely diagnostic noise.

This presentation examines obstructive airway disease through three complementary domains: clinical traits, functional abnormalities, and structural/pathological alterations. We revisit the concept that airflow limitation, as reflected by spirometry, is central to disease definition but does not fully define the underlying biology. Similar functional abnormalities may arise from fundamentally different mechanisms. In asthma, airway remodeling is characterized by type 2 (T2)-associated inflammation, basement membrane thickening, smooth muscle hypertrophy, and distal airway dysfunction, often with preserved airway architecture. In contrast, COPD is characterized by small airway narrowing, terminal bronchiole loss, loss of alveolar attachments, and emphysema, reflecting a predominantly destructive process. Yet small airway disease exists in both diseases, raising the important question of how similar functional disturbances can reflect distinct structural substrates.

Emerging imaging and physiological tools offer new opportunities to interrogate these differences. Parametric response mapping (PRM), inspiratory–expiratory quantitative CT, oscillometry, and multiple-breath washout suggest that functional small airway disease may reflect structural small airway loss in COPD, whereas in asthma similar abnormalities may more often represent dynamic airway closure or peripheral dysfunction. Additional evidence from mucus plug imaging and hyperpolarized gas MRI further supports the concept that airflow obstruction may arise from different pathobiological processes not captured by spirometry alone.

The presentation also addresses the increasingly recognized coexistence of structural destructive disease and active T2 inflammatory biology. Recent evidence from eosinophilic COPD and biologic trials suggests that emphysema, airway loss, eosinophilic inflammation, mucus plugging, and exacerbation-prone phenotypes may coexist within the same patient. These observations challenge conventional disease labels and support a multidimensional framework in which overlapping biological programs—destructive, inflammatory, and remodeling processes—may coexist in spatially heterogeneous patterns within the lung.

Practical implications for clinical phenotyping using non-invasive tools, including blood eosinophils, FeNO, oscillometry, multiple-breath washout, and quantitative CT, support an integrated framework in which traditional diagnosis provides disease context while treatable traits guide precision therapy. The central thesis is that discordance between clinical, functional, and structural domains should not be viewed as contradiction, but rather as a window into disease heterogeneity and a path toward more biologically informed management of obstructive airway disease.

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Balancing Efficacy and Safety: A Holistic Approach to Adjusting Inhaled Corticosteroid Intensity

Inhaled corticosteroids (ICS) play an important role in COPD management, particularly in reducing exacerbations in selected patients. However, optimizing ICS intensity requires careful balancing between efficacy and safety, rather than reliance on a single biomarker such as blood eosinophil count. Although a threshold of ≥300 cells/μL is often used to predict better response, whether ICS therapy should be uniformly applied to all such patients remains open to discussion.

Escalation strategies are typically driven by persistent dyspnea and exacerbation (AE) risk, yet clinical trial populations—such as those in the IMPACT and ETHOS studies—often represent patients with more severe disease, higher prior ICS use, and greater bronchodilator reversibility. These characteristics may limit generalizability to broader COPD populations, particularly those with milder disease. Indeed, in patients with lower exacerbation risk or those classified as GOLD 1–2, the benefit of ICS remains uncertain.

Disease severity and exacerbation history are central to treatment decisions. Not all exacerbations are equivalent; their definitions, triggers, and responsiveness to anti-inflammatory therapy vary. This raises the question of whether more refined phenotyping is necessary before escalating ICS. At the same time, safety considerations are critical. ICS use is associated with risks such as pneumonia and other adverse effects, necessitating individualized benefit–risk evaluation, especially in vulnerable populations.

Blood eosinophils provide useful guidance but should be interpreted cautiously. A single elevated eosinophil value may not reflect a stable inflammatory phenotype. Patients with persistently elevated eosinophils may behave differently from those with transient increases, suggesting that longitudinal assessment is more informative than isolated measurements. Therefore, ICS responsiveness may vary even among patients with similar eosinophil counts.

De-escalation of ICS is another important but often overlooked aspect. In patients experiencing pneumonia or other significant side effects, reducing or withdrawing ICS should be considered. However, in those with higher eosinophil levels (>300 cells/μL), de-escalation may increase the risk of exacerbations, highlighting the need for careful monitoring and individualized decision-making.

In conclusion, adjusting ICS intensity in COPD requires a holistic approach that integrates exacerbation risk, disease severity, biomarker patterns, and safety considerations. Blood eosinophils are a valuable tool but should not be used in isolation. A nuanced, patient-centered strategy is essential to balance therapeutic benefit against potential harm.

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Acute Exacerbation in IPF

Acute Exacerbation in Idiopathic Pulmonary Fibrosis

Idiopathic pulmonary fibrosis (IPF) is a chronic, progressive fibrosing interstitial lung disease associated with substantial morbidity and mortality. Among its clinical complications, acute exacerbation of IPF (AE-IPF) represents a sudden and often unpredictable deterioration, characterized by rapidly worsening dyspnea and respiratory failure, and remains one of the leading causes of death in patients with IPF.

AE-IPF is currently defined as an acute, clinically significant respiratory worsening, usually within one month, accompanied by new bilateral opacities on high-resolution computed tomography, after exclusion of alternative causes such as infection, heart failure, or pulmonary embolism. Despite advances in diagnostic criteria and disease recognition, the pathogenesis of AE-IPF remains incompletely understood. Proposed mechanisms include acute lung epithelial injury, dysregulated inflammation, immune imbalance, coagulation abnormalities, and external triggers such as infection or environmental stress.

Clinically, AE-IPF is associated with poor short-term outcomes and high mortality. To date, no treatment has been conclusively proven to alter prognosis. Systemic corticosteroids remain the most commonly used therapy in clinical practice, although supporting evidence is limited and optimal dosing strategies are not well established. The use of additional immunosuppressive agents remains controversial, and some therapeutic combinations have been associated with adverse outcomes. In contrast, antifibrotic agents such as pirfenidone and nintedanib have demonstrated efficacy in reducing the incidence of acute exacerbations and may contribute to long-term disease stabilization.

This presentation will review the current definition and diagnostic approach to AE-IPF, summarize proposed pathophysiological mechanisms and risk factors, and discuss contemporary strategies for prevention and management. Emphasis will be placed on early recognition, exclusion of alternative diagnoses, comprehensive supportive care, and multidisciplinary decision-making, which are essential to optimize clinical outcomes in this life-threatening condition.

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鏈結國際視野 Connecting Horizons: TSPCCM Meets APSR

Prof. Atsuyasu Sato

Associate Professor, Department of Respiratory Medicine
Kyoto University Hospital

A Paradigm Shift in COPD Management: Deciphering the Role of Type 2 Targeted Therapy in COPD

Chronic obstructive pulmonary disease (COPD) has long been characterized as a predominantly neutrophil-driven condition, with treatment strategies largely focused on bronchodilation and exacerbation prevention. However, emerging evidence suggests that a subset of patients exhibits features of T2 inflammation, including elevated blood eosinophils and associated biomarkers. This evolving understanding has prompted increasing interest in the potential role of T2-targeted therapies—originally developed for asthma—in selected patients with COPD.

Recent clinical trials evaluating biologic agents targeting pathways such as IL-4/5/13 have demonstrated variable but promising results in reducing exacerbation rates and improving clinical outcomes in specific subpopulations. These findings highlight a potential paradigm shift toward a more personalized approach in COPD management, moving beyond traditional classifications based solely on airflow limitation.

Despite the growing enthusiasm, several challenges remain, including patient selection, biomarker validation, and cost-effectiveness considerations. A balanced interpretation of current evidence is essential as the field continues to define the place of T2-targeted therapies within COPD treatment algorithms.

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鏈結國際視野 Connecting Horizons: TSPCCM Meets APSR

Prof. David CL Lam

Clinical Associate Professor, Department of Medicine, The University of Hong Kong

Publishing in Respirology

Respirology is the official journal of the Asian Pacific Society of Respirology. It is the preferred English language journal of the Japanese Respiratory Society and the Taiwanese Society of Pulmonary and Critical Care Medicine, and an official journal of the World Association of Bronchology and Interventional Pulmonology.

The journal aims to publish peer-reviewed articles of scientific quality in clinical and clinically-relevant experimental respiratory biology and disease. Respirology encourages the submission of manuscripts focusing on clinical or laboratory research in areas relevant to the practice of respiratory medicine. The relevant topical fields are cell and molecular biology; epidemiology; immunology; pathology; pharmacology; physiology; intensive and critical care; paediatric respiratory medicine; interventional pulmonology and thoracic surgery are welcomed.

At the same time, we publish studies into the mechanism of disease, where the rationale for clinical relevance is clearly indicated. These should involve patient samples or experimental models of disease. Editorials, commentaries and reviews are generally commissioned by the Editors-in-Chief. Correspondences that provide relevant, meaningful comments on an article recently published in the journal. It is expected that authors of a Correspondence draw upon their experience in respiratory clinical care and research to raise relevant matters of genuine interest. Respirology also welcomes Clinical Practice Guidelines and Position Statements developed by a respiratory society.

Respirology follows ICMJE Recommendations and requires all clinical trials commenced after 1 July 2005 to be registered, prior to enrolment of the first participant, in any of the primary registries of the WHO International Clinical Trials Registry Platform or in ClinicalTrials.gov.

In this session, the peer review inside Respirology will be discussed. The review process involves invitation to experts in relevant subspecialty interests to evaluate the integrity and impact of new research as valuable additions to the medical literature, upholding fairness in making editorial decisions.

The journal is always on the outlook for eligible reviewers. Engagement of potential peer reviewers in editorial activities especially in early career will help cultivate expertise in academic publishing with the necessary skills, knowledge and research expertise to provide feedback to authors that is perceived as constructive criticism to improve the manuscript and write-up, and the potential impact when published. Participating in the peer review process is an important contribution that early career clinicians and researchers can make to an academic journal within their discipline. Yet the experience is equally valuable for their own professional development and long-term career in academic medicine.

In this session, experience and learnings on peer review activities will be shared, which range from expansion of professional network to public recognition of contributions to peer review within the field of Respirology, hopefully with further engagement of TSPCCM colleagues into academic publishing in Respirology.

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鏈結國際視野 Connecting Horizons: TSPCCM Meets APSR

Prof. Yung-Hung Luo

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Emerging Advances in Lung Cancer

Lung cancer is being redefined from a disease categorized primarily by anatomic stage and histologic subtype into a continuum of biologically distinct conditions. Recent studies have shown that the most important advances arise not from a single therapeutic class, but from earlier detection, more accurate staging, comprehensive molecular profiling, and the disciplined integration of local and systemic treatment. In early-stage disease, radiographic and pathologic features, including histologic invasiveness, together with molecular risk, are being used to refine postoperative surveillance and adjuvant decisions. In resectable stage II and III disease, perioperative therapy has moved from adjuvant chemotherapy toward biomarker-informed treatment. Recent studies have shown that neoadjuvant immune-checkpoint inhibition combined with platinum-based chemotherapy can increase major pathologic response and prolong survival outcomes in selected patients, whereas oncogene-driven tumors require a different strategy, with targeted agents now altering the natural history of resected EGFR-, ALK-, and RET-altered disease. For unresectable stage III NSCLC, concurrent chemoradiotherapy remains the foundation, but consolidation treatment is no longer uniform. PD-L1 blockade has established a durable role in patients without actionable oncogenic drivers, and recent evidence supports EGFR-directed consolidation after definitive chemoradiotherapy for EGFR-mutated tumors. In stage IV disease, treatment selection is increasingly guided by the results of upfront molecular testing. Tissue and plasma genotyping now guide first-line therapy for tumors with EGFR, ALK, ROS1, RET, MET exon 14, BRAF, NTRK, KRAS G12C, HER2, and EGFR exon 20 alterations. For tumors without dominant oncogenic drivers, combinations of immunotherapy, chemotherapy, and antiangiogenic therapy remain central. New antibody-drug conjugates, bispecific antibodies, and rational combinations are extending options after resistance and are narrowing the historical boundary between targeted therapy and immunotherapy. In small-cell lung cancer, progress remains more modest but meaningful, with immune maintenance, radiotherapy integration, DLL3-directed therapy, and improved brain surveillance reshaping care. Taken together, these developments underscore the importance of timely diagnostic and molecular testing, stage-specific therapeutic goals, precision therapy, and equitable implementation in contemporary lung cancer care.

萬享宴會廳 C

Jun 27 SAT

13:20-14:00 P21 Acute Exacerbation of Interstitial Lung Disease: Early Diagnosis and Treatment
林玠模 醫師

14:00-14:40 P22 Comorbidities in Idiopathic Pulmonary Fibrosis and Progressive Pulmonary Fibrosis
唐士恩 醫師

15:10-15:50 P23 Promises and Pitfalls: Respiratory Vaccines in Patients with Chronic Lung Diseases
鄭孟軒 醫師

15:50-16:30 P24 Less Is More? The Potential Role of Short-Course Tuberculosis Therapy in Taiwan
樹金忠 醫師

16:30-17:40 P25 **賽諾菲股份有限公司**

Precision Biologic Selection: Unlocking Higher Clinical Remission in Severe Asthma
Prof. Atsuyasu Sato

Dupilumab Across Respiratory Indications: Evidence from Clinical Trials and Real-World Practice
蕭逸函 醫師

Jun 28 SUN

09:00-09:40 P27 Resuscitation & Post–Cardiac Arrest Care: 2025 Guideline Updates
張維安 醫師

09:40-10:20 P28 Hemodynamic Monitoring & Shock Management: From Guidelines to Precision Resuscitation
陳鍾岳 醫師

10:50–11:30 P29 Beyond Survival: Optimizing Patient-Centered Outcomes via the PADIS Framework
趙文震 醫師

11:30–12:10 P30 Automated closed-loop ventilation systems versus protocolized conventional ventilation in critically ill adults
郭耀文 醫師

Jun 27 SAT

林玠模 醫師

嘉義長庚醫院 內科部 副部長
嘉義長庚醫院 肺感染及重症科主任

Acute Exacerbation of Interstitial Lung Disease: Early Diagnosis and Treatment

Acute exacerbation of interstitial lung disease (AE-ILD) is a catastrophic clinical event characterized by sudden respiratory deterioration, newly developed bilateral pulmonary infiltrates, and high short-term mortality. Despite advances in the management of chronic interstitial lung diseases, AE-ILD remains a major challenge in pulmonary and critical care medicine due to its heterogeneous etiologies, rapidly progressive course, and limited evidence-based therapeutic options.

Early recognition is crucial because delayed diagnosis often leads to irreversible diffuse alveolar damage and poor survival. Clinicians must promptly differentiate AE-ILD from common mimickers such as infection, cardiogenic pulmonary edema, pulmonary embolism, drug-induced pneumonitis, and procedural complications. A systematic diagnostic approach integrating clinical history, inflammatory biomarkers, arterial blood gas analysis, microbiologic assessment, and high-resolution computed tomography is essential to identify exacerbation at a potentially reversible stage.

Current management strategies are largely based on expert consensus and observational evidence. High-dose systemic corticosteroids remain the cornerstone of initial therapy, while broad-spectrum antimicrobial coverage is often empirically administered until occult infection is excluded. In selected patients, adjunctive immunomodulatory therapies, individualized ventilatory support, extracorporeal oxygenation, and rescue lung transplantation may be considered. Recent studies have further highlighted the importance of phenotype-oriented treatment, particularly in connective tissue disease-associated ILD and progressive fibrotic ILD.

This lecture will provide an updated and practical review of AE-ILD, focusing on early warning signs, critical diagnostic algorithms, differential diagnosis in the intensive care setting, and currently available therapeutic interventions. Through integration of recent literature and real-world critical care experience, this presentation aims to offer clinicians a bedside-oriented framework to improve timely decision-making and optimize patient outcomes in this highly fatal condition.

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國防部醫務組組長

Comorbidities in Idiopathic Pulmonary Fibrosis and Progressive Pulmonary Fibrosis

Idiopathic pulmonary fibrosis (IPF) is a chronic, progressive, and irreversible fibrosing interstitial lung disease associated with substantial morbidity and mortality. By contrast, progressive pulmonary fibrosis (PPF) is not a single disease diagnosis, but a progressive fibrotic phenotype that can occur across a spectrum of non-IPF fibrosing interstitial lung diseases. Although antifibrotic therapy has reshaped the treatment landscape, real-world outcomes remain strongly influenced by comorbidities.

Comorbidities in IPF and PPF are not merely background conditions; they modify symptom burden, confound the interpretation of disease progression, affect pulmonary function assessment, and worsen survival. High-impact comorbidities—including pulmonary hypertension, lung cancer, and combined pulmonary fibrosis and emphysema (CPFE)—are associated with poorer prognosis, while cardiometabolic and psychological factors substantially influence quality of life.

This lecture will review current evidence regarding the prevalence, prognostic relevance, and clinical implications of comorbidities in IPF and PPF. Special attention will be given to how comorbidities may mimic respiratory deterioration and why alternative explanations should be excluded before labeling disease worsening as PPF. A practical integrated care framework will be discussed, incorporating multidisciplinary diagnosis, structured comorbidity assessment, longitudinal monitoring of FVC, DLCO, and HRCT, individualized treatment, pulmonary rehabilitation, and palliative care.

Understanding IPF and PPF through a fibrosis-plus-comorbidity framework is essential to improving risk stratification, treatment decisions, and patient-centered outcomes.

Keywords

Idiopathic pulmonary fibrosis; Progressive pulmonary fibrosis; Comorbidities; Interstitial lung disease; Integrated care.

Jun 27 SAT

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萬享宴會廳C

Promises and Pitfalls: Respiratory Vaccines in Patients with Chronic Lung Diseases

Respiratory infections remain a major cause of morbidity and mortality in patients with chronic lung diseases. Structural airway abnormalities, impaired mucociliary clearance, and dysregulated immune responses contribute to increased susceptibility to infection and a higher risk of acute exacerbations. Vaccination is therefore a critical component of preventive care in this population.

This review focuses on the current evidence and clinical considerations regarding pneumococcal vaccines and respiratory syncytial virus (RSV) vaccines in patients with chronic lung diseases. Pneumococcal vaccination has been shown to reduce the incidence of invasive pneumococcal disease and may decrease the risk of pneumonia and disease exacerbations. The introduction of higher-valent conjugate vaccines has further expanded protective coverage and may offer improved immunogenicity in older adults and high-risk populations.

Recent advances in RSV vaccine development represent a significant milestone in adult respiratory medicine. Newly approved RSV vaccines targeting the prefusion F protein have demonstrated substantial efficacy in preventing lower respiratory tract disease in older adults. These findings are particularly relevant for patients with chronic lung diseases, who are at increased risk of severe RSV infection and its complications.

Despite these advances, several challenges remain. Vaccine effectiveness in patients with chronic lung diseases may be attenuated due to underlying immune dysfunction. Optimal vaccination strategies, including timing, sequencing, and potential co-administration with other vaccines, remain areas of ongoing investigation. In addition, long-term effectiveness, safety in specific subpopulations, and real-world impact on exacerbation prevention require further study.

In conclusion, pneumococcal and RSV vaccines play an increasingly important role in the management of patients with chronic lung diseases. Continued research is needed to refine vaccination strategies and maximize clinical benefit in this vulnerable population.

Jun 27 SAT

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Less Is More? The Potential Role of Short-Course Tuberculosis Therapy in Taiwan

Tuberculosis (TB) remains a significant public health challenge in Taiwan despite sustained control efforts and a gradual decline in incidence. Standard treatment for drug-susceptible TB typically requires at least six months of multidrug therapy, which, although effective, is associated with challenges including poor adherence, drug-related toxicity, and substantial healthcare resource utilization. These limitations have prompted increasing interest in shorter-course treatment regimens that could maintain efficacy while improving tolerability and programmatic feasibility.

Recent clinical trials have demonstrated that selected four-month regimens may achieve non-inferior outcomes compared to conventional six-month therapy in carefully selected populations. These findings raise the possibility of redefining TB treatment paradigms, particularly in settings with robust diagnostic and monitoring capacity.

In Taiwan, where healthcare infrastructure is well-developed and TB control programs are well-established, the implementation of short-course TB therapy presents both opportunities and challenges. On one hand, shortened regimens could improve treatment adherence, reduce adverse events, and decrease overall healthcare costs. On the other hand, concerns remain regarding patient selection, the risk of relapse, emergence of drug resistance, and applicability to populations with comorbidities such as diabetes or chronic lung disease, which are prevalent among TB patients in Taiwan.

This presentation will review current evidence supporting short-course TB therapy, discuss its potential applicability within the Taiwanese healthcare context, and highlight key considerations for clinical practice and policy implementation. Particular attention will be given to patient stratification, microbiological monitoring, and integration with existing TB control strategies.

Ultimately, while shorter (less) treatment durations may offer meaningful benefits, careful evaluation is essential to ensure that “more” treatment efficacy and long-term disease control are not compromised.

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Prof. Atsuyasu Sato

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Precision Biologic Selection: Unlocking Higher Clinical Remission in Severe Asthma

Precision selection of biologic therapies has become fundamental to achieving higher rates of clinical remission in severe asthma. Advances in the understanding of asthma heterogeneity highlight Type 2 inflammation as a predominant driver in a large proportion of patients with severe disease.

Dupilumab targets the shared IL-4/IL-13 pathway, a central driver of Type 2 inflammation, enabling broad disease control across multiple asthma phenotypes. By acting upstream, dupilumab reduces exacerbations, improves lung function, and enhances symptom control in patients with elevated eosinophils, increased FeNO, or Type 2–related comorbidities. Its mechanism-based approach allows effective treatment beyond single biomarker dependence, supporting a shift from symptom management toward sustained disease control and the potential achievement of clinical remission in severe asthma.

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Dupilumab Across Respiratory Indications: Evidence from Clinical Trials and Real-World Practice

Dupilumab has emerged as a foundational biologic therapy across multiple respiratory diseases driven by Type 2 inflammation. By blocking the shared IL-4 and IL-13 signaling pathway, dupilumab targets a central mechanism underlying airway inflammation, mucus hypersecretion, and tissue remodeling. Robust evidence from randomized clinical trials has demonstrated its efficacy in reducing exacerbations, improving lung function, and enhancing disease control in severe asthma, COPD as well as in chronic rhinosinusitis with nasal polyps (CRSwNP).

Importantly, findings from real-world studies complement clinical trial data, confirming sustained effectiveness and safety in broader, more heterogeneous patient populations. Real-world evidence highlights dupilumab's ability to deliver consistent benefits regardless of baseline biomarker variability, prior biologic exposure, or the presence of overlapping Type 2 comorbidities. These data reinforce its role as a versatile treatment option in routine clinical practice.

Jun 28 SUN

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Resuscitation & Post–Cardiac Arrest Care: 2025 Guideline Updates

The 2025 American Heart Association (AHA) Post-Cardiac Arrest Care (PCAC) guidelines emphasize systematic care to optimize patient outcomes. After the return of spontaneous circulation (ROSC), maintaining cerebral and organ perfusion is the primary goal. Regarding respiratory care, goal-directed strategies should be utilized to maintain SpO2 between 90% and 98% (or PaO2: 60–105 mmHg) and PCO2 between 35–45 mmHg to avoid secondary injury from hypoxemia or improper ventilation. Simultaneously, maintaining a mean arterial pressure (MAP) of at least 65 mmHg is essential to secure organ perfusion. For patients with suspected cardiac etiology, a 12-lead ECG should be obtained as soon as feasible, and for specific emergent conditions, emergency coronary angiography and intervention should be considered.

In terms of neurological care, for patients who remain unresponsive to verbal commands after ROSC, a protocolized temperature control strategy (32°C–37.5°C) is recommended and should be maintained for at least 36 hours. Regarding seizures or myoclonus, it is crucial to use EEG for accurate diagnosis; the use of antiseizure medications without an EEG correlate is not recommended.

Neuro-prognostication should adopt a "multimodal approach," and decisions must not be based on any single finding. It is recommended that prognostic impressions be consolidated at least 72 hours after normothermia and the discontinuation of sedatives, integrating results from clinical examinations, neuroimaging, electrophysiological tests, and serum biomarkers.

Finally, survivorship care must extend beyond saving lives. Before hospital discharge, patients and caregivers should receive structured emotional distress assessment and treatment, along with a multidisciplinary discharge plan. Efforts to address healthcare professional burnout should also be prioritized. For patients who cannot be resuscitated, organ donation should be considered as a critical part of systematic care and integrated into legal medical decision-making. Through the systematic implementation of these strategies, we aim to elevate post-cardiac arrest care to new heights and provide patients with better opportunities for recovery.

Jun 28 SUN

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Hemodynamic Monitoring & Shock Management: From Guidelines to Precision Resuscitation

The 2026 Surviving Sepsis Campaign guidelines reinforce hemodynamic monitoring and shock management as central components of sepsis care, emphasizing timely resuscitation, individualized perfusion assessment, and evidence-based vasoactive support. Initial fluid resuscitation continues to favor crystalloids, with balanced crystalloids preferred over normal saline for most patients. After early resuscitation, further fluid administration should be guided by dynamic measures of fluid responsiveness rather than static variables alone. Recommended tools include passive leg raising, stroke volume change, pulse pressure variation, and response to fluid challenge. Blood pressure monitoring remains essential throughout septic shock management. Noninvasive monitoring may be appropriate initially, but invasive arterial pressure monitoring is recommended when vasopressor requirements increase, multiple agents are required, frequent arterial sampling is needed, or cuff measurements are unreliable. Norepinephrine remains the first-line vasopressor for septic shock. Vasopressin is recommended as an adjunct when escalating norepinephrine doses are needed, while epinephrine may be added for persistent hypotension despite combined therapy. For patients with septic myocardial dysfunction and ongoing hypoperfusion despite adequate preload and arterial pressure, inotropic support such as dobutamine may be considered. Serial lactate measurement and capillary refill time are endorsed as complementary perfusion endpoints to guide resuscitation response. Importantly, the guideline highlights individualized treatment rather than fixed protocols, recognizing heterogeneity in shock physiology, comorbidities, and resource availability. Overall, SSC 2026 promotes a precision-based approach integrating monitoring, reassessment, and targeted intervention to optimize outcomes in septic shock.

Jun 28 SUN

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台中榮民總醫院 重症醫學部科主任

Beyond Survival: Optimizing Patient-Centered Outcomes via the PADIS Framework

The paradigm of critical care medicine has a profound transformation, shifting from short-term outcome to the long-term outcome. Despite of advancements in life-support technologies to improve acute survival rates of critically ill patients, survivors often encounter a functional impairments known as Post-Intensive Care Syndrome (PICS). PICS affects nearly 40-60% of intensive care unit (ICU) survivors, manifesting as cognitive decline, physical disability through ICU-acquired weakness (ICU-AW), and psychological sequelae such as anxiety and depression in both the patient and family. To address these challenges, PADIS framework is developed to cover Pain, Agitation/Sedation, Delirium, Immobility, and Sleep Disruption. This presentation will report the evolution of the aforementioned guideline in 2025, with the formal integration of anxiety as a distinct clinical domain. Furthermore, the 2025 update issues a conditional recommendation for dexmedetomidine over propofol when light sedation and delirium reduction are prioritized. Additionally, the administration of melatonin is now suggested to improve sleep quality and potentially mitigate delirium. To operationalize these recommendations, the ABCDEF bundle provides a structured framework for daily bedside care. Successful application of the PADIS framework requires a robust interprofessional collaboration involving physicians, nurses, pharmacists, and physical therapists, alongside family engagement to address PICS-Family (PICS-F). In conclusion, the high-quality recovery from critical illness requires a multidisciplinary collaboration in accordance with the PADIS framework.

Jun 28 SUN

郭耀文 醫師

臺大醫院 綜合診療部呼吸診療科科主任

Automated closed-loop ventilation systems versus protocolized conventional ventilation in critically ill adults

Mechanical ventilation requires repeated adjustment according to gas exchange, respiratory mechanics, patient effort, and disease severity change over time. Automated closed-loop ventilation systems attempt to make these adjustments more continuously by using real-time physiologic feedback to modify ventilator support. PAV+, ASV, INTELLiVENT-ASV, and SmartCare/PS use different principles, including proportional assistance based on patient effort, mechanics-based selection of breathing pattern, automated adjustment of ventilation and oxygenation using $EtCO_2$ and SpO_2, and automated reduction of pressure support during weaning.

Protocolized conventional ventilation remains clinician directed. It relies on structured bedside strategies such as low tidal volume ventilation, limitation of plateau pressure, monitoring of driving pressure, PEEP-FiO_2 tables, daily readiness assessment, and spontaneous breathing trials. Compared with manual protocols, closed-loop systems can provide more frequent adjustments and may reduce variation between clinicians and shifts. These advantages seem most relevant during weaning, where automated systems may shorten weaning duration or time on mechanical ventilation, while effects on mortality and ICU length of stay remain inconsistent.

For ARDS, the evidence is less settled. Closed-loop ventilation has been feasible and generally safe in selected patients, and may increase the proportion of time spent within predefined ventilation and oxygenation targets. Whether this translates into better clinical outcomes remains uncertain. Current systems still rely mainly on global signals and may miss regional lung heterogeneity, recruitability, overdistension, or complex patient–ventilator interaction. In practice, closed-loop ventilation is best viewed as an adjunct to protocolized clinician-guided care, useful for standardization and weaning efficiency, but not a substitute for individualized lung-protective decision-making in complex ARDS.

萬享宴會廳 D

Jun 27 SAT

13:20-14:00 P32 Advances in Mediastinal Tissue Acquisition in the Era of Interventional Pulmonology
黃彥翔 醫師

14:00-14:40 P33 Endobronchial Microwave Ablation- A Single Center Experience
施慧瑄 醫師

15:10-15:30 P34 Sleep-Related Breathing Disorders – CSA
廖培雅 醫師

15:30-15:50 P35 Sleep-Related Breathing Disorders – OSA
孫傳硯 醫師

15:50-16:10 P36 Sleep-Related Hypoventilation (SRH) Disorders: Pathophysiology and Clinical Subtypes
鍾心珮 醫師

16:10-16:30 P37 Sleep-Related Hypoxemia: Pathophysiology and Clinical Implications
鄭至宏 醫師

16:30-17:40 P38 **荷商葛蘭素史克藥廠股份有限公司台灣分公司**

GOLD Highlights: COPD Exacerbation and RSV related CV-risk
許健威 醫師

Early Optimization With Triple Therapy
蕭惠元 醫師

Anti-IL5 Therapy for eosinophilic COPD
莊立邦 醫師

Jun 28 SUN

09:00-09:40 P41 Review of Small Cell Lung Cancer Treatment
江起陸 醫師

09:40-10:20 P42 Review of Pre/Post Operation Treatment in Lung Cancer
鄭文建 醫師

10:50–11:30 P43 Review of Immunotherapy in Lung Cancer
蘇柏嵐 醫師

11:30–12:10 P44 Review of TKIs in Lung Cancer (Besides of EGFR & ALK)
吳寬澧 醫師

12:10-13:20 P45 **台灣百靈佳殷格翰股份有限公司**

The Marathon of Targeted Therapy: Navigating Long-Term Care in the TKI Era
陳友木 醫師

Precision Medicine in NSCLC: Navigating the Landscape of HER2 mutations
王馨儀 醫師

Jun 27 SAT

黃彥翔 醫師

台中榮民總醫院 胸腔部胸腔介入醫學科科主任

Advances in Mediastinal Tissue Acquisition in the Era of Interventional Pulmonology

萬享宴會廳 D

Advances in mediastinal tissue acquisition have significantly transformed the diagnostic and therapeutic landscape of thoracic diseases in the era of interventional pulmonology. Accurate sampling of mediastinal lesions is essential for the diagnosis, staging, and molecular characterization of lung cancer, as well as for the evaluation of benign conditions such as granulomatous and infectious diseases. Over the past decade, minimally invasive bronchoscopic techniques have largely replaced surgical approaches as the first-line modality.

This presentation will focus on the application and practical experience of endobronchial ultrasound-guided techniques, including EBUS-TBNA, EBUS-guided intranodal forceps biopsy (EBUS-IFB), and EBUS-guided cryobiopsy. These modalities provide complementary approaches to tissue acquisition, improving diagnostic yield and sample quality, particularly in cases requiring histologic architecture or extensive molecular testing. Technical considerations, procedural workflow, safety profiles, and clinical outcomes will be discussed, along with real-world experience from TCVGH.

Jun 27 SAT

施慧瑄 醫師

馬偕紀念醫院 胸腔內科主治醫師

Endobronchial Microwave Ablation- A Single Center Experience

Lung cancer has been the leading cause of cancer death in Taiwan for forty years. A large study published in 2011 by the U.S. National Lung Cancer Screening Trial found that low-dose computed tomography (CT) scans can reduce lung cancer mortality by 20%. The Health Promotion Administration has also been providing low-dose CT screenings for high-risk groups of lung cancer since July 1, 2022, which has significantly improved the detection rate of lung nodules. However, traditional surgical biopsies and excisions are invasive and often cause patients anxiety and prolong the recovery period.

We has introduced the "Archimedes Bronchoscope Navigation System" in MacKay Memorial Hospital. We applied the navigation system to integrate diagnosis, precise localization, and endobronchial microwave ablation of lung nodules in one stop, minimizing the burden on patients and increasing efficiency and safety.

Jun 27 SAT

廖培雅 醫師

臺中榮民總醫院 胸腔內科主治醫師

Sleep-Related Breathing Disorders – CSA

Central sleep apnea (CSA) accounts for approximately 5–10% of sleep-disordered breathing cases and is characterized by a cessation or marked reduction of airflow lasting at least 10 seconds in the absence of respiratory effort. Unlike obstructive events, in which airway collapse persists despite ongoing inspiratory effort, CSA reflects a transient loss of the neural drive to breathe. The underlying pathophysiology is complex and involves instability in ventilatory control, particularly fluctuations in arterial partial pressure of carbon dioxide ($PaCO_2$). When $PaCO_2$ falls below an individual's apneic threshold, central respiratory output from the brainstem is temporarily suppressed, leading to apnea. This instability is often amplified by heightened chemosensitivity, delayed circulatory feedback, and impaired integration of afferent and efferent neural pathways regulating diaphragmatic activity.

Several etiologies have been identified for CSA. Heart failure, especially with reduced ejection fraction, is one of the most common causes and is frequently associated with Cheyne–Stokes respiration, a cyclic pattern of crescendo–decrescendo breathing. Medications that depress central respiratory drive, such as opioids, can also precipitate CSA. In addition, treatment-emergent CSA may occur in patients with obstructive sleep apnea (OSA) who initiate continuous positive airway pressure (CPAP) therapy, reflecting a shift in ventilatory control dynamics once upper airway obstruction is alleviated. Other contributing conditions include cerebrovascular disease, high-altitude exposure, and certain neurological disorders.

Management of CSA remains challenging and requires a comprehensive, individualized approach. Treatment strategies should primarily focus on addressing underlying conditions, such as optimizing heart failure therapy or adjusting causative medications. Positive airway pressure modalities, including CPAP or adaptive servo-ventilation (ASV), may be considered in selected patients, although their use must be carefully evaluated, particularly in those with reduced cardiac function. Overall, a thorough understanding of the underlying mechanisms is essential to guide appropriate therapeutic decisions and improve clinical outcomes.

Jun 27 SAT

孫傳硯 醫師

臺北榮民總醫院 胸腔部主治醫師
台灣慢性阻塞性肺病學會 副秘書長

Sleep-Related Breathing Disorders – OSA

The International Classification of Sleep Disorders, Third Edition, Text Revision (ICSD-3-TR), published by the American Academy of Sleep Medicine (AASM) in 2023, represents an updated framework for the nosology and diagnostic criteria of sleep disorders. Building upon ICSD-3 (2014), the text revision incorporates evolving evidence across major diagnostic categories, including sleep-related breathing disorders, central disorders of hypersomnolence, circadian rhythm sleep-wake disorders, parasomnias, and sleep-related movement disorders.

Key updates in ICSD-3-TR include refined diagnostic criteria for obstructive sleep apnea (OSA), with greater emphasis on symptom burden and objective polysomnographic parameters beyond AHI thresholds alone. The revision also addresses emerging entities and clarifies the nosological boundaries between overlapping conditions, such as REM sleep behavior disorder and other parasomnias, and updates the classification of central sleep apnea subtypes.

Clinically, ICSD-3-TR provides a standardized diagnostic language essential for accurate patient phenotyping, guiding individualized treatment decisions, and ensuring consistency in research and epidemiological reporting. Its adoption facilitates alignment with ICD coding systems, supporting interdisciplinary communication across sleep medicine, neurology, pulmonology, and psychiatry.

This presentation uses a clinical case to illustrate the application of ICSD-3-TR diagnostic criteria for OSA within the sleep-related breathing disorders category, highlighting how updated classification criteria inform clinical reasoning and management.

Jun 27 SAT

鍾心珮 醫師

馬偕紀念醫院 胸腔內科主治醫師

Sleep-Related Hypoventilation (SRH) Disorders: Pathophysiology and Clinical Subtypes

Sleep-related hypoventilation (SRH) disorders comprise a range of respiratory conditions defined by insufficient ventilation during sleep, leading to abnormally elevated carbon dioxide levels. The physiological diagnosis of SRH is established when arterial or transcutaneous or end-tidal PCO2 is > 55 mmHg for > 10 minutes, or when PCO2 increases by > 10 mmHg relative to the awake supine baseline. While all SRH subtypes share these hallmark polysomnographic findings of elevated PCO2 and typically (though not essentially) accompany oxygen desaturation, Obesity Hypoventilation Syndrome (OHS) is characterized by the additional criteria of awake hypercapnia ($PaCO_2 \geq 45$ mmHg) and presence of obesity (BMI ≥ 30 kg/m²).

SRH disorders are categorized into different clinical subtypes based on underlying etiologies. OHS involves obesity-related mechanical changes, such as impaired chest wall compliance and increased airway resistance, alongside a blunted ventilatory response to hypercapnia. Congenital Central Alveolar Hypoventilation Syndrome (CCHS) is a disorder of central respiratory control, frequently caused by PHOX2B gene mutations, with hypoventilation typically most pronounced during NREM sleep. SRH due to medication or substance use is driven by the chronic intake of CNS depressants that suppress the respiratory drive. Finally, SRH due to a medical disorder encompasses conditions such as COPD and neuromuscular diseases, where the physiological atonia of respiratory muscles during REM sleep creates a mechanical disadvantage that exacerbates hypoventilation.

If left unrecognized, chronic hypercapnia and severe hypoxemia can lead to systemic complications, including pulmonary hypertension, cor pulmonale, polycythemia, cardiac arrhythmias, and neurocognitive dysfunction. Accurate subtyping of these disorders is essential to guide appropriate therapeutic interventions, such as continuous positive airway pressure (CPAP) or non-invasive ventilation (NIV), which are important for mitigating mortality and cardiovascular risks.

Jun 27 SAT

鄭至宏 醫師

高雄醫學大學附設醫院 內科部
胸腔暨重症醫學科 主治醫師

Sleep-Related Hypoxemia: Pathophysiology and Clinical Implications

Sleep-related hypoxemia disorder is defined by significant nocturnal oxygen desaturation not fully explained by other sleep-related breathing disorders. Diagnosis requires SpO_2 ≤88% for ≥5 minutes during sleep, typically identified by polysomnography or nocturnal oximetry. The underlying mechanisms are multifactorial, including ventilation-perfusion mismatch, diffusion impairment, shunt, hypoventilation, and reduced inspired oxygen. It commonly occurs in patients with pulmonary diseases such as COPD or interstitial lung disease. Clinically, hypoxemia may present as sustained or intermittent desaturation, with the latter suggesting coexisting sleep apnea. Chronic hypoxemia is associated with complications including pulmonary hypertension, cor pulmonale, and neurocognitive impairment. Differential diagnosis should exclude hypoventilation syndromes and other contributing conditions. Comprehensive evaluation, including CO monitoring, is essential to determine etiology. Further research is needed to define optimal treatment thresholds and the role of oxygen therapy.

Jun 27 SAT | 荷商葛蘭素史克藥廠股份有限公司台灣分公司

許健威 醫師

高雄榮總 重症醫學部主任
高雄榮總 胸腔內科兼任主治醫師

GOLD Highlights: COPD Exacerbation and RSV related CV-risk

According to the 2026 GOLD guideline, RSV vaccination is the only vaccine recommendation supported by **Evidence A** in COPD patients, with a higher level of evidence than influenza and pneumococcal vaccination recommendations. Emerging observational studies have shown that RSV infection may increase the risk of COPD exacerbation and acute cardiac events.

This course will highlight the growing clinical impact of RSV in COPD patients, including:

- Taiwan real-world data showed that nearly 60% of hospitalized COPD patients with RSV infection experienced acute exacerbation events.
- RSV accounted for **13.5% of respiratory viral infections last year**, highlighting its increasing disease burden.
- New real-world evidence for **AREXVY** has also demonstrated an association with reduced **RSV-related hospitalization** and **RSV-related COPD exacerbations** in COPD patients.

Together, these findings reinforce the importance of earlier RSV prevention in high-risk respiratory populations.

Jun 27 SAT | 荷商葛蘭素史克藥廠股份有限公司台灣分公司

蕭惠元 醫師

高雄榮總 胸腔內科主治醫師
高雄榮總 屏東分院胸腔內科主治醫師

Early Optimization With Triple Therapy

Based on the latest update in the 2026 GOLD Guideline, this session will re-emphasize the central role of acute exacerbations in COPD disease control.

In addition, the session will incorporate Real-World Evidence (RWE) and clinical case sharing to explore the practical value of Trelegy in routine clinical practice. Key areas of focus will include its benefits in:

1. Reducing the risk of acute exacerbations and mortality
2. Improving symptom control and lung function
3. Enhancing overall disease stability

Through a balanced integration of guideline insights and clinical evidence, this session aims to provide participants with a comprehensive understanding of the role of Trelegy in long-term COPD management, ultimately helping to optimize patient outcomes.

Jun 27 SAT | 荷商葛蘭素史克藥廠股份有限公司台灣分公司

莊立邦 醫師

林口長庚 肺感染及免疫科主任
林口長庚 胸腔內科副教授級主治醫師

Anti-IL5 Therapy for eosinophilic COPD

Eosinophilic inflammation has emerged as an important treatable trait in COPD, particularly among patients with frequent exacerbations despite optimized inhaled therapy. Increasing evidence suggests that IL-5 plays a central role not only in eosinophil activation and survival, but also in mucus hypersecretion, mucus plug formation, and airway remodeling, all of which contribute to disease progression and exacerbation risk.

This session will review the scientific rationale and latest clinical evidence supporting anti-IL5 therapy in eosinophilic COPD, including its impact on exacerbation reduction and long-term disease control. In addition, the speaker will share personal clinical experiences in identifying appropriate eosinophilic COPD patients and integrating biologic therapy into real-world practice. Particular focus will be placed on the potential role of IL-5 blockade in reducing mucus plugs and improving airway inflammation, offering new perspectives for precision treatment in COPD management.

Jun 28 SUN

江起陸 醫師

臺北榮民總醫院 胸腔部主治醫師

Review of Small Cell Lung Cancer Treatment

Small cell lung cancer (SCLC) is an aggressive malignancy with limited treatment options and poor prognosis. However, recent advancements in immunotherapy, targeted therapies, and antibody-drug conjugates (ADCs) are reshaping the treatment landscape and providing new hope for patients.

The integration of immune checkpoint inhibitors (ICIs) into first-line treatment has led to a significant improvement in survival for extensive-stage SCLC (ES-SCLC). The addition of atezolizumab or durvalumab to platinum-etoposide chemotherapy has become the standard of care, demonstrating a meaningful survival benefit. Durvalumab was also approved as a maintenance strategy following chemoradiotherapy in limited-stage SCLC (LS-SCLC) according to the ADRIATIC trial.

DLL3-targeting therapies have emerged as a significant advancement. Tarlatamab, a bispecific T-cell engager (TCE) targeting DLL3, has shown an objective response rate (ORR) of 40% and a median overall survival (OS) of 15.2 months in relapsed/refractory ES-SCLC. Ongoing studies are investigating its role in earlier treatment settings and in combination with PD-L1 inhibitors.

Antibody-drug conjugates (ADCs) are being explored as potential alternatives for chemotherapy-resistant SCLC. Trop2 and B7-H3 ADCs are currently under investigation for their ability to deliver targeted cytotoxicity while minimizing systemic toxicity. However, optimizing patient selection and managing treatment-related adverse events remain key challenges.

These recent advances mark a paradigm shift in SCLC management. Ongoing research is focused on identifying predictive biomarkers, refining treatment sequencing, and developing rational combination strategies to further improve patient outcomes. The evolving landscape of immunotherapy, TCEs, and ADCs highlights the potential for more effective, personalized treatment approaches for SCLC.

Jun 28 SUN

鄭文建 醫師

中國醫藥大學附設醫院 內科部胸腔科主治醫師

Review of Pre/Post Operation Treatment in Lung Cancer

The management of resectable non–small cell lung cancer (NSCLC) has evolved substantially with the integration of perioperative systemic therapies, including chemotherapy, immunotherapy, and targeted agents. Historically, platinum-based adjuvant chemotherapy has been the cornerstone of treatment, conferring a modest but significant overall survival (OS) benefit, particularly in stage II–III disease. However, the advent of precision oncology has reshaped perioperative strategies, emphasizing the importance of molecular profiling even in early-stage disease.

Recent evidence underscores the critical role of comprehensive molecular testing at diagnosis to identify actionable oncogenic drivers such as EGFR and ALK. Reflex next-generation sequencing is increasingly recommended to ensure timely therapeutic decision-making, although tissue limitations remain a challenge in early-stage disease. In EGFR-mutant NSCLC, the phase III ADAURA trial established adjuvant osimertinib as a new standard of care, demonstrating a profound improvement in disease-free survival (DFS) and a significant OS benefit. Similarly, the ALINA trial showed that adjuvant alectinib significantly prolongs DFS in resected ALK-positive NSCLC, further supporting genotype-directed perioperative therapy.

Neoadjuvant approaches have also gained momentum. Immune checkpoint inhibitors (ICIs), particularly when combined with chemotherapy, have demonstrated improved event-free survival (EFS) and pathologic complete response (pCR) rates in unselected populations. Landmark trials such as CheckMate-816, KEYNOTE-671, and AEGEAN have established perioperative chemo-immunotherapy as a new standard for resectable stage II–III NSCLC. However, patients with oncogene-driven tumors, particularly EGFR- or ALK-positive NSCLC, derive limited benefit from ICIs due to lower tumor mutational burden and distinct tumor biology. Moreover, concerns regarding increased toxicity—especially pneumonitis when ICIs are followed by tyrosine kinase inhibitors (TKIs)—limit their routine use in this subgroup .

The role of neoadjuvant targeted therapy remains investigational. Early-phase studies of EGFR TKIs have demonstrated modest response rates but have not consistently translated into durable long-term outcomes. NeoADAURA, are evaluating combined strategies incorporating TKIs with chemotherapy to optimize perioperative outcomes. Similarly, emerging data on KRAS, RET, and other rare oncogenic drivers suggest potential benefits of targeted therapies in the perioperative setting, although robust phase III evidence is still lacking.

Despite these advances, key challenges remain, including optimal sequencing and duration of perioperative therapies, the role of molecular residual disease (MRD), and the identification of predictive biomarkers. Balancing efficacy and toxicity is particularly important in curative-intent settings. In conclusion, perioperative management of NSCLC is shifting toward a biomarker-driven, personalized approach. While targeted therapies have improved outcomes in selected patients, the integration of immunotherapy and targeted strategies requires further validation. Ongoing trials will be critical to refine treatment paradigms and improve long-term survival.

Jun 28 SUN

蘇柏嵐 醫師

國立成功大學醫學院附設醫院 內科部胸腔內科主治醫師

Review of Immunotherapy in Lung Cancer

Lung cancer remains the leading cause of cancer-related mortality worldwide and continues to impose a substantial clinical and public health burden. Over the past decade, immunotherapy has fundamentally transformed the therapeutic landscape of lung cancer, shifting treatment goals from short-term disease control toward durable survival and long-term remission. Immune checkpoint inhibitors targeting the PD-1/PD-L1 axis, with or without CTLA-4 inhibition, have become integral components of treatment for both non-small cell lung cancer (NSCLC) and small cell lung cancer (SCLC). Their clinical application has expanded across multiple disease settings, including perioperative therapy for early-stage NSCLC, consolidation after chemoradiotherapy for locally advanced disease, and first-line or subsequent-line treatment for metastatic lung cancer. This presentation will provides a comprehensive overview of the current status and future directions of immunotherapy in lung cancer, as well as key mechanisms of resistance as well as evolving role of predictive biomarkers. Beyond checkpoint inhibition, next-generation immunotherapeutic approaches will also be reviewed, such as T-cell engagers, antibody–drug conjugate–immunotherapy combinations, cellular therapies, oncolytic viruses, and cancer vaccines.

Jun 28 SUN

吳寬澧 醫師

高雄醫學大學附設中和紀念醫院 胸腔內科主治醫師
高雄醫學大學後醫系 臨床助理教授

Review of TKIs in Lung Cancer (Besides of EGFR & ALK)

2026 represents a golden era of expansion in precision therapy for lung cancer. The current NCCN guidelines include a wide range of actionable oncogenic drivers beyond EGFR and ALK, such as ROS1, BRAF, KRAS G12C, MET exon 14 skipping, RET, HER2, NTRK, and NRG1, encompassing up to 60-70% of patients with lung adenocarcinoma.

Despite this progress, the development of tyrosine kinase inhibitors (TKIs) across these targets remains heterogeneous, with variable efficacy, central nervous system activity, and durability of response. While some drivers have achieved meaningful clinical outcomes, others are limited by modest benefit or rapid emergence of resistance.

This presentation will review the current landscape of TKIs in lung cancer beyond EGFR and ALK, focusing on kinase inhibitor development strategies, including structural design (type I vs. type II inhibitors, covalent vs. non-covalent binding) and optimization for CNS penetration. In addition, the limitations of a single-driver targeting paradigm will be discussed, highlighting the roles of tumor evolution, intratumoral heterogeneity, and the tumor microenvironment.

Finally, future directions will be explored, including combination strategies and integration with emerging modalities such as antibody–drug conjugates, protein degraders, and RNA-based therapeutics. These approaches aim to move beyond single-pathway inhibition toward multi-dimensional tumor control, with the goal of achieving more durable responses and, ultimately, curative outcomes.

Jun 28 SUN | 台灣百靈佳殷格翰股份有限公司

陳友木 醫師

鳳山醫院 胸腔內科主治醫師
高雄長庚醫院 胸腔內科主治醫師

The Marathon of Targeted Therapy: Navigating Long-Term Care in the TKI Era

Strategic management of EGFR-mutant NSCLC requires a forward-looking approach to the entire treatment journey. Supported by global and Asian real-world insights, establishing a robust clinical foundation early in the treatment sequence provides a versatile and reliable pathway for addressing the complexities of the disease in daily practice.

The focus remains on maximizing overall survival (OS) through a holistic optimization of the treatment continuum. By prioritizing a sustainable long-term trajectory, clinicians can effectively navigate the evolving demands of targeted therapy, ensuring a superior prognosis and lasting clinical benefits for patients across the duration of their care.

Jun 28 SUN | 台灣百靈佳殷格翰股份有限公司

王馨儀 醫師

台大醫院 綜合內科部主治醫師

Precision Medicine in NSCLC: Navigating the Landscape of HER2 mutations

Precision medicine has reshaped the treatment landscape of non-small cell lung cancer (NSCLC), with HER2 mutations emerging as a clinically relevant molecular subgroup. Although historically associated with limited treatment options, emerging clinical data on next-generation HER2 tyrosine kinase inhibitors (TKIs) demonstrate improved response and disease control in selected patients. Ongoing research focuses on optimizing patient selection and integrating HER2-targeted therapies into evolving treatment paradigms.

萬享宴會廳 E

Jun 27 SAT

12:00-13:10 P48 **友華生技醫藥股份有限公司**

Extrafine Triple Therapy: A New Approach to Asthma Control
陳彥甫 醫師

Targeting Small Airways with Extrafine ICS/LABA in Asthma
陳威志 醫師

13:20-14:00 P50 From 'Seeing' to 'Reaching': Practical Clinical Applications of CT Navigation and Lung Tumor Ablation
張凌愷 醫師

14:00-14:40 P51 From Clinical Data to High-Impact Publications: Translational Strategies for Building and Publishing ILD Cohort Studies
傅彬貴 醫師

15:10-15:50 P52 The Health Burden of Air Pollution in Taiwan
蘇一峰 醫師

15:50-16:30 P53 Work-Related Asthma
潘奕宏 醫師

16:30-17:40 P54 **臺灣阿斯特捷利康股份有限公司**

Navigating the First-Line Challenge: Using Evidence-Led Strategies to Secure Durable Survival
楊景堯 醫師

Overcoming the Gap in Recurrence Risk with Adjuvant Therapy for Early-Stage EGFRm NSCLC
莊政皓 醫師

Jun 28 SUN

12:10-13:20 P56 **臺灣阿斯特捷利康股份有限公司**

三重優勢 共懸驅動！別讓肺傷心！
陳家弘 醫師

TSLP in Mixed-Phenotype Severe Asthma: From Mechanism to Clinical Impact
蕭逸函 醫師

Jun 27 SAT | 友華生技醫藥股份有限公司

陳彥甫 醫師

臺大醫院雲林分院 胸腔科門診部主任

Extrafine Triple Therapy: A New Approach to Asthma Control

演講將聚焦於 extrafine 三合一吸入治療（ICS/LABA/LAMA）在氣喘控制中的新角色，特別強調其於「小呼吸道功能障礙（Small Airway Dysfunction, SAD）」中的臨床意義。小呼吸道直徑小於 2 毫米，長期以來因檢測不易而被低估，然而近年研究顯示，小呼吸道發炎與阻塞與氣喘症狀持續、控制不佳及急性惡化風險增加密切相關，已成為影響疾病進展的重要關鍵區域。

Extrafine 顆粒技術可使藥物更有效沉積於小氣道與肺周邊區域，相較於傳統吸入劑粒徑，有助於提升整體肺部藥物分布，提供更全面的抗發炎與支氣管擴張效果。透過臨床試驗（如 TRIMARAN 與 TRIGGER）數據，本演講將探討 extrafine triple therapy 在 ICS/LABA 控制不佳患者中的應用價值，包括肺功能改善、症狀控制提升及惡化風險降低。

綜合最新實證與臨床觀察，本演講旨在強調：氣喘治療不應僅侷限於大氣道，而應重新關注小呼吸道這一關鍵病灶。透過精準藥物遞送與治療升階策略，exrafine 三合一治療有望成為優化氣喘控制與改善長期預後的重要新方向。

Jun 27 SAT | 友華生技醫藥股份有限公司

陳威志 醫師

臺北榮民總醫院 胸腔部呼吸治療科主任

Targeting Small Airways with Extrafine ICS/LABA in Asthma

本演講將探討小呼吸道（small airways）在氣喘病理機轉中的關鍵角色，並聚焦於 extrafine ICS/LABA 治療策略於此病灶的臨床應用。小呼吸道直徑小於 2 毫米，過去因評估困難而常被忽視，然而越來越多證據顯示，小呼吸道發炎與氣流受限與氣喘症狀持續、控制不佳及急性惡化風險增加密切相關，是影響整體疾病控制的重要因子。

Extrafine 顆粒吸入製劑可更有效進入遠端小氣道，提升藥物在整個肺部的分布均勻性，進而強化抗發炎與支氣管擴張效果。相較於傳統吸入劑粒徑，exrafine ICS/LABA 不僅可改善肺功能指標，亦有助於提升症狀控制與生活品質。本演講將透過相關臨床研究與真實世界證據，說明其在氣喘治療中的臨床價值與定位。

綜合現有實證，本演講強調：精準治療小呼吸道已成為氣喘管理的重要趨勢。透過 extrafine ICS/LABA 的應用，有助於填補未被充分治療的病灶，進一步優化氣喘控制並降低未來惡化風險，為氣喘治療帶來更完整且個人化的策略。

Jun 27 SAT

張凌愷 醫師

臺大醫院 癌醫中心主治醫師

From 'Seeing' to 'Reaching': Practical Clinical Applications of CT Navigation and Lung Tumor Ablation

As cross-sectional imaging expands, the detection of indeterminate pulmonary tumors in oncology patients has surged. Accurately distinguishing benign processes, new primary cancers, and metastatic disease is imperative. While imaging provides clues, pathological confirmation remains the gold standard. For medically inoperable patients—due to cardiopulmonary limitations, age, or the need for lung preservation—image-guided thermal ablation offers a curative-intent, parenchyma-sparing alternative.

For suspected metastases, histological confirmation before or concurrent with ablation is strongly recommended to avoid overtreating benign lesions or misclassifying primary cancers, which alters staging and prognosis. A definitive biopsy enables multidisciplinary teams to tailor systemic therapies based on the tumor's molecular profile. To minimize patient discomfort and risks like pneumothorax, a "one-stop" approach using a coaxial cannula allows for synchronous core-needle biopsy and immediate thermal ablation during a single session.

Targeting small, indeterminate metastatic tumors is technically challenging but effectively addressed by Intra-Parenchymal Fine Adjustment (IPFA). IPFA enables interventionalists to fine-tune the needle's trajectory after entering the lung parenchyma, correcting minor deviations to perfectly center the probe under real-time guidance. This enhanced precision significantly increases the diagnostic biopsy yield for subcentimeter lesions and ensures the subsequent ablation zone encompasses the tumor with an adequate safety margin. Combining mandatory biopsy with precise IPFA targeting maximizes local tumor control and preserves healthy lung parenchyma, a critical benefit for inoperable patients who may require sequential interventions for future recurrences.

In this section, we present our institutional experience with image-guided lung ablation.

Jun 27 SAT

傅彬貴 醫師

臺中榮民總醫院 醫學研究部臨床試驗科主任

From Clinical Data to High-Impact Publications: Translational Strategies for Building and Publishing ILD Cohort Studies

This lecture provides a structured, translational roadmap for transforming real-world clinical data into high-impact publications in interstitial lung disease (ILD). Using fibrotic ILD, particularly idiopathic pulmonary fibrosis, as a model, we outline a “zero-to-one” strategy spanning cohort construction, analytical design, and publication positioning.

First, we discuss the establishment of high-quality prospective cohorts, emphasizing multidisciplinary diagnosis, standardized functional assessment, and longitudinal data capture to ensure both internal validity and external generalizability. Second, we present methodological approaches that move beyond descriptive analyses toward mechanism-informed and prognostically meaningful research. This includes integrating cardiopulmonary exercise metrics, functional performance tests, and patient-reported outcomes to develop composite and dynamic risk models. Advanced statistical frameworks, particularly time-dependent modeling, are highlighted as essential tools for capturing disease trajectories and improving predictive accuracy.

Third, we delineate key principles for translating clinical observations into publishable scientific evidence. These include refining research questions into testable hypotheses, aligning analytical strategies with journal expectations, and constructing a coherent narrative that bridges clinical relevance with biological plausibility. We also discuss how to strategically position real-world evidence alongside randomized trial data to enhance scientific impact.

Finally, we propose an integrated translational pipeline linking phenotyping, functional decline, treatment response, and mechanistic insights, enabling a continuous feedback loop between bedside observations and research innovation. This framework not only facilitates successful publication in high-impact journals but also advances precision medicine and clinical decision-making in fibrotic ILD.

Jun 27 SAT

蘇一峰 醫師

市立聯合陽明醫院 胸腔內科主治醫師
臺北榮民總醫院 胸腔部特約主治醫師

The Health Burden of Air Pollution in Taiwan

Air pollution is a major global health crisis, causing approximately 7–8 million premature deaths annually. It leads to severe respiratory and cardiovascular diseases—including strokes, lung cancer, and asthma—and impacts nearly every organ in the body.

Key Health Effects:

Respiratory Issues: Chronic obstructive pulmonary disease (COPD), asthma exacerbation, reduced lung function, and increased respiratory infections.

Cardiovascular Disease: Elevated risk of heart attacks, stroke, and ischaemic heart disease.

Cancer: Outdoor air pollution and particulate matter are classified as carcinogens, specifically causing lung cancer.

Other Health Impacts: Damages nervous and reproductive systems, causes cognitive impairment, dementia, diabetes, and adverse birth outcomes.

Air pollution (mainly $PM_{2.5}$) is a major environmental risk factor in Taiwan. It contributes significantly to premature death, chronic disease, and healthcare costs. Cardiovascular and respiratory diseases dominate the burden. Improvements are happening but the burden is still substantial and policy-relevant.

Jun 27 SAT

潘奕宏 醫師

安泰醫療社團法人安泰醫院 胸腔內科主治醫師

Work-Related Asthma

Work-related asthma (WRA) plays a pivotal role in adult respiratory health, accounting for approximately **25%** of all adult asthma cases. Unlike general asthma, the etiology of WRA is multifaceted, involving not only chemical irritants but also environmental factors such as temperature, humidity, and psychosocial workplace stress. A significant subset of WRA is **Occupational Asthma (OA)**, which represents roughly **16%** of adult asthma cases. The classical paradigm of OA focuses on airborne exposures and is characterized by the synergy between **IgE-mediated sensitization** and specific occupational triggers. Conversely, **Irritant-Induced Asthma (IIA)** represents a distinct clinical entity that occurs without the presence of IgE-related sensitization, often resulting from high-level acute or chronic irritant exposure. Recognizing the distinction between these phenotypes is essential for accurate diagnosis and effective workplace intervention. This presentation explores the transition from classical exposure theories to a more comprehensive understanding of work-related respiratory triggers.

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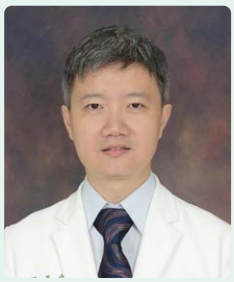

楊景堯 醫師

臺大醫院 胸腔科主治醫師

Navigating the First-Line Challenge: Using Evidence-Led Strategies to Secure Durable Survival

The treatment landscape of advanced non-small cell lung cancer (NSCLC) has evolved rapidly, with multiple first-line options available. However, selecting the optimal initial strategy remains a critical challenge, as first-line decisions have a profound impact on long-term outcomes and survival.

While targeted therapies and immunotherapy-based regimens have demonstrated significant efficacy, variability in patient characteristics, molecular profiles, and treatment responses necessitates a more evidence-driven approach. Achieving durable survival requires careful integration of clinical evidence with individualized treatment strategies.

In this presentation, I will review key clinical data guiding first-line treatment selection in NSCLC, focusing on strategies to maximize long-term benefit. Practical considerations, including patient selection and treatment sequencing, will also be discussed to support optimal decision-making in real-world practice.

In addition, emerging evidence and evolving treatment paradigms will be explored to better understand how to optimize outcomes across different patient subgroups. Particular attention will be given to balancing efficacy, safety, and treatment sustainability in clinical practice. By aligning evidence with personalized treatment approaches, clinicians can enhance long-term disease control and improve survival outcomes in advanced NSCLC.

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莊政皓 醫師

高雄醫學大學附設中和紀念醫院 胸腔內科檢查室主任
高雄醫學大學醫學院 後醫學系臨床助理教授

Overcoming the Gap in Recurrence Risk with Adjuvant Therapy for Early-Stage EGFRm NSCLC

Early-stage EGFR-mutated non-small cell lung cancer (EGFRm NSCLC) remains at substantial risk of recurrence despite complete resection. Although low-dose CT screening enables earlier diagnosis, a significant proportion of patients still relapse, often with distant metastases.

Adjuvant chemotherapy has shown limited survival benefit, particularly in stage IB disease, underscoring an unmet need for more effective postoperative strategies. The emergence of adjuvant EGFR-targeted therapies has reshaped the treatment paradigm by addressing residual disease that may persist after surgery.

This presentation will review the clinical challenges of recurrence in early-stage EGFRm NSCLC and the rationale for early systemic intervention. Key clinical evidence supporting adjuvant targeted therapy will be discussed, along with practical considerations for patient selection and integration into routine clinical practice.

In addition, the evolving role of early systemic intervention will be explored in the context of reducing recurrence risk and improving long-term outcomes. Emphasis will be placed on timely treatment initiation and consistent implementation of evidence-based strategies in clinical practice. By bridging the gap between surgical management and long-term disease control, adjuvant targeted therapy plays a central role in advancing outcomes for patients with early-stage EGFRm NSCLC.

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陳家弘 醫師

中國醫藥大學附設醫院 呼吸治療科主任

三重優勢 共懸驅動！別讓肺傷心！

Chronic obstructive pulmonary disease (COPD) remains a significant global health burden, with many patients continuing to experience persistent symptoms and exacerbations despite maintenance therapy. Timely treatment optimization is therefore essential to improve disease control and reduce future risk.

Triple therapy has become an important option for symptomatic and uncontrolled COPD patients requiring escalation beyond dual therapy. Budesonide/glycopyrronium/formoterol fumarate, a single-inhaler triple therapy combining ICS, LAMA, and LABA, has demonstrated clinical benefits across multiple studies. Evidence suggests that BGF can improve lung function, reduce exacerbation rates, relieve dyspnea, and enhance quality of life in patients with COPD.

This presentation will review the evolving role of triple therapy in COPD management, with a focus on identifying appropriate patients for earlier treatment optimization. Key clinical evidence and practical experiences will be discussed, including strategies to improve symptom control, reduce exacerbation burden, and support comprehensive disease management in routine clinical practice.

By addressing both current symptoms and future risks, BGF may provide an effective treatment approach for achieving better overall COPD control and improving patient outcomes.

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蕭逸函 醫師

臺北榮民總醫院 胸腔部一般胸腔科主治醫師

TSLP in Mixed-Phenotype Severe Asthma: From Mechanism to Clinical Impact

Severe asthma is a heterogeneous disease driven by multiple inflammatory pathways. Mixed-phenotype severe asthma, often characterized by overlapping eosinophilic inflammation, atopic features, elevated IgE, airway hyperresponsiveness, or variable type 2 signals, represents a high-unmet-need patient group. These patients frequently experience recurrent exacerbations, persistent symptoms, and incomplete response when treatment decisions rely only on a single biomarker such as eosinophils, IgE, or FeNO.

This lecture will explore the role of thymic stromal lymphopoietin (TSLP) as an upstream epithelial cytokine at the top of the inflammatory cascade. Triggered by allergens, viruses, pollutants, and airway injury, TSLP activates multiple downstream pathways, including eosinophilic and allergic inflammation. In mixed-phenotype patients, where multiple drivers coexist, targeting TSLP provides a strong scientific rationale by controlling inflammation closer to its source rather than blocking only one downstream pathway.

The session will connect TSLP biology with clinical decision-making and patient outcomes. It will discuss how upstream inhibition may simplify biologic selection for patients with overlapping or unclear biomarker profiles, support earlier biologic intervention, and address key physician priorities, including reducing exacerbations, emergency visits, hospitalizations, and oral corticosteroid burden.

Through a mechanism-to-impact framework, this course will help healthcare professionals better identify mixed-phenotype severe asthma patients and understand how TSLP-targeted therapy can reshape severe asthma management. The goal is to translate upstream science into practical clinical value and support more precise, earlier, and more comprehensive care for patients with severe asthma.

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處方資訊摘要 (詳細內容請參照衛生福利部核准之完整產品仿單)

[適應症] 無法切除或轉移性黑色素瘤 單一療法或併用ipilimumab適用於治療無法切除或轉移性黑色素瘤病人。 **黑色素瘤之輔助治療** 適用於黑色素瘤侵犯至淋巴結或已轉移且曾進行完全切除性手術之病人的輔助治療。**可切除之非小細胞肺癌的術前輔助治療** 併用含鉑化學治療適用於可切除（腫瘤≥4公分或淋巴結陽性）且不具EGFR或ALK腫瘤基因異常之非小細胞肺癌(NSCLC)成年病人的術前輔助治療。**非小細胞肺癌** • 併用ipilimumab適用於帶有PD-L1 (≥1%)且不具EGFR或ALK腫瘤基因異常的轉移性或復發性非小細胞肺癌(NSCLC)成年病人的第一線治療。 • 併用ipilimumab及2個週期含鉑化學治療適用於不具EGFR或ALK腫瘤基因異常的轉移性或復發性非小細胞肺癌(NSCLC)成年病人的第一線治療。•併用carboplatin、paclitaxel及bevacizumab適用於不具EGFR或ALK腫瘤基因異常的轉移性或復發性非鱗狀非小細胞肺癌(non-squamous NSCLC) 成年病人的第一線治療。• 適用於接受含鉑化學治療時或之後疾病惡化的晚期非小細胞肺癌(NSCLC)病人，病人若具有EGFR或ALK腫瘤基因異常者，則須經EGFR或ALK抑制劑治療後出現疾病惡化現象。 **惡性肋膜間皮瘤** 併用ipilimumab適用於無法切除之惡性肋膜間皮瘤成人病人的第一線治療。**腎細胞癌** • 併用ipilimumab可用於治療中度/重度風險(intermediate/poor-risk)先前未曾接受治療的晚期腎細胞癌病人。• 併用cabozantinib適用於未曾接受治療的晚期腎細胞癌病人。• 適用於先前經抗血管新生療法治療(anti-angiogenic therapy)的晚期腎細胞癌病人。**頭頸部鱗狀細胞癌** 適用於接受含鉑化學治療時或之後疾病惡化的復發或轉移性頭頸部鱗狀細胞癌(SCCHN)病人。**典型何杰金氏淋巴瘤** 適用於經下列方式治療後復發或惡化的典型何杰金氏淋巴瘤成人病人：• 接受自體造血幹細胞移植(HSCT)與brentuximab vedotin，或 • 接受3種或3種以上全身性療法，包括自體造血幹細胞移植(HSCT)。**泌尿道上皮癌** • 適用於治療接受含鉑療法期間或之後惡化的局部晚期無法切除或轉移性泌尿道上皮癌病人。• 適用於在接受根治性切除術後，有高度復發風險的泌尿道上皮癌病人的輔助治療。**胃癌、胃食道癌或食道腺癌** • 併用fluoropyrimidine及含鉑化學治療適用於治療晚期或轉移性，且不具有HER2過度表現的胃癌或胃食道癌或食道腺癌的病人。• 適用於治療先前經兩種或兩種以上化學治療的晚期或復發性胃癌或胃食道癌(Gastroesophageal Junction, GEJ)的病人。**肝細胞癌** 併用ipilimumab適用於先前經sorafenib治療的肝細胞癌(HCC)病人。 **轉移性大腸直腸癌** 單一藥物或併用ipilimumab適用於接受fluoropyrimidine、oxaliplatin和irinotecan治療後疾病惡化之具有高度微衛星不穩定性(MSI-H)或錯誤配對修復缺陷(dMMR)的轉移性大腸直腸癌(CRC)成人病人。**食道鱗狀細胞癌** • 併用含fluoropyrimidine及含鉑的化學治療適用於晚期或轉移性食道鱗狀細胞癌(ESCC)病人的第一線治療。•併用ipilimumab適用於晚期或轉移性食道鱗狀細胞癌(ESCC)病人的第一線治療。•適用於曾接受合併含鉑及fluoropyrimidine化學治療之後惡化的無法切除晚期或復發性食道鱗狀細胞癌(esophageal squamous cell carcinoma)病人。**食道癌或胃食道癌(Gastroesophageal Junction Cancer)切除的輔助治療** 適用於曾接受前導式同步化學與放射治療(neoadjuvant concurrent chemoradiotherapy)與手術完全切除，經病理檢查確認切除組織仍殘留腫瘤之食道癌或胃食道癌病人的輔助治療。**[用法] OPDIVO單一藥物的建議劑量** • 黑色素瘤、非小細胞肺癌、腎細胞癌、何杰金氏淋巴瘤、頭頸部癌、局部晚期或轉移性泌尿道上皮癌、胃癌、大腸直腸癌：3 mg/kg或240mg每2週一次*;或480mg每4周一次*，直到疾病惡化或出現無法接受之毒性為止。• 黑色素瘤輔助治療：3 mg/kg或240mg每2週一次*;或480mg每4周一次*，直到疾病復發或出現無法接受之毒性為止，治療至多為期1年• 泌尿道上皮癌的輔助治療：240 mg每2週一次*或480 mg每4週一次*，直到疾病復發或出現無法接受的毒性為止，至多為期1年。• 食道鱗狀細胞癌：240 mg每2週一次*或480 mg每4週一次*，直到疾病惡化或出現無法接受之毒性為止。• 食道癌或胃食道癌切除的輔助治療：240 mg每2週一次*或480 mg每4週一次*，完成16週治療後，接著以480 mg每4週給藥一次，直到疾病惡化或出現無法接受的毒性為止，總治療期間為1年。**OPDIVO併用其他治療藥物的建議劑量** • 黑色素瘤：每3週一次1 mg/kg*，隨後同一天給予ipilimumab 3 mg/kg*。併用ipilimumab最多4次劑量或出現無法接受之毒性為止，以較早發生者為準。後續OPDIVO單一療法的建議劑量為3 mg/kg或240mg，每2週一次*;或480mg每4周一次*，直到疾病惡化或出現無法接受之毒性為止。• 可切除之非小細胞肺癌的術前輔助治療: 360 mg每3週一次*並於同一天給予含鉑化學治療，每3週一次併用含鉑化學治療，共3個週期。• PD-L1(≥1%)非小細胞肺癌：3 mg/kg每2週一次*或360mg每3週一次*，隨後給予ipilimumab 1 mg/kg，每6週一次*。併用ipilimumab直到疾病惡化、出現無法接受的毒性為止，或若病人無疾病惡化則至多治療2年。• 非小細胞肺癌：360 mg每3週一次*隨後給予ipilimumab 1 mg/kg，每6週一次*並依組織學給予含鉑雙藥化學治療，每3週一次，共2個週期。併用ipilimumab直到疾病惡化、出現無法接受的毒性為止，或若病人無疾病惡化則至多治療2年。• 轉移性或復發性非鱗狀非小細胞肺癌：360 mg每3週一次*併用carboplatin、 paclitaxel及 bevacizumab，每3週一次。併用bevacizumab直到疾病惡化或出現無法接受的毒性為止。Carboplatin及paclitaxel至多治療6個週期。• 惡性肋膜間皮瘤：360 mg每3週一次*隨後給予ipilimumab 1 mg/kg，每6週一次*；或3 mg/kg每2週一次*隨後給予ipilimumab 1 mg/kg，每6週一次*。併用ipilimumab直到疾病惡化、出現無法接受的毒性為止，或若病人無疾病惡化則至多治療2年。• 腎細胞癌及大腸直腸癌：3 mg/kg，每3周一次*，隨後同一天給予ipilimumab 1 mg/kg每3週一次*，併用ipilimumab共4次劑量。後續OPDIVO單一療法的建議劑量為3 mg/kg或240mg每2週一次*或480mg每4週一次*，直到疾病惡化或出現無法接受之毒性為止。• 腎細胞癌: 240 mg每3週一次*，分併用cabozantinib 40 mg每日口服一次(且不得與食物併服)。OPDIVO治療直到疾病惡化、出現無法接受的毒性為止，或至多治療2年；Cabozantinib治療直到疾病惡化或出現無法接受的毒性為止。• 胃癌、胃食道癌或食道腺癌： 240 mg每2週一次*；或360 mg每3週一次*，隨後給予fluoropyrimidine及含鉑化學治療。直到疾病惡化、出現無法接受的毒性為止，或治療至多為期2年。• 食道鱗狀細胞癌: ‧240 mg每2週一次*或480 mg每4週一次*，併用含fluoropyrimidine及含鉑的化學治療。OPDIVO治療直到疾病惡化、出現無法接受的毒性為止，或至多治療2年。化學治療直到疾病惡化或出現無法接受的毒性為止。‧3 mg/kg每2週一次*或360 mg*每3週一次，隨後給予ipilimumab 1 mg/kg，每6週一次*。併用ipilimumab直到疾病惡化、出現無法接受的毒性為止，或至多治療2年。• 肝細胞癌: 1 mg/kg，每3周一次*，隨後同一天給予ipilimumab 3 mg/kg每3週一次*，併用ipilimumab共4次劑量。併用治療完成4次劑量後，後續OPDIVO單一療法的建議劑量為240mg每2週一次*，直到疾病惡化或出現無法接受的毒性為止。後續OPDIVO單一療法的建議劑量為240mg每2週一次*，直到疾病惡化或出現無法接受之毒性為止。 *連續靜脈輸注30分鐘。**[禁忌症]** 無相關禁忌症。**[警語及注意事項]** • 使用OPDIVO作為單一藥物或併用其他藥物治療有可能發生定義為需要以皮質類固醇治療且無明顯其他病因之免疫媒介性肺炎(曾通報死亡病例)、免疫媒介性結腸炎、免疫媒介性肝炎、免疫媒介性腎功能不全或腎炎(肌酸酐≥第2級升高)、免疫媒介性皮疹(包括SJS及TEN，曾有致死病例)、免疫媒介性腦炎、免疫媒介性內分泌病變如腦下垂體炎、腎上腺功能不全、自體免疫甲狀腺疾病及第1 型糖尿病。OPDIVO併用cabozantinib有可能導致肝毒性，其第3級和第4級ALT和AST增加的發生率高於單獨使用OPDIVO時。使用OPDIVO和cabozantinib合併療法時可能導致原發性或繼發性腎上腺功能不全。已有皮質類固醇抗藥性免疫媒介性結腸炎病人巨細胞病毒(Cytomegalovirus, CMV)感染/再活化的報告。• 應監測病人的肺炎、結腸炎、腦下垂體炎及腎上腺功能不全徵兆及症狀，甲狀腺功能及高血糖。應監測病人是否發生皮疹。開始OPDIVO併用cabozantinib治療之前及整個治療期間皆應定期監測肝臟酵素；相對於單獨使用OPDIVO，應考慮更頻繁地監測肝臟酵素。對肝臟酵素升高者，應中斷OPDIVO和cabozantinib治療，並視需要考慮使用皮質類固醇。視腎上腺功能不全疾病嚴重度，考慮暫停使用cabozantinib或OPDVIO任一藥物或兩種藥物。病人應於治療前及治療期間定期監測肝功能是否異常、是否有血清肌酸酐濃度上升。出現SJS或TEN之徵兆及症狀時，應暫時停用OPDIVO，並將病人轉介至專科進行評估及治療。應評估病人的神經學症狀。應密集追蹤病人移植相關併發症的早期症狀。有可能發生腫瘤出血，此與腫瘤消退/壞死有關。• **其他免疫媒介性不良反應** 在OPDIVO作為單一藥物或併用Ipilimumab治療的臨床試驗中，曾出現下列具臨床意義的免疫媒介性不良反應：心肌炎、橫紋肌溶解症、肌炎、葡萄膜炎、虹膜炎、胰臟炎、臉部及外展神經輕癱(paresis)、髓鞘脫失(demyelination)、風濕性多發性肌痛、自體免疫神經病變、急性多發性神經炎(Guillain-Barré syndrome)、腦下垂體功能低下症、全身發炎反應症候群、胃炎、十二指腸炎、類肉瘤病(sarcoidosis)、組織細胞壞死性淋巴炎(菊池氏病)(Kikuchi lymphadenitis)、運動功能障礙、血管炎、再生性不良貧血、心包膜炎、肌無力症、噬血球性淋巴組織球增生症(hemophagocytic lymphohistiocytosis)及自體免疫溶血性貧血。若觀察到類原田氏症(Vogt-Koyanagi-Harada-like syndrome)可能需要接受全身性類固醇治療以降低永久性視力喪失之風險。• **輸注反應** 接受OPDIVO治療病人其嚴重輸注反應的發生率小於1.0%。• **OPVIDO治療後之異體造血幹細胞移植(HSCT)併發症** 在PD-1受體阻斷抗體治療前或後接受異體造血幹細胞移植(HSCT)之病人曾發生致死或其他嚴重併發症。**[懷孕及胚胎毒性]** 應告知孕婦OPDIVO對胎兒的潛在風險。應告知具有生育能力的婦女在接受OPDIVO治療期間至OPDIVO最後一劑用藥後至少5個月內，需採取有效的避孕措施。**[腫瘤出血]** 有可能發生腫瘤出血，此與腫瘤消退/壞死有關。**[患有多發性骨髓瘤的病人在使用thalidomide衍生物和dexamethasone治療的過程中加入OPDIVO會使其死亡率上升]** PD-1或PD-L1抑制性抗體未核准於併用thalidomide衍生物和dexamethasone。**[授乳]** 目前尚不清楚OPDIVO是否會分泌至人體乳汁中。應告知婦女在OPDIVO治療期間停止哺乳。**[嚴重不良反應]** 試驗CA209037中為腹痛、低血鈉症、AST增加及脂肪酶增加。試驗CA209238中為腹瀉、澱粉酶增加和脂肪酶增加。試驗CA209067中為腹瀉、結腸炎及發熱。試驗CA209816中為肺炎(pneumonia)及嘔吐。試驗CA209227中為肺炎(pneumonia)、腹瀉/結腸炎、肺炎(pneumonitis)、肝炎、肺栓塞、腎上腺功能不全與腦下垂體炎。試驗CA2099LA中為肺炎(pneumonia)、腹瀉、嗜中性白血球減少合併發燒、貧血、急性腎損傷、肌肉骨骼疼痛、呼吸困難、肺炎(pneumonitis)與呼吸衰竭。試驗ONO-4538-52中為嗜中性白血球減少合併發燒、肺炎(pneumonia)、發燒、肺炎(pneumonia)、結腸炎、氣胸、腎上腺功能不全、嗜中性白血球數目減少、間質性肺病及斑丘疹。試驗CA209017及CA209057中為肺炎、肺栓塞、呼吸困難、發熱、肋膜積水、肺炎(pneumonitis)及呼吸衰竭。試驗CA209743中肺炎(pneumonia)、發燒、腹瀉、肺炎(pneumonitis)、肋膜積水、呼吸困難、急性腎臟損傷、輸注相關反應、肌肉骨骼疼痛和肺栓塞。試驗CA209025中急性腎臟損傷、肋膜積水、肺炎、腹瀉及高血鈣症。試驗CA2099ER中為腹瀉、肺炎(pneumonia)、肺發炎(pneumonitis)、肺栓塞、尿道感染和低鈉血症。有3名（0.9%）病人發生致命小腸穿孔。試驗CA209214中腹瀉、發燒、肺炎(pneumonia)、肺炎(pneumonitis)、腦下垂體炎、急性腎臟受損、呼吸困難、腎上腺功能不全及結腸炎。試驗CA209141中為肺炎、呼吸困難、呼吸衰竭、呼吸道感染及敗血症。試驗CA209205及CA209039中為肺炎(pneumonia)、輸注相關反應、發熱、結腸炎或腹瀉、肋膜積水、肺炎(pneumonitis)及皮疹。試驗CA209275中為泌尿道感染、敗血症、腹瀉、小腸阻塞與整體生理健康衰退。試驗CA209274中為泌尿道感染。試驗CA209649中為嘔吐、肺炎(pneumonia)、貧血、發熱、腹瀉、嗜中性白血球減少症合併發燒及肺炎(pneumonitis)。試驗CA209040中為發燒、貧血、腹瀉、AST增加、腎上腺功能不全、腹水、食道靜脈曲張出血、低血鈉症、血中膽紅素增加及肺炎(pneumonitis)。試驗CA209142中為結腸炎/腹瀉、肝臟事件、腹痛、急性腎臟損傷、發熱及脫水。試驗CA209648(OPDIVO併用化學治療)中為肺炎(pneumonia)、吞嚥困難、食道狹窄、急性腎臟損傷及發熱。試驗CA209648(OPDIVO併用Ipilimumab)中為肺炎(pneumonia)、發熱、非感染性肺炎(pneumonitis)、吸入性肺炎、吞嚥困難、肝功能異常、食慾降低、腎上腺功能不全及脫水。試驗CA209577中為肺發炎(pneumonitis)。**[不良反應]** 試驗CA209037中為皮疹。試驗CA209067中為疲倦、腹瀉、皮疹、噁心、發熱、搔癢、肌肉骨骼疼痛、嘔吐、食慾降低、咳嗽、頭痛、呼吸困難、上呼吸道感染、關節痛和轉胺酶增加。試驗CA209238中為疲倦、腹瀉、皮疹、肌肉骨骼疼痛、搔癢、頭痛、噁心、上呼吸道感染及腹痛。試驗CA209816發生率(>20%)的為噁心、便祕、疲倦、食慾減退及皮疹。試驗CA209227中為疲倦、食慾不振、肌肉骨骼疼痛、腹瀉/結腸炎、呼吸困難、咳嗽、肝炎、噁心和搔癢。試驗CA2099LA中為倦怠、肌肉骨骼疼痛、噁心、腹瀉、皮疹、食慾減退、便祕以及搔癢。試驗ONO-4538-52中為便祕、禿髮、周邊感覺神經病變、嗜中性白血球數目減少、皮疹、食慾減退、白血球數目減少、貧血、噁心、腹瀉、肌痛、發燒、高血壓、關節痛、全身無力、蛋白尿、血小板數目減少、搔癢、口腔炎及周邊神經病變。試驗CA209017及CA209057中為疲倦、肌肉骨頭疼痛、咳嗽、呼吸困難和食慾減退。試驗CA209078中為皮疹、疲倦、ALT增加、AST增加、搔癢、食慾降低、澱粉酶增加。試驗CA209743中為疲倦、肌肉骨骼疼痛、皮疹、腹瀉、呼吸困難、噁心、食慾降低、咳嗽和搔癢。試驗CA209025中為虛弱狀態、咳嗽、噁心、皮疹、呼吸困難、腹瀉、便秘、食慾降低、背痛及關節痛。試驗CA2099ER中為腹瀉、疲勞、肝毒性、肢端紅腫症、口腔炎、皮疹、高血壓、甲狀腺功能低下、肌肉骨骼疼痛、食慾下降、噁心、味覺障礙、腹痛、咳嗽和上呼吸道感染。試驗CA209214中為疲倦、皮疹、腹瀉、肌肉骨骼疼痛、搔癢、噁心、咳嗽、發燒、關節痛及食慾降低。試驗CA209141中為咳嗽、呼吸困難。試驗CA209205及CA209039中為上呼吸道感染、疲倦、咳嗽、腹瀉、發熱、肌肉骨骼疼痛、皮疹、噁心及搔癢。試驗CA209275中為疲倦、肌肉骨骼疼痛、噁心與食慾減退。試驗CA209274中為皮疹、疲倦、腹瀉、搔癢、肌肉骨骼疼痛及泌尿道感染。試驗CA209649中為周邊神經病變、噁心、疲倦、腹瀉、嘔吐、食慾降低、腹痛、便秘及肌肉骨骼疼痛。試驗ONO-4538-12中發生率≥5%的為搔癢、腹瀉、皮疹、及疲倦。試驗CA209142中為疲倦、腹瀉、發熱、肌肉骨骼疼痛、腹痛、搔癢、噁心、皮疹、食慾降低及嘔吐。試驗ONO-4538-24中發生率≥5%的為皮疹、腹瀉、甲狀腺功能低下、搔癢、食慾下降、發燒及疲倦。試驗CA209648(OPDIVO併用化學治療)中發生率≥20%的為噁心、食慾降低、便秘、口腔炎、疲倦、腹瀉及嘔吐。試驗CA209648(OPDIVO併用Ipilimumab)中發生率≥20%的為皮疹、發熱、噁心、腹瀉、疲倦及便秘。試驗CA209649中發生率≥20%的為疲倦、腹瀉、噁心、皮疹、肌肉骨骼疼痛及咳嗽。CA209040中發生率≥20%的為皮疹、搔癢、肌肉骨骼疼痛、腹瀉、咳嗽、食慾降低、疲倦、發燒、腹痛、頭痛、體重減輕、甲狀腺功能低下、頭暈及噁心。

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