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037
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AUG 2026

FEATURE STORY: A Silent Killer No More: How New Discoveries Are Changing Pancreatic Cancer Outcomes

Essential Phospholipids (EPLs): Liver-Supportive Agent in Real-World MASLD Management

How Vitamin B3 Stands Guard Over Your Eyes

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Repair What Is Damaged

TBI = traumatic brain injury. PSCI = poststroke vascular cognitive impairment.

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Dear Reader,

Welcome to the August 2026 issue of V.Pulse.

This month, our Feature Story explores the remarkable progress being made in pancreatic cancer care. Once regarded as one of the most challenging cancers to diagnose and treat, pancreatic cancer is now witnessing significant advances in early detection, precision medicine, immunotherapy, and targeted treatments. Groundbreaking developments, including promising KRAS-targeted therapies such as daraxonrasib, are offering new hope and reshaping expectations for patient outcomes.

In our Focus Section, we highlight World Leukaemia Day 2026, a global initiative dedicated to raising awareness, promoting early diagnosis, and supporting those affected by leukaemia. We examine the importance of recognising symptoms early, recent treatment advances, and the impact of awareness campaigns on patient care.

Our Industry Update reviews the evolving understanding of metabolic dysfunction-associated steatotic liver disease (MASLD) and discusses current management strategies, including the role of essential phospholipids in real-world clinical practice.

Meanwhile, the Epoch Section in this issue features a rich collection of infographics and medical updates, including emerging evidence on the potential role of Vitamin B3 in preventing open-angle glaucoma.

As always, our mission is to bring you concise, clinically relevant insights that bridge medical innovation and everyday practice.

We hope you enjoy this issue.

Warm regards,

Dr. Feng Xue
MPH, PhD
Managing Editor, V.Pulse

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A Silent Killer No More: How New Discoveries Are Changing Pancreatic Cancer Outcomes

For decades, pancreatic cancer has been one of the most feared diagnoses in medicine. Often called a “silent killer,” the disease typically develops with few symptoms and is frequently discovered only after it has reached an advanced stage. As a result, survival rates have historically lagged behind those of most other major cancers. However, a wave of scientific breakthroughs is beginning to change this narrative. Advances in imaging, biomarker discovery, genetic profiling, surgical techniques, immunotherapy, and precision medicine are creating new opportunities for earlier detection and more effective treatment. Most notably, the emergence of targeted therapies aimed at KRAS-driven tumours—long considered impossible to treat—has generated unprecedented optimism. Among these innovations, daraxonrasib has attracted worldwide attention after a major clinical trial demonstrated a near doubling of median overall survival in patients with previously treated metastatic pancreatic cancer. These developments suggest that pancreatic cancer may be entering a new era in which outcomes are steadily improving and personalised treatment becomes the standard of care. This article explores the scientific advances transforming pancreatic cancer care and examines what they could mean for patients in the years ahead.

Why Pancreatic Cancer Has Been So Deadly

Pancreatic cancer has long occupied a unique and devastating position among human cancers. Unlike breast, prostate, or colorectal cancers, which can often be detected early and treated successfully, pancreatic cancer frequently progresses unnoticed until it has already spread beyond the pancreas. Symptoms such as abdominal pain, unexplained weight loss, jaundice, or digestive disturbances often appear only after the disease has advanced.¹

The biology of pancreatic cancer compounds this challenge. Pancreatic tumours tend to grow aggressively, develop resistance to treatment, and create a dense surrounding tissue environment that prevents drugs from effectively reaching cancer cells.

Consequently, survival rates have remained stubbornly low despite decades of research.²

Historically, treatment options have been limited. Surgery offers the best chance of cure, but only a minority of patients are eligible at diagnosis because the disease has already spread or invaded nearby blood vessels.³ Chemotherapy has provided meaningful benefits for many patients, yet improvements in survival have generally been measured in months rather than years.⁴

Today, however, the landscape is changing. Scientists are no longer approaching pancreatic cancer with the same limited toolkit. Instead, they are attacking the disease from multiple angles simultaneously.



Why Earlier Detection Is Important

One of the greatest opportunities for improving pancreatic cancer outcomes lies in diagnosing the disease earlier. Researchers increasingly recognise that the poor prognosis associated with pancreatic cancer is not solely due to the aggressiveness of the disease itself. It is also a consequence of delayed diagnosis. Detecting tumours before they spread dramatically increases the likelihood of successful treatment.⁵

Several promising strategies are under investigation:

Blood-Based Biomarkers

Scientists are developing blood tests capable of detecting tiny traces of cancer-related molecules circulating in the bloodstream. These include circulating tumour DNA (ctDNA), exosomes, microRNA signatures, and protein biomarkers. Such approaches aim to identify pancreatic cancer before symptoms emerge, potentially enabling intervention at a much earlier stage.^{6,7,8,9}

Artificial Intelligence and Imaging

Artificial intelligence is being used to analyse medical images with remarkable precision. Machine learning algorithms can identify subtle abnormalities on computed tomography (CT) and magnetic resonance imaging (MRI) scans, and endoscopic ultrasound images that might otherwise go unnoticed. Researchers hope these technologies will eventually help physicians detect pancreatic tumours months—or even years—earlier than current methods allow.¹⁰

Screening High-Risk Populations

Although routine screening for the general population is not currently recommended, targeted screening programs are increasingly used for individuals with strong family histories of pancreatic cancer, inherited genetic mutations such as BRCA1 or BRCA2, certain hereditary cancer syndromes, and chronic pancreatitis. These programs are already demonstrating the value of early detection in high-risk groups.^{11,12}

● Precision Medicine Changes the Rules

A major shift in oncology over the last decade has been the move towards precision medicine—tailoring treatment according to the molecular characteristics of an individual patient's tumour. Pancreatic cancer was once considered relatively uniform. Today, researchers understand that it is genetically complex. Tumour sequencing can identify mutations and molecular pathways that may be vulnerable to targeted therapies. Important genetic alterations include KRAS, BRCA1, BRCA2, and PALB2 mutations, NTRK gene fusions, and mismatch repair deficiencies.^{13,14}

Identifying these alterations helps physicians select treatments more likely to benefit specific patients. For example, patients carrying BRCA-related mutations may respond particularly well to PARP inhibitors, drugs that exploit weaknesses in cancer-cell DNA repair mechanisms. The broader adoption of genomic testing is helping transform pancreatic cancer from a disease treated with one-size-fits-all therapies into one increasingly managed with personalised approaches.¹⁵

● Targeting KRAS: Cracking an "Undruggable" Target

Among all scientific advances in pancreatic cancer, few are more significant than the successful targeting of KRAS. KRAS is a gene that functions as a molecular switch controlling cell growth and division. In more than 90% of pancreatic cancers, KRAS is mutated, causing the switch to become permanently activated. The result is uncontrolled tumour growth. For decades, KRAS was considered virtually impossible to target with drugs. Scientists frequently described it as "undruggable" because the protein lacked obvious sites where medications could bind effectively.¹⁶

The breakthrough came through advances in structural biology, medicinal chemistry, and molecular engineering. Researchers developed innovative approaches capable of disrupting KRAS-driven signalling pathways, opening a therapeutic avenue once thought unattainable. The success of these efforts represents one of the most important achievements in modern cancer research.¹⁶

● Daraxonrasib: A Landmark Advance

The most exciting recent development in pancreatic cancer treatment involves a novel targeted therapy called daraxonrasib, which is an investigational oral medication designed to inhibit RAS signalling pathways that drive cancer growth. Unlike earlier approaches that targeted only specific KRAS mutations, daraxonrasib

employs a broader strategy capable of affecting multiple KRAS variants.¹⁷

In 2026, results from a large international Phase 3 clinical trial (RASolute 302) generated considerable excitement throughout the oncology community. The study involved 500 patients with previously treated metastatic pancreatic ductal adenocarcinoma. Investigators reported that daraxonrasib achieved a remarkable improvement in survival compared with standard chemotherapy.^{17,18}

Perhaps most strikingly, in the RAS G12 population, the median overall survival (mOS) increased from approximately 6.6 months with chemotherapy to 13.2 months with daraxonrasib—nearly doubling the survival time. The treatment reduced the risk of death by approximately 60% (**Figure 1**). The median progression-free survival (mPFS) was 7.3 months versus 3.5 months (hazard ratio [HR], 0.45). In the overall population, there was an mOS of 13.2 months versus 6.7 months (HR, 0.40) and an mPFS of 7.2 months versus 3.6 months (HR, 0.49). The overall response rate (ORR) in this group was 31.6% compared with 11.2% ($P < 0.0001$). Notably, the study saw similar benefits for mOS, mPFS, and ORR in both the RAS G12 and overall populations, suggesting that a RAS mutation may not be needed to derive benefit from daraxonrasib.¹⁸

For a disease where progress has often come in incremental steps, these results were viewed as potentially practice-changing. Experts believe evidence that targeting KRAS in pancreatic cancer is both feasible and effective.¹⁷ Patients receiving daraxonrasib additionally reported quality-of-life benefits and fewer treatment discontinuations compared with traditional chemotherapy despite predictable side effects such as rash, mouth sores, nausea, and diarrhoea.¹⁹

While regulatory review and long-term follow-up continue, daraxonrasib has emerged as one of the strongest signals yet that precision oncology may fundamentally alter outcomes in pancreatic cancer.

● Improvements in Surgery

Surgery remains the only potentially curative treatment for localised pancreatic cancer. Fortunately, surgical care has improved dramatically over the past two decades. Modern advances include:

- **Better Patient Selection:** Improved imaging allows physicians to evaluate tumours more accurately and identify patients most likely to benefit from surgery.²⁰

- **Neoadjuvant Therapy:** Increasingly, patients receive chemotherapy and sometimes radiation before surgery. This approach can shrink tumours, eliminate microscopic disease, improve surgical outcomes, and increase the likelihood of complete tumour removal.²¹
- **Enhanced Surgical Techniques:** High-volume pancreatic surgery centres have developed specialised expertise that significantly reduces complications and postoperative mortality.²²

🗨 The Expanding Role of Immunotherapy

Immunotherapy has revolutionised treatment for cancers such as melanoma and lung cancer. Unfortunately, pancreatic cancer has proven more resistant. The pancreatic tumour environment contains extensive immune suppression, making it difficult for immune cells to infiltrate and attack cancer effectively.²³

Nevertheless, researchers are making progress. Current investigations include cancer vaccines, checkpoint inhibitors, cellular therapies, tumour microenvironment modifiers, and combination treatment strategies. While immunotherapy has not yet transformed pancreatic cancer care to the same extent seen in some other cancers, many researchers believe combination approaches may unlock its potential in the future.²⁴

🗨 Understanding the Tumour Microenvironment

A unique feature of pancreatic cancer is its dense stromal tissue, often described as a protective fortress surrounding tumours. This environment

restricts drug delivery, suppresses immune responses, promotes treatment resistance, and supports cancer growth. Scientists increasingly focus on altering this tumour microenvironment to improve therapeutic effectiveness.²⁵

New drugs aim to break down the stromal barriers, improve blood supply to tumours, enhance immune-cell infiltration, and increase chemotherapy penetration. Combining these approaches with targeted therapies such as daraxonrasib may further improve outcomes in the coming years.^{26,27}

🗨 Advances in Chemotherapy Still Matter

Although targeted therapies receive much of the attention, conventional chemotherapy remains a cornerstone of pancreatic cancer treatment. Improved regimens such as FOLFIRINOX and gemcitabine plus nab-paclitaxel have significantly extended survival compared with older treatment approaches.²⁸

Researchers continue refining dosing schedules and combination strategies to maximise benefit while minimising toxicity. Importantly, emerging therapies are often expected to complement rather than completely replace chemotherapy, creating more powerful multimodal treatment plans.²⁹

🗨 The Rise of Personalised Cancer Care

Modern pancreatic cancer treatment increasingly recognises that every tumour is different. Future

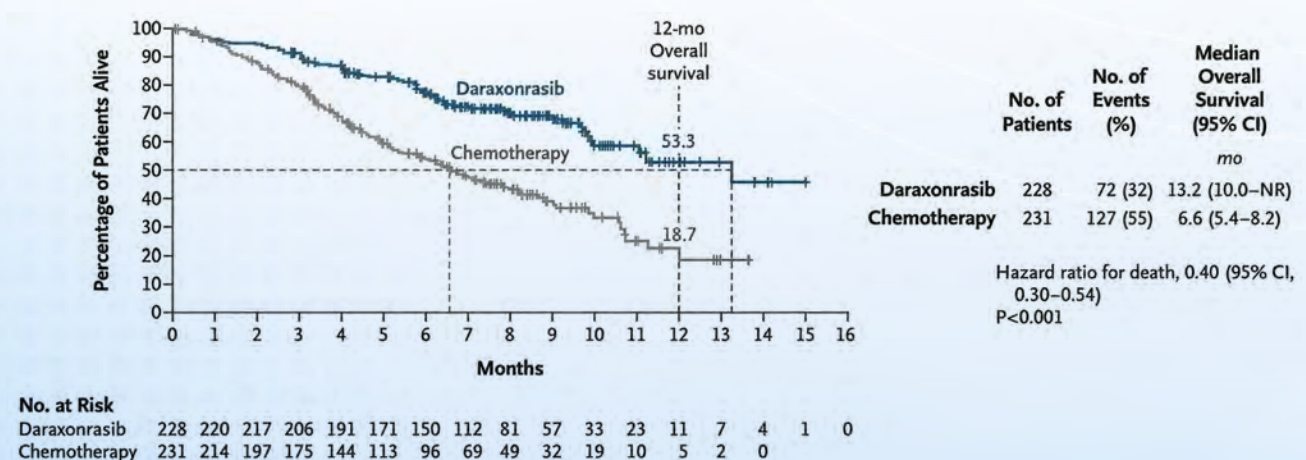


Figure 1. Overall Survival in the RAS G12 population¹⁸

treatment decisions may be guided by tumour genetics, blood-based biomarkers, imaging characteristics, immune profiles, and treatment-response monitoring.³⁰ Technologies such as liquid biopsies may allow physicians to track tumour evolution in real time and adjust therapies as resistance develops. This personalised approach has the potential to improve outcomes while reducing unnecessary side effects.³¹

Reasons for Optimism

For many years, news about pancreatic cancer was dominated by discouraging statistics. Today, there are genuine reasons for optimism. Researchers have finally achieved breakthroughs in several areas simultaneously:

- Earlier detection technologies are advancing rapidly⁵⁻¹²
- Genetic testing is guiding individualised treatment decisions¹³⁻¹⁵
- Novel targeted therapies are producing survival gains previously considered unattainable¹⁸
- Surgical outcomes continue to improve²⁰⁻²²
- Immunotherapy research is accelerating^{23,24}
- Understanding of tumour biology has deepened significantly²⁵⁻²⁷

The success of daraxonrasib is particularly important because it demonstrates that a major driver of pancreatic cancer can be targeted effectively. What was once considered an impossible therapeutic challenge is now producing real clinical benefits for patients.¹⁷⁻¹⁹

Conclusion

Pancreatic cancer has earned its reputation as a silent killer through decades of late diagnoses, aggressive disease progression, and limited treatment options. Yet the outlook is changing. Scientific discoveries are steadily reshaping how physicians detect, understand, and treat this formidable cancer.

Among the most significant advances is the emergence of precision medicine, especially therapies targeting KRAS mutations that drive most pancreatic tumours. Daraxonrasib has become a landmark example of this progress, demonstrating a near doubling of median overall survival in patients with previously treated metastatic pancreatic cancer and providing compelling evidence that even long-standing “undruggable” targets can be successfully attacked.

While pancreatic cancer remains a serious and often life-threatening disease, patients and clinicians now face a future filled with far more promise than in previous decades. Earlier detection, molecularly guided treatment, innovative drug development, and ongoing research into tumour biology are converging to create meaningful improvements in survival and quality of life. The era in which pancreatic cancer was viewed as nearly untreatable may finally be coming to an end.



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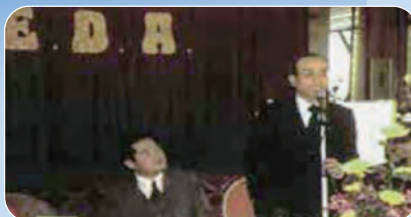
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Essential Phospholipids (EPLs): Liver-Supportive Agent in Real-World MASLD Management



Dr. Jose D Sollano Jr, MD

*Professor of Medicine
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Non-alcoholic fatty liver disease has been reframed in recent years as metabolic dysfunction-associated steatotic liver disease (MASLD) to emphasize its origin in cardio-metabolic dysregulation¹. MASLD affects approximately 25% of the global population, with an even higher prevalence rate of 27% in Asia². Importantly, MASLD is not just a liver problem—it represents a manifestation of the broader metabolic syndrome and carries significant cardiometabolic consequences³. At a recent symposium, Dr. Sollano reviewed the evolving therapeutic landscape in his lecture and highlighted the role of essential phospholipids (EPLs)—especially phosphatidylcholine—as a useful liver-supportive agent in real-world MASLD management, particularly where access to newer treatments remains constrained.

Pathobiology and Rationale for EPLs

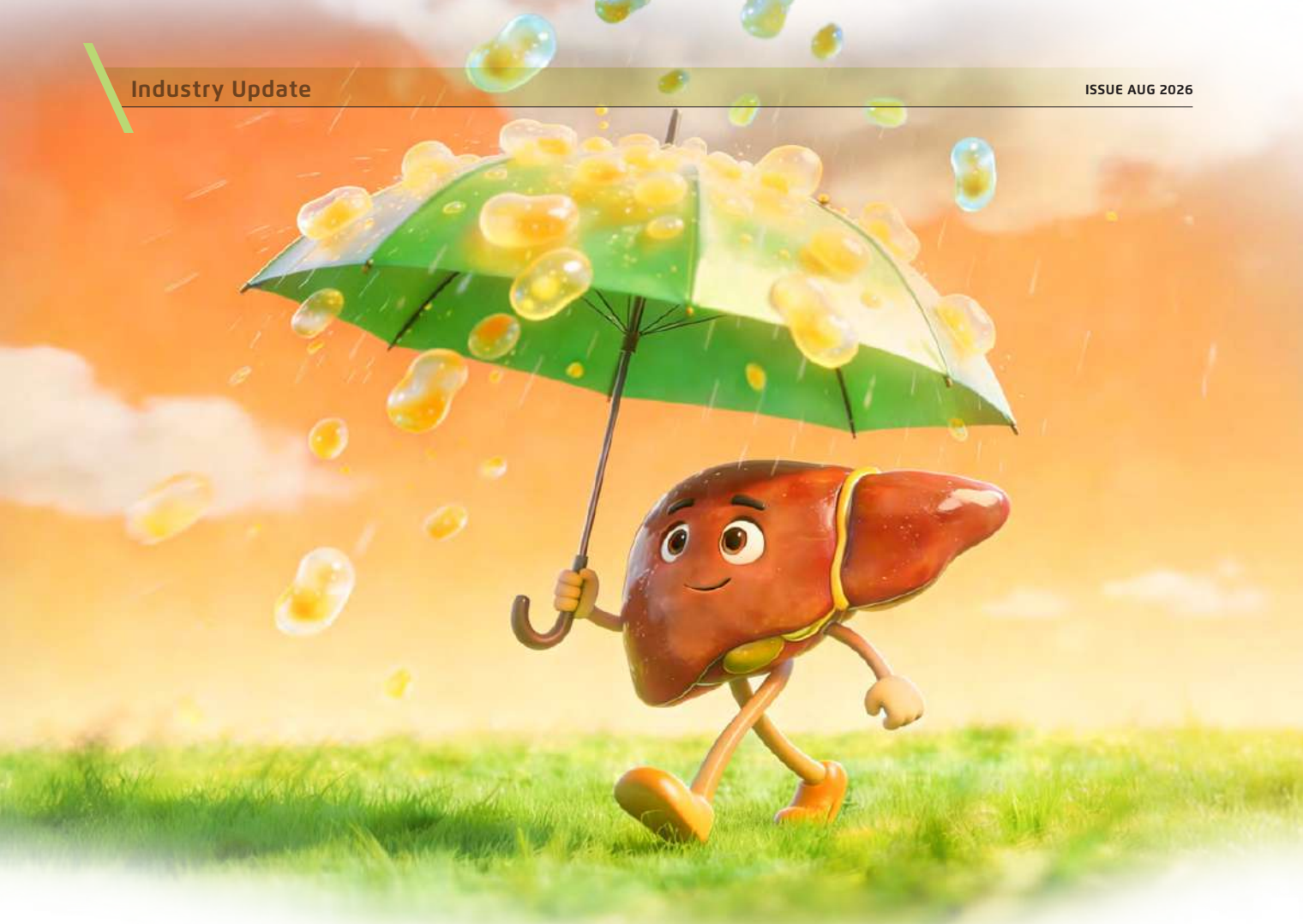
In the pathogenesis of MASLD, cellular architecture and organelle function of hepatocytes become altered as lipids accumulate, leading to elevated activation of inflammatory pathways and lipotoxic endoplasmic reticulum (ER) stress. These processes contribute to progressive hepatocellular dysfunction and death, promote tissue damage, and drive the progression of fibrosis⁴. Lipid droplets may therefore present as a potential therapeutic target in MASLD.

EPLs act at multiple points in this cascade principally by integrating into hepatocellular membranes. This helps in restoring membrane

structure and fluidity, attenuating lipid peroxidation, and facilitating lipid handling by enhancing membrane-associated metabolic functions⁵. EPLs enhance β -oxidation by serving as a supportive adjunct when combined with peroxisome proliferator-activated receptor (PPAR) agonists to reduce the size and number of intra-hepatocellular lipid droplets⁶. Recent *in vitro* evidence also showed that the application of EPLs to steatotic hepatocytes mitigates inflammation and reduced the size of accumulating lipid droplets in a dose-dependent manner⁷.

Clinical Evidence for EPLs in MASLD

Clinical research into EPLs span randomized



trials, observational cohorts, and meta-analyses. While trial design may vary, EPLs' efficacy was demonstrated in a meta-analysis, where relieved steatosis, decreased lipid profiles, and improvements in liver enzymes (alanine transaminase [ALT]/aspartate aminotransferase [AST]) were observed⁸.

According to a randomized trial which evaluated high-dose EPLs (1800 mg/day for 24 weeks followed by maintenance at 900 mg/day) in MASLD patients with or without type 2 diabetes, significant reduction in ALT and AST was observed in both patients with MASLD alone and those with comorbid diabetes (**Figure 1**)⁹. Ultrasonography findings after treatment completion showed a

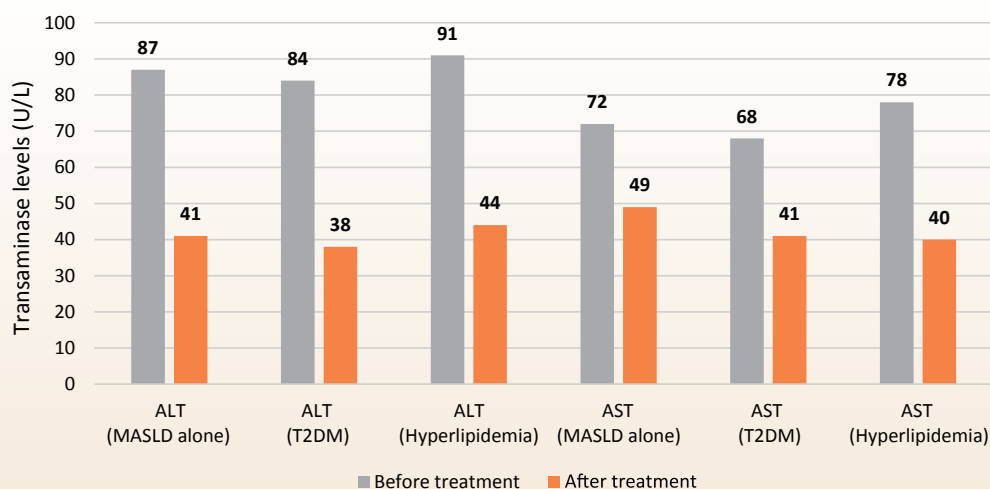


Figure 1: Mean ALT and AST values before and after EPLs treatment⁹. ALT, alanine transaminase; AST, aspartate aminotransferase; EPLs, essential phospholipids; MASLD, metabolic dysfunction-associated steatotic liver disease; T2DM, type 2 diabetes mellitus

noticeable reduction in the number of patients with grade II fatty infiltration, an increased number of patients with grade I infiltration and normal post-treatment ultrasonography. A reduction of liver stiffness as a measure of fibrosis was also observed by elastography in all treated cohorts⁹.

A more recent meta-analysis of 10 studies encompassing nearly 22,000 patients further reinforced the therapeutic role of EPLs by demonstrating benefit in individuals with fatty liver, diabetes, and obesity¹⁰. When used alongside antidiabetic therapy, EPLs not only reduced triglycerides and cholesterol but also improved liver ultrasonography findings and produced meaningful declines in ALT and AST.

Taken together, evidence supports a role for EPLs to act as adjunctive hepatoprotective therapy.

ALT as a Critical Biomarker in MASLD for Assessment and Management

Dr. Sollano then raised the critical question of whether ALT serves as a sufficiently reliable biomarker for assessing treatment efficacy in real-world practice.

While some older expert opinions may suggest limited reliance on ALT alone for diagnosis, a meta-analysis of 11 studies covering over 4000 patients showed a high rate in the prevalence of elevated ALT amongst patients¹¹. Of note, 75% of patients with MASLD and up to 80% of patients with nonalcoholic steatohepatitis (NASH) had high ALT levels in the reviewed publications. Dr. Sollano therefore suggested that ALT should be viewed as a surrogate marker for patients with MASLD.

Evidence from major trials, including those investigating efficacy of resmetirom and semaglutide for the treatment of MASLD, also consistently tracked and showed marked reductions in liver enzyme levels among treatment responders^{12,13}.

Therefore, in clinical practice, ALT improvements can be utilized to communicate to patients that their liver is showing a degree of improvement in transamination following therapy, whether through lifestyle changes, comorbidity management, or specific liver treatments. To provide a more comprehensive assessment of

improvement in steatosis and fibrosis, Dr. Sollano recommended the use of additional non-invasive liver disease assessment parameters, such as transient elastography and controlled attenuation parameters (CAP), which have become increasingly accessible in the clinical settings in assessing the degree of liver fibrosis and steatosis.

Recent Studies Assessing EPLs' Benefits

To conclude his presentation, Dr. Sollano highlighted several recent clinical studies that further strengthen the evidence base for EPLs, beginning with the RELIEF study, a large real-world study from China enrolling several thousand patients with MASLD (Please refer to the next page for study details)¹⁴.

Beyond the RELIEF study, an Eastern European trial comparing EPLs with another hepatoprotective agent reported greater reductions in ALT and AST, along with more pronounced improvements in FibroScan-assessed steatosis and fibrosis among EPLs-treated patients¹⁵. Additional emerging data from other trials also demonstrated improvements in patient-reported quality-of-life measures, underscoring EPLs' potential benefits¹⁶.

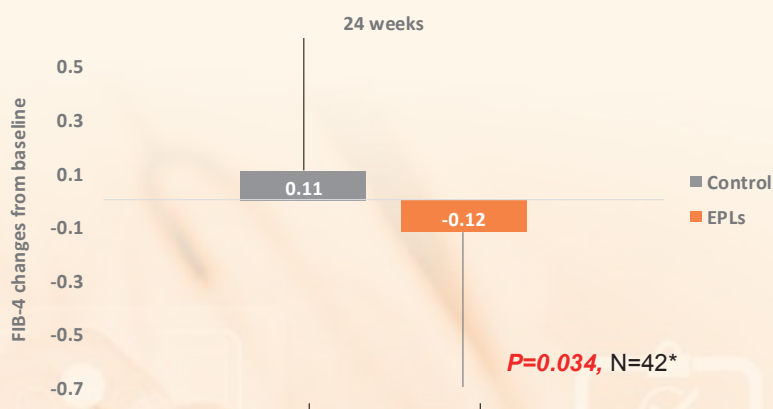
Conclusion

- MASLD is a multifaceted condition closely linked to the broader metabolic syndrome; its growing global prevalence requires a comprehensive and practical treatment strategy.
- Although new pharmacotherapies are emerging, real-world care still depends largely on hepatoprotective agents like EPLs, especially where cost and access limit newer drugs.
- Managing patients with multiple metabolic comorbidities remains challenging, particularly in supporting adherence to medication and lifestyle changes.
- A multidisciplinary approach—combining hepatology, endocrinology, cardiology, and psychological support—addresses the full spectrum of patient needs.
- Expanding evidence from clinical trials and real-world studies supports the continued relevance of EPLs as an adjunctive option in improving biochemical, imaging, and symptomatic outcomes in MASLD.

RELIEF Study (Published in 2025): Real-World Experience in MAFLD and EPLs Treatment

The RELIEF study's primary endpoint was the change in the Fibrosis-4 Index (FIB-4), a validated non-invasive marker of fibrosis, with secondary outcomes assessing liver biochemistry at 12 and 24 weeks and exploratory outcomes evaluating lipid parameters. The results showed that EPLs produced a significant reduction in FIB-4 by week

24, meeting the primary endpoint and signaling meaningful improvement in fibrosis risk (**Figure 2A**). Secondary outcomes were also achieved, with notable declines in ALT and AST observed as early as week 12 (**Figure 2B**). Exploratory analyses demonstrated an improvement in LDL cholesterol within the same timeframe¹⁴.



*A sample size of 84 subjects, divided into 2 groups, achieves a power of 100% with a significance level of 0.05

Figure 2A: Primary endpoint: EPLs significantly decreased FIB-4 index after 24 weeks of treatment¹⁴. EPLs, essential phospholipids; FIB-4, Fibrosis-4 Index

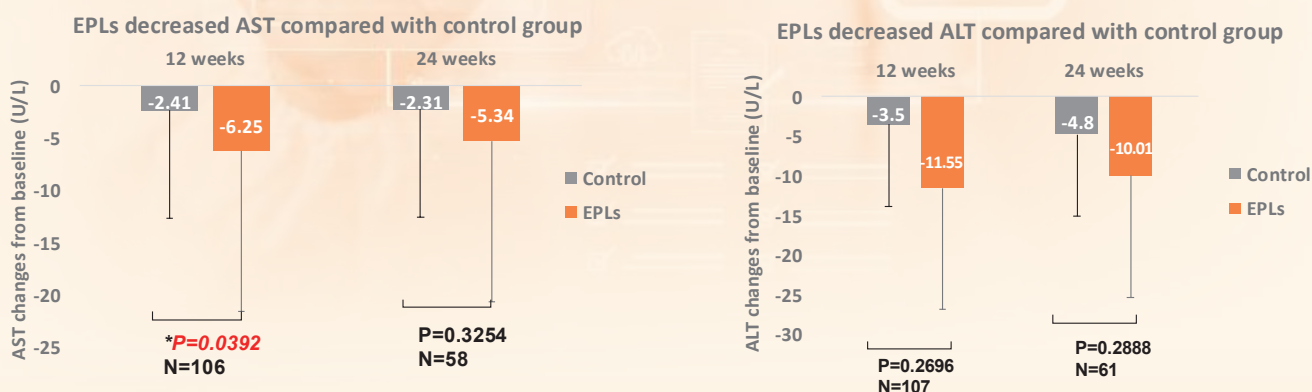


Figure 2B: Secondary endpoint: EPLs decreased ALT/AST after 12 weeks of treatment¹⁴. ALT, alanine transaminase; AST, aspartate aminotransferase; EPLs, essential phospholipids



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World Leukaemia Day 2026: Raising Awareness, Promoting Early Diagnosis, and Supporting Patients Worldwide

World Leukaemia Day 2026 will be observed on September 4, 2026, as part of a global awareness campaign that unites patients, healthcare professionals, researchers, advocacy organizations, and policymakers in the fight against leukaemia. The annual observance aims to educate the public about leukaemia, increase awareness of its signs and symptoms, promote timely medical evaluation, and emphasise the importance of early diagnosis. Because leukaemia symptoms are often vague and easily mistaken for other illnesses, many patients experience delayed diagnosis, which can negatively affect treatment outcomes. Through education, advocacy, and community engagement, World Leukaemia Day encourages people to recognise warning signs, seek medical attention promptly, and support individuals and families affected by the disease. This article explores the significance of World Leukaemia Day, provides an overview of leukaemia, discusses symptoms and diagnostic approaches, highlights advances in treatment, and examines the role of awareness campaigns in improving patient outcomes.

Introduction

World Leukaemia Day is a global awareness initiative observed annually on 4 September. The campaign brings together leukaemia patient organisations and supporters from around the world with a shared mission: increasing public understanding of leukaemia and improving outcomes through education and early detection. By promoting awareness of the disease and encouraging individuals to seek medical attention when symptoms arise, the campaign seeks to reduce the burden of leukaemia worldwide.^{1,2}

Leukaemia remains one of the most significant forms of blood cancer globally. Hundreds of thousands of new cases are diagnosed each year, and the disease continues to account for a substantial number of cancer-related deaths. Public awareness initiatives such as World Leukaemia Day play a crucial role in helping communities identify symptoms earlier and support affected individuals more effectively.³

Understanding Leukaemia

Leukaemia is a haematologic malignancy characterised by the neoplastic proliferation of abnormal white blood cells within the bone marrow and peripheral blood. This uncontrolled cellular growth impairs haematopoiesis, suppressing the generation of functional erythrocytes, platelets, and leukocytes. Consequently, the accumulation of these malignant cells compromises immune defence, oxygen transport, and haemostasis.⁴

Leukaemia is generally classified into several major types:⁴

- **Acute Lymphoblastic Leukaemia (ALL):** A rapidly progressing leukaemia that primarily affects lymphoid cells and is most common in children, although adults can also develop it.
- **Acute Myeloid Leukaemia (AML):** A fast-growing leukaemia affecting myeloid cells. AML is one of the most common forms of acute leukaemia in adults.
- **Chronic Lymphocytic Leukaemia (CLL):** A slowly progressing leukaemia that affects lymphocytes and is most commonly diagnosed in older adults (aged 60 to 70).
- **Chronic Myeloid Leukaemia (CML):** A chronic leukaemia characterised by the abnormal growth of myeloid cells and often associated with a specific genetic abnormality known as the Philadelphia chromosome.

Each type of leukaemia differs in its progression, treatment, and prognosis, making accurate diagnosis essential for effective management.⁵

The Implications of World Leukaemia Day

World Leukaemia Day was established to address a major challenge in leukaemia care: lack of awareness. Many leukaemia symptoms are nonspecific and may resemble common conditions such as influenza, fatigue, or minor infections. As a result, some patients are diagnosed only after their condition becomes severe. The campaign seeks to improve public understanding



so that warning signs are recognised and evaluated sooner.¹

The goals of World Leukaemia Day include:³

- Increasing public awareness about leukaemia
- Educating people about symptoms and risk factors
- Promoting earlier diagnosis and treatment
- Supporting patients and families
- Encouraging healthcare professional education
- Advocating for improved access to care and research

By bringing together global advocacy groups, the campaign demonstrates that leukaemia is a worldwide public health concern requiring coordinated action.¹

● Recognising leukaemia symptoms

One of the central messages of World Leukaemia Day is the importance of recognising leukaemia symptoms. While symptoms can vary among individuals and leukaemia types, several warning signs occur frequently.

- **Persistent Fatigue:** Many individuals with leukaemia experience ongoing tiredness that does not improve with rest.²
- **Frequent Infections:** As leukaemia disrupts normal immune system function, patients may develop frequent or recurring infections that take longer than usual to resolve.⁶
- **Unexplained Bruising or Bleeding:** Low platelet counts can lead to easy bruising, nosebleeds, bleeding gums, or prolonged bleeding from minor injuries.⁷
- **Fever and Night Sweats:** Persistent fever and excessive sweating, particularly during the night, can be important warning signs requiring medical evaluation.⁸
- **Bone and Joint Pain:** Some individuals experience pain or tenderness in bones and joints due to the accumulation of leukaemia cells within the bone marrow.⁹
- **Swollen Lymph Nodes:** Enlarged lymph nodes, particularly in the neck, underarms, or groin, may occur and should be assessed by a healthcare professional.⁸

● Early Diagnosis Saves Lives

Early diagnosis is one of the most effective ways to improve outcomes for people with leukaemia. Prompt

detection allows healthcare professionals to begin treatment before the disease progresses and causes severe complications.¹

Doctors may use several methods to diagnose leukaemia, including complete blood counts, bone marrow examination, genetic and molecular testing, flow cytometry, and imaging studies when appropriate. The information gathered from these tests helps determine the specific leukaemia subtype and guides personalised treatment decisions. It is worthy of note that molecular insights have ushered in the era of precision medicine in paediatric haematology, where treatment decisions are increasingly based on genomic context rather than solely on clinical features (**Figure 1**).¹⁰

● Advances in Leukaemia Treatment

Significant progress has been made in leukaemia treatment over recent decades. Research has led to more targeted and effective therapies, improving survival and quality of life for many patients.²

Targeted Therapies

These medications block specific proteins, genes, or tissue environments that contribute to cancer growth,

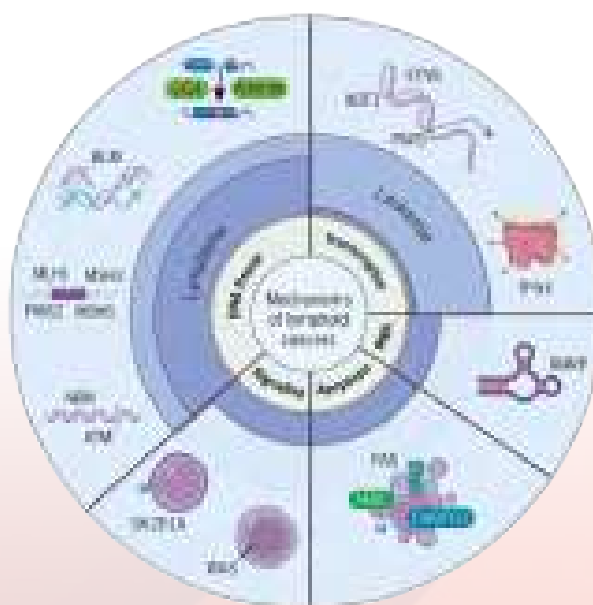


Figure 1: Molecular landscape of common paediatric haematologic malignancies¹⁰

minimising damage to healthy cells:

- Tyrosine Kinase Inhibitors (TKIs) revolutionised the management of CML by directly targeting the abnormal BCR-ABL protein.¹¹
- FLT3 and IDH Inhibitors: These targeted agents are used for specific genetic mutations commonly found in AML patients.¹²

Immunotherapy and Cellular Therapies

These treatments harness the patient's own immune system to recognise and destroy leukaemia cells:

- CAR T-Cell Therapy: A landmark advancement where a patient's T-cells are genetically engineered to attack cancer cells.¹³
- Monoclonal Antibodies and Bispecific T-cell Engagers (BiTEs): These drugs act as bridging agents, binding simultaneously to cancer cells and immune cells to trigger targeted destruction.¹⁴

Stem Cell Transplantation

This procedure replaces diseased, leukaemia-ridden bone marrow with healthy, blood-forming stem cells. For some patients, a stem cell or bone marrow transplant offers the possibility of long-term disease control or cure. Continued investment in research remains essential for developing more effective and accessible treatments worldwide.³

Chemotherapy and Radiation Therapy

These traditional treatments remain fundamental foundational pillars, especially for fast-growing acute leukaemias. Central nervous system prophylaxis delivers chemotherapy directly into the spinal fluid (intrathecal chemotherapy) to prevent leukaemia from spreading to the brain.¹⁵

The Role of Patients, Researchers, and Advocacy Groups

World Leukaemia Day succeeds because of collaboration among diverse stakeholders. Patient organisations provide education, emotional support, and practical resources. Researchers advance scientific understanding and develop new treatments, while healthcare professionals work to improve diagnosis and care. Advocacy groups promote policy changes, funding, and public education initiatives.¹

Together, these groups help ensure that individuals affected by leukaemia receive appropriate support throughout their healthcare journey. Their collective efforts also contribute to reducing stigma and increasing public understanding of blood cancers.²

Global Impact of Awareness Campaigns

Public health awareness campaigns have long demonstrated the ability to influence health-seeking behaviour and improve disease recognition. In the context of leukaemia, awareness initiatives can encourage individuals to pay closer attention to symptoms and seek medical care sooner. Earlier diagnosis is often associated with better treatment opportunities and improved outcomes.¹

World Leukaemia Day also strengthens international cooperation among patient advocacy networks and healthcare organisations. By sharing educational materials and best practices across countries, the campaign amplifies its reach and effectiveness.³

Conclusion

World Leukaemia Day 2026, observed on 4 September, 2026, serves as a vital reminder of the importance of leukaemia awareness, early diagnosis, patient support, and ongoing research. Leukaemia affects hundreds of thousands of people worldwide each year, and many patients face challenges related to delayed diagnosis because symptoms can be difficult to recognise. Through education and advocacy, World Leukaemia Day encourages individuals, communities, healthcare professionals, and policymakers to work together toward earlier detection and better outcomes.

By learning about leukaemia, recognising warning signs, supporting patients and families, and promoting access to quality healthcare, people everywhere can contribute to a future where more individuals receive timely diagnoses and effective treatment. As the global community observes World Leukaemia Day 2026, the message remains clear: awareness saves lives, and together we can make a meaningful difference in the fight against leukaemia.



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2026 ANNUAL SCIENTIFIC MEETING

Clinical Excellence and Advancement: Bridging Primary and Specialist Care



6 September 2026 (Sunday)
09:00 – 17:15



Format: Hybrid

- **Physical Participation:** 7/F & 8/F, Cordis Hong Kong
555 Shanghai Street, Mongkok, Kowloon, Hong Kong
- **Online Participation:** Zoom Webinar (Live)



**Deadline for
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Physical Attendance:
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Online Attendance:
30 August 2026 (Sunday)

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- Non-members of HKCMA
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 - Nurses and Allied Health Professionals (Early-bird HK\$200, before 15 July) **HK\$ 300**
 - Hong Kong Medical Students **Free of charge**
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Prof. Tom LAWTON

Head of Clinical Analytics, Improvement Academy Data Analytics Unit, Bradford Institute for Health Research, UK



Prof. William W. WONG, M.D

Professor and Chair Emeritus, Department of Radiation Oncology, Mayo Clinic Arizona, USA

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Prof. LU Lin 陸林院士

Academician, Chinese Academy of Sciences
Boya Chair Professor, Peking University
Director, National Center for Mental Disorders



Prof. Hung-hsueh CHOU 周宏學教授

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Specialist in Infectious Disease
Immediate Past President, The Hong Kong Society for Infectious Diseases

CME & CNE application in progress



HONG KONG CHINESE MEDICAL ASSOCIATION

香港中華醫學會

2026 ANNUAL SCIENTIFIC MEETING

Clinical Excellence and Advancement: Bridging Primary and Specialist Care

Time	Level 7, Ballroom I & II	Level 8, Shanghai Room I & II
0830-0900	Registration (onsite and online)	
0900-0910	Opening Remarks (by co-chair Dr. Mike Yat-wah KWAN 關日華醫生)	
	Session 1 Chairpersons: Dr. Loletta Kit-ying SO 蘇潔瑩醫生 & Dr. Carol Chi-hei KWOK 郭子熹醫生	Concurrent Session 1 Chairperson: Dr. Adrian Hon-bong LEUNG 梁漢邦醫生
0910-0940	Lecture 1A Difficult-to-Treat and Severe Asthma in Clinical Practice: Insights from an Outpatient Clinic Prof. Diahn-Warng PERNG, Taiwan 彭殿王教授 (via Online)	FibroScan Workshop (Hands-on experience on Fibroscan)
0940-1010	Lecture 2A Proton Therapy in Prostate Cancer Prof. William W. WONG, M.D, USA (via Online)	
1010-1040	Lecture 3A Colorectal Cancer Screening in Asia Pacific - Scientific Updates, Implementation Journey, and Innovative Approaches Prof. Martin Chi-sang WONG, HKSAR 黃至生教授	
1040-1100	Coffee Break	
1100-1145	Opening Ceremony Opening Address by Guest of Honour Dr. Fei-chau PANG, Commissioner for Primary Healthcare 基層醫療健康專員彭飛舟醫生	
1145-1230	Chinese Academy Distinguished Lecture 中國院士傑出講座 Orator: Dr. Catherine Xiao-rui CHEN 陳曉瑞醫生 AI in Clinical Medicine: Opportunities, Applications and Future Perspective Prof. LU Lin - Academician, Chinese Academy of Sciences 中國科學院陸林院士	
1230-1245	Session 2 Chairpersons: Dr. Mike Yat-wah KWAN 關日華醫生 & Prof. Daniel Tak-mao CHAN 陳德茂教授	
1245-1330	Lunch Symposium 1 The New Era of RSV Protection: Redefining Infant Respiratory Health from Clinical Trials to Real World Practice Prof. YANG Zifeng, Guangzhou 楊子峰教授	
1330-1400	Lunch Symposium 2 Improving CKD Management and Advancing Novel Approaches to CKD-Related Anaemia Prof. Sunil BHANDARI, UK (via Online)	
	Session 3 (CKM/obesity session) Chairpersons: Dr. Catherine Xiao-rui CHEN 陳曉瑞醫生 & Dr. Simon Chi-yuen CHEUNG 張志遠醫生	Concurrent Session 3 (vaccinology) Chairpersons: Dr. Chun-kong NG 吳振江醫生 & Dr. Wilson LAM 林緯遜醫生
1400-1430	Lecture 4A Achieving Long Term CKM Preservation Across the CKD Spectrum Dr. Edith Wing-kar CHOW, HKSAR 周穎嘉醫生	Lecture 4B HPV Gender Neutral Vaccination: Learning from Taiwan Prof. Hung-hsueh CHOU, Taiwan 周宏學教授 (via Online)
1430-1500	Lecture 5A Advancing DKD Management: The Emerging Role of Finerenone Dr. Alan Chun-hong LEE, HKSAR 李俊康醫生	Lecture 5B Japanese Encephalitis: Fresh Takes Dr. Ada Wai-chi LIN, HKSAR 連慰慈醫生
1500-1530	Lecture 6A Are GLP1 Receptor Agonists the Pan-vascular Panacea? Dr. Adrian Yan-yue CHEONG, HKSAR 張仁宇醫生	Lecture 6B From Evidence to Practice: COVID-19 Burden, Chronic Disease Impact, and Expert Perspectives Dr. Christopher Kim-ming HUI, HKSAR 許建名醫生
1530-1545	Coffee break	
	Session 4 Chairpersons: Dr. Jane Chun-kwong CHAN 陳真光醫生 & Prof. Bernard Man-yung CHEUNG 張文勇教授	
1545-1615	Lecture 7A Bridging Public Health and Primary Care: Adult Immunisation in Hong Kong Dr. Albert Ka-wing AU, HKSAR 歐家榮醫生	
1615-1645	Lecture 8A Exploring Cutting-Edge Advancements in Early Alzheimer's Treatment Prof. Jung-lung HSU, Taiwan 徐榮隆教授	
1645-1710	Lecture 9A Clinicians, AI, and the Liability Sink Prof. Tom LAWTON, UK (via Online)	
1710-1715	Closing Remarks (by co-chair Dr. Catherine Xiao-rui CHEN 陳曉瑞醫生)	

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Can Melatonin Be an Adjuvant Treatment for Musculoskeletal Pain?

by Jasmine Lai

Musculoskeletal (MSK) pain affects nearly 50% of the global population; it is the main contributor to disability worldwide, with people experiencing MSK pain more than any other type of pain.^{1,2}



MSK pain has a well-documented, antagonistic, bidirectional relationship with sleep: stress responses and discomfort from pain make it harder to fall and stay asleep, while sleep disturbances promote inflammatory substances and disrupt pain inhibitory mechanisms which heighten pain severity and sensitivity.¹

Non-opioid analgesics are the most commonly prescribed pain treatment, however personalised approaches combining pharmacological and non-pharmacological methods are increasingly recommended to address the breadth of MSK pain phenotypes.²

Interestingly, **animal studies have suggested that melatonin may have analgesic properties**, and clinical trials have been conducted to understand the role of melatonin supplements in different types of pain.¹ Indeed, the **World Health Organization has proposed a four-step ladder for nociceptive MSK disorders**, with the first step being the use of **non-opioid analgesics with/without effect-enhancing adjuvants**.² With its overt prevalence and burden, melatonin may offer a low-cost addition to a multimodal pain management approach.

MELATONIN:¹

Plausible Mechanisms for Pain Relief

Pain:



- Reduce neuronal excitability, alleviate allodynia, and reduce central sensitization



- Antioxidant and anti-inflammatory effects: scavenges free radicals and suppresses proinflammatory mediators (i.e. cytokines, prostaglandins)

Sleep:



- Improve sleep quality and reduce anxiety may attenuate pain perception



- Restore circadian rhythms that are disrupted in chronic pain syndromes

STUDY DESIGN:¹

Systematic Review and Meta-Analysis of Placebo and Active Controlled Trials (Analgesics)

23 studies with 2,028 participants



Risk of Bias: Low-Moderate

Outcomes:



Chronic MSK Pain Intensity



Post-Operative MSK Pain Intensity



Sleep Quality

RESULTS:¹

Chronic MSK Pain: 9 Studies

Including chronic low back pain, neuropathic pain, knee osteoarthritis, rheumatoid arthritis, and temporomandibular disorders

Melatonin doses:
3-10 mg/day



Significant reduction in pain compared with all comparator treatments

Pooled mean difference: **-8.96/100**
(95% CI: -12.85 to -5.08; **P<0.0001**; $I^2=60\%$)

Melatonin vs Analgesic (E.g., diclofenac)

Mean difference: **-10.20**
(95% CI: -17.61 to -2.79; **P=0.007**; $I^2=21\%$)



Melatonin vs Non-Analgesic (E.g., fluoxetine, amitriptyline)

Mean difference: **-11.03**
(95% CI: -15.18 to -6.88; **P<0.0001**; $I^2=30\%$)

Melatonin vs Placebo

Mean difference: **-6.76/100**
(95% CI: -15.89 to 2.36; **P=0.15**; $I^2=68\%$)

SENSITIVITY ANALYSES:

Results from Low Risk of Bias Studies 4 Studies

Longer treatment duration
was associated with more
favourable effects

$\beta=-0.97$

(95% CI -1.62 to -0.32, **P=0.009**)

Significant pain reduction compared with

- **Placebo** (**P=0.01**; heterogeneity decreased from $I^2=68\%$ (primary analysis) to $I^2=51\%$)
- **and non-analgesic medication** (**P<0.0001**)

No studies available for analgesic comparators

Improved sleep quality with melatonin vs all treatments in chronic MSK pain 7 studies

Mean difference: **-11.35/100**
(95% CI: -18.65 to -4.06; **P=0.002**; $I^2=92\%$)

No significant difference between melatonin and all comparator treatments for post-operative MSK pain intensity and sleep quality

CONCLUSION

Melatonin may provide modest benefits for pain in people with chronic MSK pain, and has the potential adjunct in MSK pain treatment. However, the effects of melatonin generally did not reach the accepted minimal clinically important difference threshold for pain intensity in chronic pain populations (11-14 mm).

Abbreviations: CI, confidence interval; MSK, musculoskeletal

Reference: 1. Puterflam J, et al. Pain. 2026. Published ahead of print. <https://doi.org/10.1097/j.pain.0000000000004045> 2. Moretti A, et al. Ther Adv Musculoskelet Dis. 2024;16:1759720X241253656.

Diagnosis of Liver Fibrosis (LF) in the General Population

by Andrew Chan

Key Highlights

- High prevalence of **undiagnosed LF**¹
- Associated with **harmful alcohol use** and number of **metabolic diseases**¹⁻³
- Urgent need for primary prevention of LF¹



Introduction

- LF, the main hallmark of Chronic Liver Disease (CLD), is a leading cause of death^{1,4}
- Vibration-Controlled Transient Elastography (VCTE) for early detections¹
- CLD and LF still vastly underdiagnosed^{1,5}
- There is a need to investigate aetiologies of CLD and LF in undiagnosed population¹



LiverScreen Study Design¹

Population-based | Prospective | Multinational Cohort Study across 35 Sites in Europe

67074 Invited Participants



30541 at Screening Visit*



30199 Reliable VCTE Results Obtained and Visit Completed



Primary Outcome

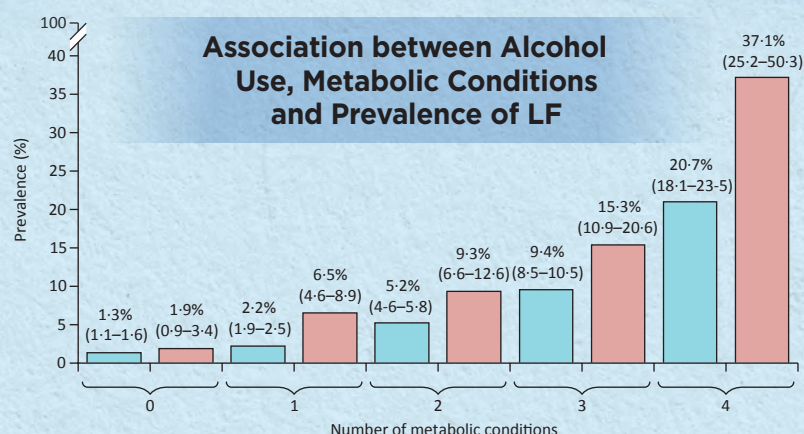
1. Assessing the prevalence of LF
2. Evaluating relationship of LF with
 - Metabolic Risk Factors
 - Alcohol Consumption



* 40 years old or older without known chronic liver disease (except liver steatosis), mental incapacity, language barrier, insufficient social support, extrahepatic diseases that impair short-term prognosis

Results¹

High Prevalence 2075 (6.9%) were LF-positive[‡]



Blue bars=no harmful alcohol use
Pink bars=harmful alcohol use

Harmful alcohol use was associated with an additional increase in the prevalence of elevated LSM, beyond that conferred by metabolic risk factors alone



Prevalence of liver stiffness measurement (LSM; ≥ 8 kPa) according to the number of metabolic conditions and its association with harmful alcohol use

Top Clinical Conditions Related to LF



Obesity

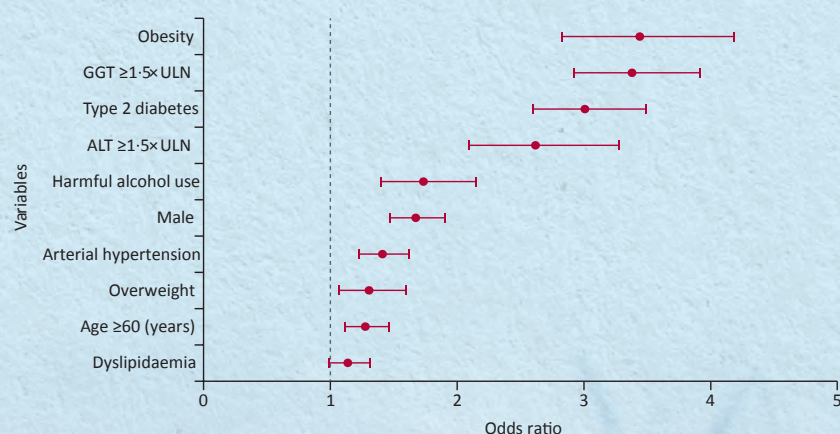


Type 2 Diabetes



Harmful Alcohol Use

Clinical and Laboratory Variables Related to Increased LSM



Multivariable analysis of factors related to increased liver stiffness measurement (LSM; ≥ 8 kPa). Values shown are adjusted odds ratio and 95% CI, adjusted by country, sex, and all variables included in the model.

Overall Estimated Prevalence of CLD with Fibrosis[#] 1.6% (477/30199)

[‡]LF-positive defined as an LSM of 8 kPa and greater or an alanine aminotransferase concentration of at least 1.5 the upper limit of normal, or a combination of both

[#]Participants with a positive screening test were subsequently referred to a second visit with an hepatologist to confirm the presence of CLD. A diagnosis of CLD with fibrosis was made in 477 of 1491 participants, almost one-third (32%) of individuals who completed the evaluation for liver disease, which represents an overall estimated prevalence of CLD with fibrosis of at least 1.6% (477 of 30 199 participants)

Abbreviations: ALT, alanine aminotransferase; CI, confidence interval; CLD, chronic liver disease; GGT, gamma-glutamyl-transpeptidase; kPa, kilopascal; LF, liver fibrosis; LSM, liver stiffness measurement; ULN, upper limit of normal; VCTE, vibration-controlled transient elastography

Reference: 1. Graupera I, et al. The Lancet. 2026;407(10537):1448-58. 2. Fu J, et al. npj Gut and Liver. 2025;2(1):13. 3. Leal-Lassalle H, et al. npj Gut and Liver. 2025;2(1):1. 4. Liu T, et al. Biomarker Insights. 2012;7:105-17. 5. Patel KM, et al. Digestive Diseases and Sciences. 2024;69(10):3721-8.

Polyendocrine Metabolic Ovarian Syndrome (PMOS): More Than an Update of Terminology

Polycystic ovary syndrome (PCOS), now referred to as polyendocrine ovarian syndrome (PMOS), is a complex cardiometabolic hormonal disorder prevalent in reproductive-age females. Due to the complexity and variability of its symptoms, the management of this disorder continues to be optimised: including efforts to uncover its pathophysiology, identify better treatment options, and refine terminology. This article seeks to outline what PMOS is, how it is managed, and highlight the recent extensive change in terminology and its rationale. These actions serve to improve the management of PMOS in healthcare, providing the patients with a better tomorrow.

What is Polyendocrine Metabolic Ovarian Syndrome (PMOS)?

Polyendocrine metabolic ovarian syndrome (PMOS), previously known as polycystic ovary syndrome, is a common cardiometabolic hormonal disorder affecting one in eight women.¹ The most prominent symptoms of PMOS include irregular menstrual cycles, infertility, acne vulgaris, androgenetic alopecia, acanthosis nigricans, hirsutism, secondary dysmenorrhoea, and weight gain.^{2,3} PMOS is also associated with anxiety, depression, sleep apnoea, diabetes, hypertension, dyslipidaemia, and even endometrial cancer.² As illustrated above, the features of PCOS are often multisystemic, encompassing metabolic, reproductive, psychological, and dermatological manifestations.¹ Indeed, the aetiology and pathophysiology of PMOS are complex and unclear. Therefore, it is most important that the proposed mechanisms be examined cautiously.

Evidence shows that genetic and epigenetic factors may be the culprits for PMOS. Contemporary theories point to genes associated with insulin resistance, steroidogenesis, and gonadotrophic dysregulation.⁴ One of the numerous genes that are strongly associated with PMOS is the DENND1A (Differentially Expressed in Normal and Neoplastic Development isoform A1) gene.^{4,5} The DENND1A gene codes for 2 protein variants in the cytoplasm and nuclei of theca cells, with the second variant (DENND1A.V2) inducing increased CYP17A1 and CYP11A1 expressions, and driving androgen biosynthesis.⁶ This coheres with the established understanding of PMOS pathophysiology. Another major contributor to PMOS is epigenetics.

Various studies have found that dysregulated methylation in various tissues and peripheral blood may contribute to PMOS. Promoter hypomethylation of the AMH and AMHR2 genes is a crucial epigenetic alteration that causes the chronic overactivation of Anti-Müllerian Hormone signalling, shifting follicle regulation into a state of permanent arrest. Hypermethylation of the INSR gene is a crucial epigenetic mechanism that drives ovarian-specific insulin resistance and metabolic dysfunction.⁵ These factors are just a small part of the complex aetiology of PMOS. However, they demonstrate the pathways by which PMOS progresses.

The pathophysiology of PMOS is similarly complex. Insulin resistance, as previously discussed, is a major part of PMOS progression as insulin resistance leads to hyperandrogenism in three ways: increasing circulating androgens as the production of sex hormone binding globulin (SHBG) is decreased; increasing androgen production in ovarian tissues, driven by the gonadotropic property of insulin; promoting androgen production via luteinising hormone (LH) sensitisation in theca cells.^{5,6} It is also suggested that gonadotropin-releasing hormone (GnRH) from the hypothalamic-pituitary-ovarian axis plays a major role.^{5,6} It has been demonstrated that an increase in GABA innervation and AMH levels leads to increased GnRH neuron firing.^{7,8} The increased firing of GnRH neurons increases the luteinising hormone to follicle-stimulating hormone ratio (LH/FSH), contributing to ovarian hyperandrogenism.⁵ These findings not only serve as the underlying mechanisms of PMOS pathophysiology but also represent possible therapeutic targets (**Figure 1**).



How Is PMOS Managed?

The complex and multigenic nature of PMOS underscores the importance of holistic management strategies. Pharmacological interventions and lifestyle modifications remain the paradigm for PMOS care. Combined hormonal contraceptives remain the first-line medication for the alleviation of hyperandrogenic symptoms such as hirsutism, acne vulgaris and anovulation in PMOS.^{2,5} Increased attention has also been given to the use of metformin and glucagon-like-peptide-1 receptor agonists (GLP-1RA) for managing metabolic symptoms by insulin sensitisation, and managing blood glucose to prevent diabetes and dyslipidaemia.^{2,5} To improve fertility and induce ovulation, letrozole is also often utilised; similarly, GLP-1RA is used to improve reproductive function.^{2,5} While the importance of pharmacological interventions have been underscored, the effects of lifestyle modifications cannot be undermined. Dietary changes and exercise are key lifestyle interventions for improving PMOS.² Body fat composition and insulin resistance may be improved with healthy dieting and adequate exercise, with the additional benefits of preventing diabetes and cardiovascular diseases.^{2,5} It is imperative to understand that PMOS is highly heterogeneous, requiring individualised and holistic management.⁵

Why Has the Name Changed?

There are two major reasons for the change in name: improved clarity, and reduced stigmatisation.

The new name PMOS is more medically accurate and clear, facilitating diagnosis and fostering patient awareness. Prior to the renaming, PMOS was known as polycystic ovary syndrome (PCOS). However, PCOS fails to encapsulate the clinical features of the disorder: namely, it over-emphasises the gynaecological aspects

of the disorder whilst neglecting the aforementioned neuroendocrine and metabolic aspects.^{1,9} In fact, “polycystic ovary” is now known to be inaccurate, as ovarian cysts are not specific to the disorder and may not be present in all cases.^{1,9} This inaccuracy may confuse healthcare professionals and patients, resulting in delayed diagnosis, ineffective communication, and ultimately dissatisfactory healthcare.¹ PMOS improves clarity by incorporating the polygenic, endocrinal and metabolic nature of the disorder, making the name much clearer.

The renaming may also help reduce stigma surrounding women's reproductive health and fertility. In certain cultures, fertility is highly emphasised and linked to sociocultural values.¹ One such example would be Chinese culture, where a statistically significant relationship between women's fertility, and socioeconomic standing has been demonstrated.¹⁰ In such cases, PCOS diagnoses may contribute to undesirable distress and pressure on the patients.¹ This may even contribute to or even exacerbate existing

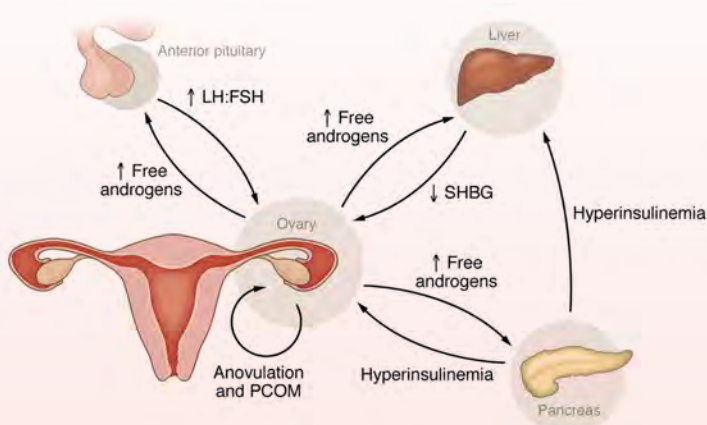


Figure 1. PMOS Pathophysiology⁵

psychological conditions such as anxiety or depression in patients. Although PMOS retains the term 'ovary', it shifts the focus towards the disorder's endocrine and metabolic features, helping to reduce stigmatisation. It is, therefore, hoped that such change will bring about improved clinical outcomes for patients suffering from PMOS (**Figure 2**).¹¹

How Is the Change Implemented?

To optimise the change in terminology, the global implementation is planned as a staged, progressive transition¹:

- Stage 1: Publish the health policy for academic dissemination.
- Stage 2: Develop healthcare resources reflecting the change in terminology.
- Stage 3: Apply the change through structured communication strategies.
- Stage 4: Integrate the new terminology into existing medical systems.
- Stage 5: Foster alignment among policymakers, publishers, and the healthcare industry to support the adoption of the updated terminology.
- Stage 6: Seek formal classification by international

bodies such as the WHO (World Health Organization).

- Stage 7: Monitor and evaluate the transition over three years.
- Stage 8: Incorporate the change into international guidelines.

Staged implementation allows for smooth adaptation by all stakeholders, ensuring clarity and efficacy.

Conclusion

PMOS, previously known as PCOS, is a common cardiometabolic hormonal disorder with complex aetiology and pathophysiology warranting further investigation to develop better treatments. Current management of PMOS focuses on pharmacological interventions and lifestyle modifications to alleviate symptoms. While research regarding its aetiology and possible therapeutic strategies continues, a change in terminology is advocated in hopes of improving clarity and reducing stigmatisation. Indeed, there is still much to be done to improve the clinical outcomes and quality of life for PMOS patients. This change serves as a reminder that improvements, no matter the scale, can be made to improve the lives of patients – “in the pursuit of excellence”.

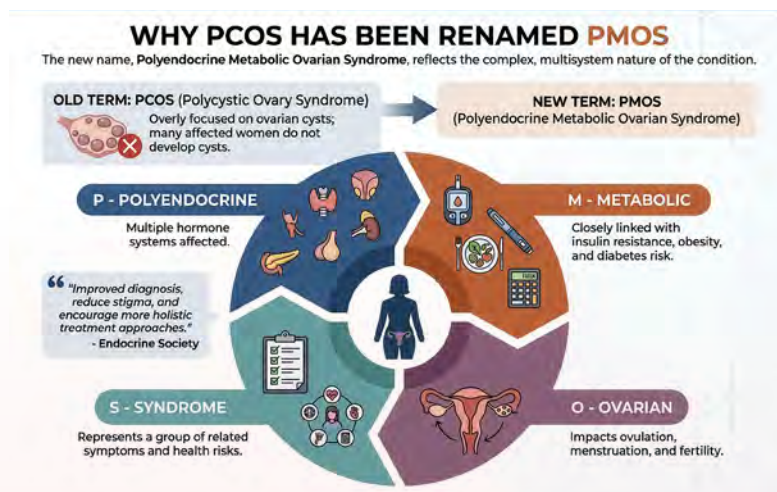


Figure 2. Why PCOS Has Been Renamed PMOS¹¹



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How Vitamin B3 Stands Guard Over Your Eyes



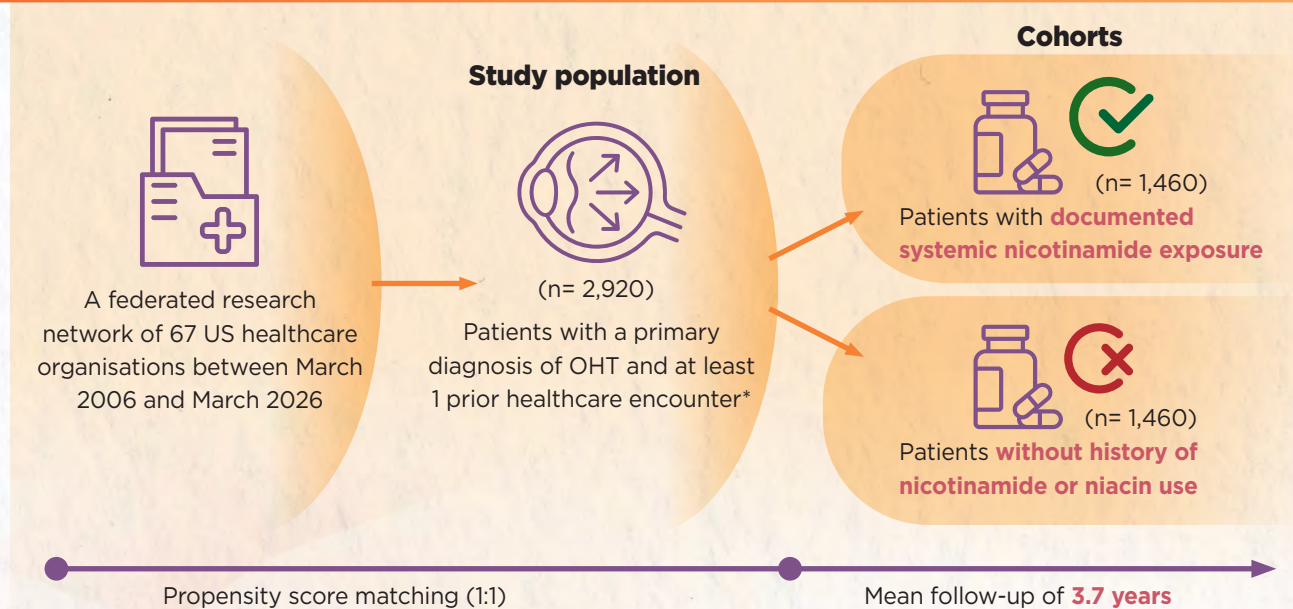
by Michelle Lee

Ocular hypertension (OHT) is the leading risk factor for developing open-angle glaucoma, a major cause of vision loss. While laboratory studies suggest that Vitamin B3 (nicotinamide) might act as a metabolic shield to protect the optic nerve, real-world proof has been missing. This landmark study investigates whether taking simple Vitamin B3 supplements can block the onset of open-angle glaucoma (POAG) and save patients from needing aggressive eye drops or surgeries to protect their sight.

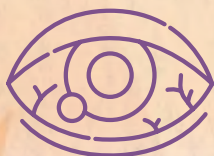
Objective

To determine whether **systemic nicotinamide supplementation** is associated with **reduced risk of POAG** and delayed need for medical or surgical escalation in patients with OHT.

Cohort Study Design



Primary Outcome

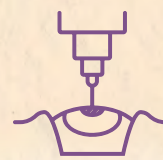


Risk of POAG diagnosis

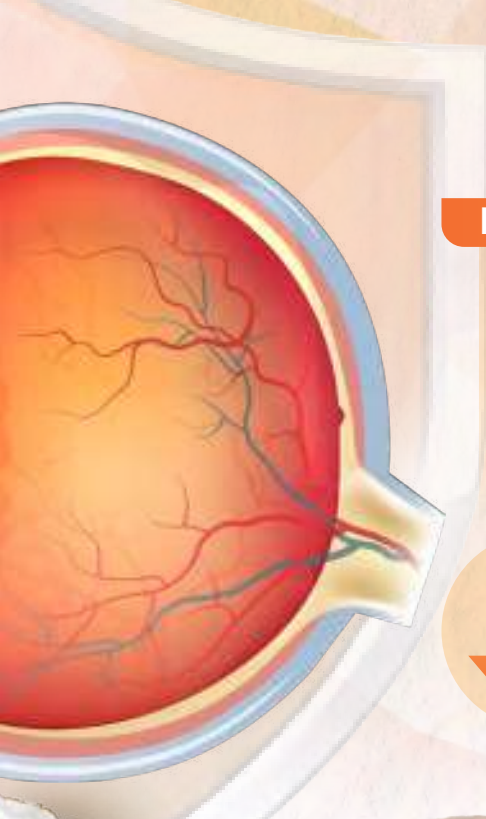
Key Secondary Outcomes



Initiation of first-line topical glaucoma therapy



Requirement for laser trabeculoplasty



Results

POAG diagnosis occurred in:



3.7%
in nicotinamide group
(n=51/1,460)

vs.

9.0%
in nicotinamide group
(n=132/1,460)



66% Lower risk of
POAG diagnosis
with nicotinamide
(HR=0.34; 95% CI: 0.25-0.47; p<0.001)

5.5% Absolute risk
reduction
(95% CI: 3.8-7.3)

Topical intraocular pressure-lowering therapy was initiated in:



13.6%
in nicotinamide group
(n=198/1,460)

vs.

21.2%
in control group
(n=309/1,460)



43% Lower risk of starting
topical therapy with
nicotinamide
(HR=0.57; 95% CI: 0.48-0.68; p<0.001)

Laser trabeculoplasty was performed in:



0.8%
in nicotinamide group
(n=12/1,460)

vs.

1.9%
in control group
(n=28/1,460)



62% Lower risk of
undergoing laser
trabeculoplasty with
nicotinamide
(HR=0.38; 95% CI: 0.19-0.74; p=0.003)

Conclusion

- Nicotinamide **potentially delays POAG diagnosis and reduces the rate of subsequent prescribed medical interventions in patients with OHT.**
- Nicotinamide supplementation represents a **potential adjunctive therapy to standard care** that warrants further prospective clinical trials.



Abbreviations: CI, confidence interval; HR, hazard ratio; OHT, ocular hypertension; POAG, primary open-angle glaucoma; US, United States.

Reference: 1. Muayad J, et al. JAMA Ophthalmol. Published online July 09, 2026. doi:10.1001/jamaophthalmol.2026.2472

Long-Term Obesity Management with Tirzepatide: Insights from the SURMOUNT-MAINTAIN Trial



Obesity is a chronic, progressive disease that requires long-term management to sustain weight loss and reduce cardiometabolic risk. While modern anti-obesity medications can achieve substantial bodyweight reduction, maintaining these benefits after initial treatment remains a significant challenge. The SURMOUNT-MAINTAIN trial evaluated whether continuing tirzepatide at the maximum tolerated dose (MTD), reducing the dose to 5 mg, or discontinuing treatment could best preserve weight loss achieved during a 60-week intensive treatment phase. The study demonstrated that continued tirzepatide therapy was highly effective in maintaining bodyweight reduction and associated cardiometabolic improvements. Although dose reduction to 5 mg resulted in some weight regain, it remained significantly superior to treatment discontinuation and may represent a practical alternative for certain patients. These findings reinforce the concept that obesity should be managed as a chronic disease requiring ongoing therapy and support individualised, patient-centred treatment strategies.

Introduction

The treatment of obesity has entered a new era with the development of highly effective medications such as tirzepatide, a dual glucose-dependent insulinotropic polypeptide (GIP) and glucagon-like peptide-1 (GLP-1) receptor agonist. Clinical trials have shown that tirzepatide can produce substantial and clinically meaningful weight loss while improving multiple cardiometabolic risk factors.¹ However, achieving weight loss is only part of the challenge. Long-term maintenance of reduced body weight remains a critical issue in obesity care.²

Weight regain frequently occurs when treatment is discontinued due to the chronic nature of obesity and the body's biological tendency to restore lost weight. The SURMOUNT-MAINTAIN trial was designed to address an important clinical question: can the benefits of tirzepatide be maintained by continuing treatment, reducing the dose, or discontinuing therapy altogether?³

Understanding the SURMOUNT-MAINTAIN Trial

SURMOUNT-MAINTAIN was a multicentre, randomised, double-blind, placebo-controlled Phase 3b trial conducted across 20 sites in the United States. The study enrolled adults with obesity or overweight accompanied by weight-related comorbidities. Participants initially underwent a 60-week open-label weight-loss phase during which all received tirzepatide at their maximum tolerated dose of either 10 mg or 15 mg.³

After achieving significant weight reduction, eligible participants were randomly assigned to one of three

groups for an additional 52-week maintenance period:³

- Continued tirzepatide at the maximum tolerated dose (MTD)
- Reduced tirzepatide to 5 mg weekly
- Switched to placebo

This design allowed investigators to directly compare different maintenance strategies after successful weight loss.³

Continued Tirzepatide Provides the Strongest Weight-Loss Maintenance

The study's results clearly demonstrated the value of maintaining treatment intensity. Participants who remained on tirzepatide MTD achieved an average bodyweight reduction of 21.9% from baseline at week 112, compared with 16.6% among those reduced to 5 mg and 9.9% among those switched to placebo (**Figure 1**).³

Importantly, patients who continued on MTD essentially maintained their weight loss during the maintenance phase, showing only a negligible change of -0.2% from the time of randomisation. In contrast, participants receiving 5 mg experienced an average weight regain of 7.0%, while those switched to placebo regained 15.2% of their body weight.³

Among individuals who had reached a weight plateau before randomisation, continued MTD therapy preserved 96.5% of the weight lost during the initial treatment period. This finding suggests that ongoing full-dose treatment can effectively prevent the substantial weight regain commonly observed after discontinuation of obesity medications.³



💡 Dose Reduction to 5 mg: A Viable Alternative for Some Patients

One of the most innovative aspects of SURMOUNT-MAINTAIN was its evaluation of dose reduction rather than simply comparing treatment continuation with discontinuation. Patients who reduced to 5 mg maintained 67.9% of their initial weight reduction, significantly outperforming placebo recipients, who maintained only 42.8% of their initial weight loss. Furthermore, 42.4% of participants receiving 5 mg were able to maintain at least 80% of their original weight loss, compared with only 10.4% in the placebo group.³

These findings suggest that reducing tirzepatide to 5 mg may offer a reasonable compromise for patients who experience tolerability concerns, prefer lower medication exposure, or face other barriers to continued MTD treatment. However, responses varied considerably between individuals, indicating that dose reduction

may not be suitable for everyone. The study therefore supports a personalised approach, where clinicians can balance efficacy, tolerability, patient preferences, and cost considerations when determining long-term treatment plans.³

💡 Cardiometabolic Benefits Extend Beyond Weight Loss

At week 112, participants continuing tirzepatide MTD experienced greater reductions in triglycerides, systolic blood pressure, fasting glucose, fasting insulin, and HbA1c compared with placebo. Notably, 92.7% of participants with prediabetes at baseline who continued MTD treatment achieved normal HbA1c levels, compared with only 51.0% of those switched to placebo.³

💡 Safety and Tolerability

The safety profile of tirzepatide in SURMOUNT-MAINTAIN was consistent with previous studies. Gastrointestinal adverse events, including nausea, vomiting, diarrhoea, and constipation, were the most commonly reported side effects. These events were generally mild to moderate in severity and occurred most frequently during dose escalation.^{4,5} Interestingly, the 5 mg group appeared to experience fewer gastrointestinal side effects. This observation suggests that dose reduction may improve tolerability while still preserving a substantial proportion of treatment benefits.³

💡 Conclusion

The SURMOUNT-MAINTAIN trial provides compelling evidence that long-term therapy is essential for maintaining weight loss and cardiometabolic improvements in adults with obesity. Continuing tirzepatide at the maximum tolerated dose produced the most robust and sustained benefits, preserving nearly all of the weight loss achieved during the initial treatment phase. While reducing the dose to 5 mg resulted in some weight regain, it remained substantially more effective than treatment discontinuation and may serve as a valuable option for patients requiring a lower treatment intensity. Overall, the findings emphasise that obesity is a chronic disease requiring ongoing management and support a patient-centred approach that tailors treatment intensity to individual needs, preferences, and clinical responses.

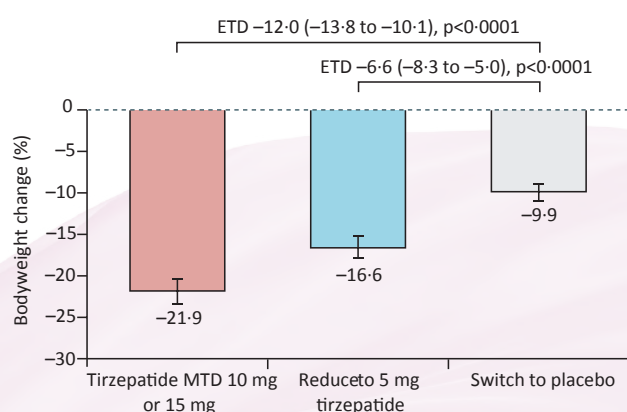


Figure 1: Model-based estimate percent change in bodyweight from baseline to week 112.³ ETD, estimated treatment difference.



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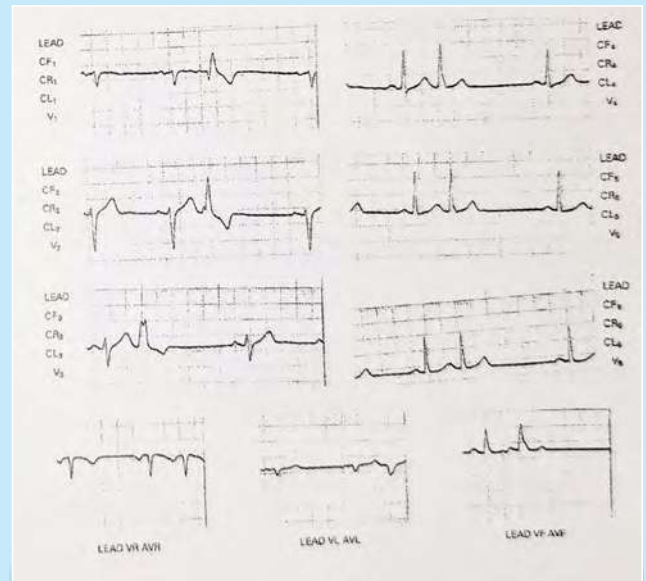
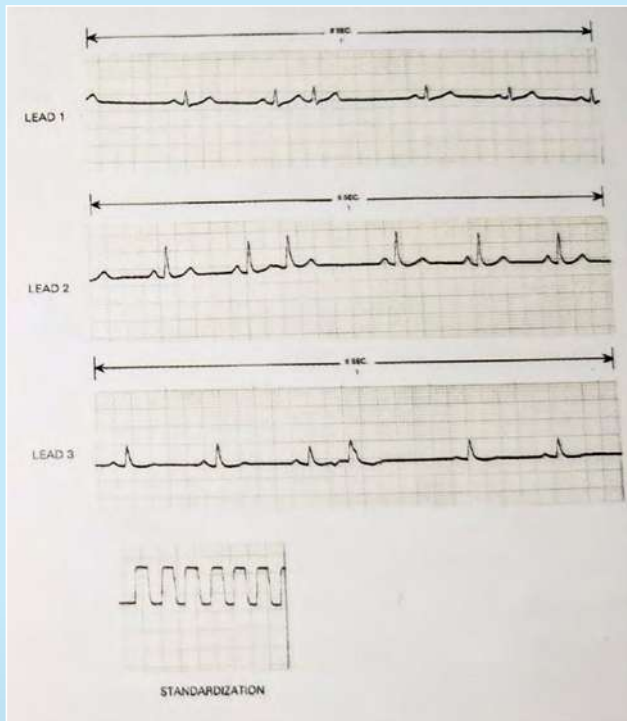
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ECG CME AUGUST 2026 (0.5 CME POINTS)

Dr. Pun Chiu On, Specialist in Cardiology

History:

This 68 year old man came to see you for routine yearly medical check up. He was a chronic smoker. Physical examination of the pulse revealed occasional irregular beats. The rest of the examination was unremarkable.



Question: What is the ECG diagnosis?

(Please tick one)

- ☐ Normal ECG
- ☐ Wolff-Parkinson-White syndrome
- ☐ Sinus arrhythmia
- ☐ Wenckebach block
- ☐ Atrial premature beat (atrial ectopic beat) with aberrant conduction

Please complete the Self-Study by visiting our website: <https://cmevideo.hkdu.org/> or scan the QR code to submit your answers on or before **15-SEPTEMBER-2026**



Answer to June 2026 Issue's CME

ECG diagnosis:

Right atrial enlargement (right atrial enlargement is also known as P-pulmonale).

Reason for the ECG diagnosis:

- P wave in leads I, or II, or III or aVF > 2.5mm in height. In this ECG, the P wave at lead II is 3.5 mm.
- The other minor criteria that could also be used in diagnosis are: P wave axis >+ 75° (+105° in this ECG), or positive part of P wave in lead V1 > 1.5 mm.

Possible aetiology:

- Chronic obstructive airway disease (commonest cause).
- Any cause of pulmonary hypertension, or right ventricular hypertrophy.
- Right atrial ischaemia or infarction (rare cause).

Significance:

It depends on the underlying aetiology.

Principle of management:

Manage the underlying cause.

Management of this patient:

This patient had chronic obstructive pulmonary disease. He was treated as acute exacerbation of chronic bronchitis, with antibiotic, mucolytic, expectorant, and bronchial dilator. He was advised to stop smoking, and to avoid dusty environment during work or to wear a mask.

Upcoming Academic Events

in Hong Kong



September–December 2026

The Chinese University of
Hong Kong

**2nd CUHK Advanced
Frontal Sinus
Surgery Course &
Olfactory Workshop**

Sep 17-19

Thursday-Saturday



Hong Kong Institute of
Diabetes and Obesity

**28th Diabetes and
Cardiovascular Risk
Factors –East Meets
West Symposium**

Oct 03-04

Saturday-Sunday



Hong Kong College of Radiologists

**Hong Kong College
of Radiologists 34th
Annual Scientific
Meeting**

Nov 21-22

Saturday-Sunday



Asia Cornea Society

**10th Biennial Scientific
Meeting of the Asia
Cornea Society**

Nov 27-29

Friday-Sunday



Jockey Club School of Public
Health and Primary Care, The
Chinese University of Hong Kong

**The 19th APRU Global
Health Conference
2026**

Dec 07-09

Monday-Wednesday



A0 Foundation

**A0 Spine Course-
Deformity: Advanced
Techniques and
Complex Cases with
Anatomical Specimen**

Dec 11-12

Friday-Saturday





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On-the-Pulse



Social Psychology

World Cup Sports Engagement Boosts Mental Wellbeing¹

A 2022 study of 1,201 Hong Kong adults demonstrated that engaging in physical activities either as an active participant or as a spectator during the FIFA World Cup significantly boosts individual flourishing. Guided by Basic Psychological Need Theory, the research revealed that both forms of sports involvement share a positive correlation with optimal mental wellbeing. Path analysis indicated that these beneficial relationships are partially mediated by satisfying the three innate human psychological needs for autonomy, competence, and relatedness. Even passive sports viewership functions as a self-driven behaviour that satisfies these needs, suggesting that policymakers and educators should promote both active participation and spectator events as accessible avenues for community mental health enhancement.



Psychology

Solo Trip Mental Health Benefits and Psychological Growth²

A November 2025 comprehensive review examines the psychological impacts and personal growth outcomes linked to independent travel. Utilising self-efficacy theory and identity development frameworks, the synthesis explores how navigating the world alone enhances self-confidence, emotional wellbeing, and cognitive flexibility. Key findings indicate measurable improvements in traveller resilience, self-reliance, and cross-cultural social skills, alongside notable reductions in daily stress and anxiety. Autonomous decision-making acts as a catalyst for personal transformation, value clarification, and existential meaning-making. Although navigating safety concerns and potential loneliness present challenges, independent journeys ultimately offer therapeutic opportunities for self-discovery and structural psychological advancement.



Endocrinology

Keto Diets for Weight Loss and Metabolic Health³

Obesity rates are rising globally, prompting researchers to seek effective weight loss strategies. While the ketogenic diet is historically used for epilepsy, evidence shows it offers significant weight loss advantages compared to higher carbohydrate diets. Reviewing recent clinical trials reveals that ketogenic diets effectively manage appetite by suppressing hunger hormones, resulting in natural calorie reduction without active restriction. Additionally, individuals experience rapid initial weight loss, improved glycaemic control, and reduced insulin resistance. Lowered markers of inflammation and a decreased reliance on obesity medications also occur, alongside positive psychological outcomes. Overall, adopting ketogenic diets may provide metabolic and health benefits and may be superior to other diets for weight loss while maintaining or even improving other health parameters.



Public Health

Climate Disruptions and the Global Medicine Chain⁴

Global environmental disruptions, driven by shifting weather patterns, alter disease dynamics and compromise the public health ecosystem. Rising temperatures and extreme events threaten the entire pharmaceutical infrastructure by interrupting manufacturing, damaging natural raw materials, and delaying distribution. Furthermore, heat and humidity cause the degradation of essential heat-sensitive medical products like vaccines and insulin, which reduces efficacy and potentially accelerates antimicrobial resistance through subtherapeutic dosing. Addressing these vulnerabilities requires urgent research into heat-stable therapeutics and resilient logistics systems to safeguard both human and veterinary medicine supplies during complex climate emergencies.



Otolaryngology

Earphone Hygiene Habits and Associated Auditory and Dermatological Complications⁵

A cross-sectional study examining 50 participants in Saudi Arabia evaluated how personal audio habits relate to bacterial contamination and ear health problems. Lab cultures revealed that 16% of female and 24% of male earphone samples tested positive for Gram-positive bacterial pathogens, specifically *Staphylococcus* species, though no fungal growth occurred. Over half of the participants experienced negative physical symptoms like ear pain, headaches, and itching, with statistical analysis revealing a significant connection between experiencing these symptoms and sharing devices with other people. Diagnostic audiometry testing on a subset of participants identified abnormal results in 23% of those tested, primarily indicating high-frequency sensorineural hearing issues. These clinical findings emphasise a critical need to improve public education regarding safe volumes, limited usage times, and device sanitation.



Immunology

Alcohol Consumption and Severe Nut Anaphylaxis Triggers⁶

A Canadian cross-sectional study analysing 1,135 adults evaluated how different triggers affect anaphylaxis symptoms and treatment. Food emerged as the primary cause, with peanut allergies being the most common, followed by shellfish and tree nuts. Drug-induced and venom-induced cases showed a strong correlation with hypotension and higher rates of severe reactions. Tree nut triggers linked to throat tightness and increased inpatient adrenaline use, whereas peanut triggers associated with outpatient adrenaline auto-injector. Notably, consuming alcohol served as a significant cofactor, raising the likelihood of severe reactions in undefined nut allergies.

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Hong Kong Medical Forum Marks 30th Anniversary with Record-Breaking Attendance and Expanded Global Reach

The 30th Hong Kong Medical Forum (HKMF), a flagship annual medical event organised by the Department of Medicine, School of Clinical Medicine, The University of Hong Kong and Queen Mary Hospital, Hong Kong, concluded successfully after a two-day programme (May 16-17) held at the Hong Kong Convention and Exhibition Centre (HKCEC).

To celebrate three decades of advancing healthcare knowledge, this year's forum adopted the theme "Advancing the frontiers of medicine for three decades and beyond," reflecting its longstanding commitment to promoting excellence in medical education and innovation. For the first time, the event expanded into the HKCEC Convention Hall, accommodating a record-breaking number of more than 2,200 registrations.

The Hong Kong Medical Forum has grown significantly since its inception, now featuring over 480 state-of-the-art lectures delivered by more than 700 distinguished speakers from 30 countries and regions. With a cumulative participation exceeding 45,000 healthcare professionals over the years, the forum continues to serve as a key platform for Continuing Professional Education (CPE), offering multidisciplinary insights across various medical specialties and subspecialties.

The opening ceremony was attended by Financial Secretary Paul Chan Mo-po, alongside other government officials and healthcare leaders. Their presence underscored Hong Kong's strategic ambition to strengthen its position as a global hub for medical innovation and research. Speaking at the event, speakers highlighted the importance of knowledge exchange and cross-border collaboration in tackling emerging healthcare challenges.

This year's forum brought together leading experts in clinical medicine, pharmacology, and allied healthcare fields. Attendees participated in a series of interactive workshops and in-depth discussions covering the





latest advancements in diagnostics, therapeutics, and patient care. The dynamic format allowed for active engagement, enabling practitioners to stay updated on cutting-edge developments and best practices.

In parallel with the academic programme, a medical exhibition was held to showcase the latest products and technologies in the healthcare industry. The exhibition provided a valuable interface between medical professionals and industry innovators, facilitating the exchange of ideas and the introduction of new solutions to the market.

Among the contributors to the event's success was Vital Base International, a Hong Kong-based medical communications agency with over two decades of experience in this industry. Participating as a sponsor, the agency reaffirmed its commitment to advancing medical knowledge exchange within the region and beyond. Its involvement formed part of a broader collaborative effort that supported the forum's organisation and outreach.

Vital Base International also took the opportunity to promote its flagship medical publication, V.Pulse, which provides healthcare professionals with insights into the latest medical research, industry trends, and clinical developments. The publication has become a recognised platform for disseminating knowledge within the medical community.

As the Hong Kong Medical Forum enters its fourth decade, organisers emphasised their vision to further expand its global footprint and strengthen its role as a premier hub for medical education and collaboration. With continued support from healthcare professionals, academic institutions, and industry partners, HKMF is expected to remain at the forefront of shaping the future of medicine in Hong Kong and across the region.

Highlights from the 30th Hong Kong Medical Forum

Optimising Advanced Therapy in IgA Nephropathy: Clinical Strategies and Considerations

Professor Hong Zhang

*Professor of Internal Medicine & Director of Renal Division,
Peking University First Hospital, Beijing, China*

At the 30th Hong Kong Medical Forum, one of the most compelling sessions focused on IgA nephropathy (IgAN), a leading cause of glomerulonephritis and progression to end-stage renal disease (ESRD) worldwide. The lecture, delivered by Professor Hong Zhang—Professor of Internal Medicine and Director of the Renal Division at Peking University First Hospital—highlighted the evolving landscape of IgAN management and the importance of integrating advanced therapies into clinical practice.

IgAN poses a substantial and growing clinical and socioeconomic burden, particularly in East Asia, where prevalence rates are among the highest globally. In Hong Kong, it is one of the significant contributors to end-stage kidney failure, placing increasing pressure on healthcare resources. The mechanism of IgAN is not yet fully understood; current research suggests it remains largely hypothetical. However, treatment approaches continue to evolve and show increasing effectiveness. However, clinical management remains complex, largely due to the heterogeneity of disease progression and response to treatment.

A key theme of Professor Zhang's presentation was the shift reflected in the KDIGO 2025 Clinical Practice Guideline—from reactive to proactive disease management. Historically, treatment approaches often began after the onset of significant renal damage.

However, recent guidelines have emphasised early intervention, highlighting the importance of initiating therapy at earlier stages. This proactive strategy also advocates a transition from sequential to simultaneous therapeutic approaches, targeting both the **IgAN-specific drivers** of nephron loss and the **generic responses** to chronic kidney progression.

- Central to this strategy is addressing the IgAN-specific drivers of nephron loss. Pathogenesis involves the production of pathogenic IgA and formation of immune complexes that deposit in the glomeruli, triggering inflammation and kidney injury. Therapeutic interventions therefore aim to interrupt these processes. Professor Zhang outlined several emerging and established options, including modified-release budesonide (Nefecon), B-cell activating factor (BAFF) and a proliferation-inducing ligand (APRIL) inhibitors, complement inhibitors such as iptacopan, and systemic glucocorticoids.

Among these, Nefecon has garnered particular interest. Data from the NeflgArd study showed that Nefecon significantly slows renal function decline, reduces proteinuria, and improves haematuria in both global and Chinese IgAN patient populations. Importantly, Nefecon demonstrated a more favourable safety profile, reducing systemic adverse effects compared to traditional corticosteroids. As of today, these findings represent a meaningful advance in



disease-specific therapy for IgAN, and Nefecon is so far the only endorsed corticosteroid that can significantly reduce the galactose-deficient IgA1 and IgA complex, as per the KDIGO Guideline.

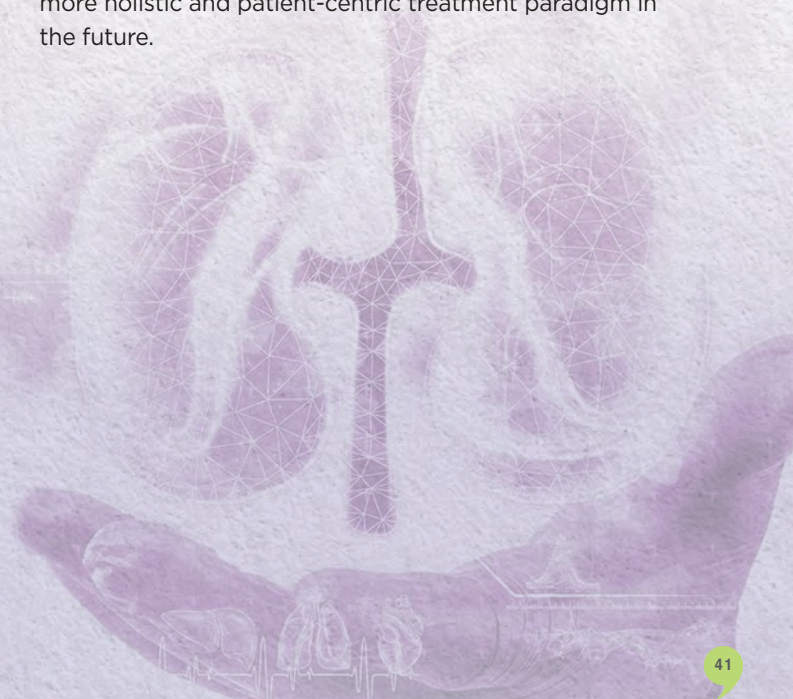
- In parallel, Professor Zhang emphasised the importance of managing the generic responses to IgAN-induced nephron loss. Even as disease-specific therapies address underlying mechanisms, supportive care remains essential to slow progression. This includes reducing glomerular hyperfiltration and mitigating the harmful effects of proteinuria on the tubulointerstitium. Blood pressure control is critical, alongside cardiovascular risk reduction and lifestyle modification.

Pharmacological interventions in this domain include renin-angiotensin system inhibitors (RASi) or dual endothelin angiotensin receptor antagonists (DEARA), often in combination with sodium-glucose co-transporter 2 inhibitors (SGLT2i). These therapies not only improve renal outcomes but also provide cardiovascular benefits, addressing the broader risk profile of patients with chronic kidney disease.

A particularly important clinical consideration highlighted in the lecture is early risk stratification and timely treatment escalation, which are key to optimising outcomes and preserving kidney function in patients with progressive IgAN. Biomarkers, clinical parameters such as proteinuria levels, and trends in renal function all contribute to risk assessment. Patients with progressive IgAN benefit most from early, intensive intervention aimed at preserving kidney function and delaying or preventing ESRD.

Professor Zhang concluded by reinforcing that optimal management of IgAN requires an integrated, patient-centred approach. Combining disease-specific therapies with supportive care measures, while tailoring treatment to individual risk profiles, offers the best chance of improving long-term outcomes. As new therapies continue to emerge, clinicians must remain vigilant in applying evidence-based strategies and adapting to evolving guidelines. For Hong Kong and mainland China, effective implementation should focus on access, treatment sequencing, and cost-effectiveness to support practical adoption in real-world care.

In summary, the session provided valuable insights into the modern management of IgAN. Advanced treatments that target the underlying causes of IgAN, combined with broader strategies addressing generic disease-response drivers, may pave the way toward a more holistic and patient-centric treatment paradigm in the future.



Neurology

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ALT: Alanine Aminotransferase CHB: Chronic Hepatitis B; eCrCl: Estimated Creatinine Clearance; ESRD: End Stage Renal Disease

*Comparison of ALT normalization, viral suppression, and impact on renal and bone function were made between tenofovir alafenamide and tenofovir disoproxil fumarate.¹⁻³



Product photo shown is not actual size.
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4. Vemlidy Prescribing Information. (Version HK-JUN22-US-SEP21).

VEMLIDY® Abbreviated Prescribing Information (Version: HK-JUN22-US-SEP21) Presentation: Tablets: 25 mg of tenofovir alafenamide - yellow, round, film-coated tablets, debossed with "GSI" on one side of the tablet and "25" on the other side. **Indications:** VEMILIDY is indicated for the treatment of chronic hepatitis B virus (HBV) infection in adults with compensated liver disease. **Dosage:** Prior to initiation of VEMILIDY, patients should be tested for HIV-1 infection. VEMILIDY alone should not be used in patients with HIV infection. **Adults:** The recommended dosage is 25 mg (one tablet) taken orally once daily with food. **Patients with Renal Impairment:** No dosage adjustment is required in patients with estimated creatinine clearance greater than or equal to 15 mL/min, or in patients with end stage renal disease (ESRD; estimated creatinine clearance below 15 mL/min) who are receiving chronic hemodialysis. On days of hemodialysis, administer VEMILIDY after completion of hemodialysis treatment. VEMILIDY is not recommended in patients with ESRD who are not receiving chronic hemodialysis. **Patients with Hepatic Impairment:** No dosage adjustment is required in patients with mild hepatic impairment (Child-Pugh A). Not recommended in patients with decompensated (Child-Pugh B or C) hepatic impairment. **Contraindications:** None. **Warnings and Precautions:** Severe acute exacerbation of Hepatitis B after discontinuation of treatment. Discontinuation of VEMILIDY may result in severe acute exacerbations of hepatitis B. Patients who discontinue VEMILIDY should be closely monitored with both clinical and laboratory follow-up for at least several months after stopping treatment. If appropriate, resumption of anti-hepatitis B therapy may be warranted. **Risk of development of HIV-1 resistance in patients coinfecting with HBV and HIV-1:** Due to the risk of development of HIV-1 resistance, VEMILIDY alone is not recommended for the treatment of HIV-1 infection. The safety and efficacy have not been established in patients coinfecting with HBV and HIV-1. HIV antibody testing should be offered to all HBV-infected patients before initiating therapy with VEMILIDY, and, if positive, an appropriate antiretroviral combination regimen that is recommended for patients coinfecting with HIV-1 should be used. **New onset or worsening renal impairment:** Prior to or when initiating VEMILIDY, and during treatment with VEMILIDY on a clinically appropriate schedule, assess serum creatinine, estimated creatinine clearance, urine glucose, and urine protein in all patients. In patients with chronic kidney disease, also assess serum phosphorus. Discontinue VEMILIDY in patients who develop clinically significant decreases in renal function or evidence of Fanconi syndrome. **Lactic acidosis/severe hepatomegaly with steatosis:** Treatment with VEMILIDY should be suspended in any patient who develops clinical or laboratory findings suggestive of lactic acidosis or pronounced hepatotoxicity (which may include hepatomegaly and steatosis even in the absence of marked transaminase elevations). **Adverse reactions:** Refer to warning and precautions for severe acute exacerbation of hepatitis B, new onset or worsening of renal impairment, and lactic acidosis/severe hepatomegaly with steatosis. Headache, abdominal pain, cough, back pain, fatigue, nausea, arthralgia, diarrhea and dyspepsia were reported in $\geq$ 5% of subjects in clinical studies. **Drug Interactions:** Anticonvulsants: carbamazepine, oxcarbazepine, phenobarbital, phenytoin. Antimycobacterial: rifabutin, rifampin, rifapentine. Herbal Products: St. John's wort. Drugs that reduce renal function or compete for active tubular secretion such as acyclovir, cidofovir, ganciclovir, valganciclovir, aminoglycosides (e.g., gentamicin), and high-dose or multiple NSAIDs. **Before prescribing, please consult full prescribing information which is available upon request.** VEMILIDY is a registered trademark of Gilead Sciences, Inc., or its related companies.

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