SLACPT NEWS

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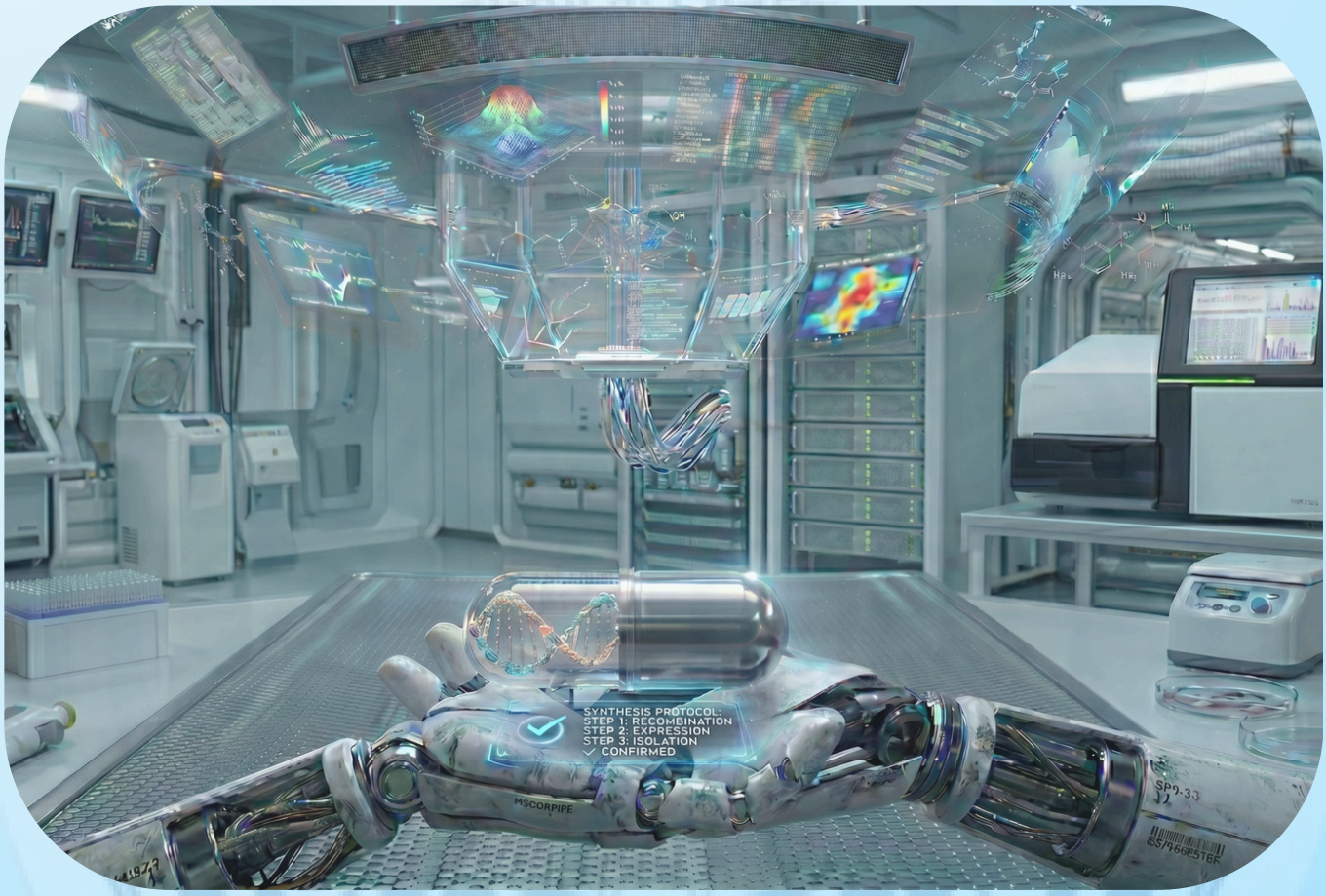

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SLACPT THEME 2025-2026

Personalized Medicine : Shaping the Future of Prescribing

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President's Message

Dear Colleagues and Friends,

The second year of the present Council's term has now completed its first quarter, and it has already been a busy and productive period for all of us.

The year commenced with a highly successful workshop on Medical Illustrations conducted by Professor Priyanga Ranasinghe, Professor in Pharmacology at the Faculty of Medicine, University of Kelaniya. The workshop effectively demonstrated how modern technology can be utilised to create impactful medical illustrations that communicate messages clearly and effectively.

We also conducted a successful webinar on Clinical Toxicology in collaboration with the Sri Lanka College of Internal Medicine. Expert resource persons discussed recent advances in the management of common poisonings encountered in Sri Lanka, making the session both timely and informative.

SLACPT is delighted to announce that Dr. Kalani Mithunika, Probationary Lecturer in Pharmacology from the Faculty of Medicine, University of Ruhuna, was awarded a fully funded scholarship by the International Union of Basic and Clinical Pharmacology (IUPHAR) to attend the World Congress of Pharmacology 2026, to be held in Melbourne, Australia.

The 3rd SLACPT Inter-Faculty Quiz for Medical Students was held virtually in May and witnessed enthusiastic participation from all faculties. It was a closely contested and exciting event, expertly conducted by Dr. Ruwnathi Jayasekera, Council Member of SLACPT.

The SLACPT Trophy was won by the team from the Faculty of Medicine, Sir John Kotelawala Defence University, while the teams from the Universities of Colombo and Peradeniya emerged as the first and second runners-up, respectively. I extend my congratulations to the winners and to all participants for contributing to a highly competitive and engaging event.

We are now preparing for our Biennial Scientific Sessions, which will be held on 6th and 7th August 2026 at the PGIM Academic Centre. The programme is included in this newsletter, and we look forward to welcoming all of you to the event.

With best wishes,

Professor Chandanie Wanigatunge
President, SLACPT

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Cover Story

Prescribing has evolved drastically from ancient mysticism and the balancing of "bodily humors" to a rigorous science. For centuries, medicine relied heavily on "eminence-based" practices, where treatments were dictated by the authority and anecdotal experience of senior physicians, often yielding inconsistent results.

A monumental shift occurred in the 20th century with the adoption of randomized clinical trials and systematic reviews, establishing Evidence-Based Prescribing (EBP). EBP replaced subjective guesswork with objective, statistically validated data.

Today, prescribing methods are highly advanced. Clinicians leverage electronic health records equipped with clinical decision support systems that automatically cross-reference patient profiles against real-time medical guidelines. Furthermore, the integration of artificial intelligence and pharmacogenomics allows doctors to predict individual metabolic responses to specific drugs based on a patient's DNA. This continuous transition from historical dogma to hyper-personalized medicine ensures unparalleled precision and safety in modern healthcare.

The Evolution of Prescribing: From Ancient Remedies to Precision Medicine

Ramindu Pathirana, Sahan Mendis
Department of Pharmacology, Faculty of Medicine, University of Ruhuna

The art and science of prescribing medicines have undergone a remarkable transformation throughout human history. What began as a practice rooted in mysticism, philosophy, and observation has evolved into one of the most evidence-driven and technologically advanced aspects of modern healthcare. Today's prescribing practices are built upon rigorous scientific research, sophisticated digital systems, and increasingly, individualized patient care. This journey reflects medicine's continuous pursuit of safer, more effective, and more precise therapeutic interventions.

Ancient Origins: Mysticism and the Theory of Humors

In ancient civilizations, illness was often believed to arise from supernatural forces, divine punishment, or imbalances within the body. Early healers and physicians relied on herbs, minerals, rituals, and spiritual practices to treat disease. One of the most influential medical concepts in history was the theory of the "four humors," developed by ancient Greek physicians such as Hippocrates and later expanded by Galen.

According to this theory, health depended on the balance of four bodily fluids: blood, phlegm, yellow bile, and black bile. Disease was thought to result from an imbalance among these humors, and treatments aimed to restore equilibrium through methods such as bloodletting, purging, dietary changes, and herbal remedies. Although many of these practices lacked scientific validity, they represented early attempts to systematically understand and manage disease.

For centuries, prescribing was largely based on tradition, personal experience, and inherited knowledge rather than objective scientific evidence.

The Era of Eminence-Based Medicine

As medical knowledge expanded through the Middle Ages and into the modern era, prescribing increasingly became dominated by "eminence-based medicine." In this model, treatments were guided primarily by the authority, reputation, and anecdotal experience of senior physicians and medical experts.

While many advances emerged during this period, clinical decisions often varied significantly between practitioners. Treatments were frequently based on intuition, personal preference, or local custom rather than standardized evidence. As a result, patients with similar illnesses could receive entirely different therapies depending on where they were treated or which physician they consulted.

This approach also allowed ineffective or even harmful treatments to persist for long periods without proper evaluation. The absence of systematic scientific testing limited the ability of clinicians to determine whether therapies truly worked or whether observed improvements were merely coincidental.

The Rise of Evidence-Based Prescribing

The 20th century marked a revolutionary turning point in the history of medicine and prescribing. The development of randomized controlled trials (RCTs), epidemiological research, and systematic scientific methodologies transformed how treatments were evaluated.

Evidence-Based Prescribing (EBP) emerged as a cornerstone of modern clinical practice. Rather than relying solely on expert opinion, EBP integrates:

- The best available scientific evidence
- Clinical expertise
- Patient values and preferences

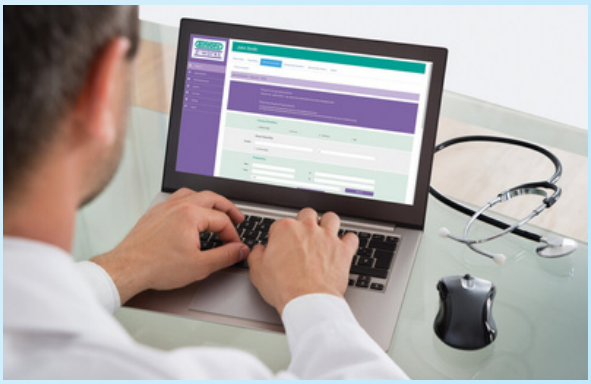
Randomized clinical trials became the gold standard for assessing the efficacy and safety of medications. Large-scale studies allowed researchers to compare treatments objectively, minimize bias, and identify adverse effects more reliably. Furthermore, systematic reviews and meta-analyses enabled clinicians to synthesize findings from multiple studies to guide practice.

The establishment of international treatment guidelines further standardized prescribing practices. Organizations and professional societies began issuing evidence-based recommendations for conditions such as hypertension, diabetes, asthma, heart failure, and infectious diseases. This dramatically improved consistency and quality of care across healthcare systems worldwide.

Evidence-based prescribing has led to substantial improvements in patient outcomes by ensuring that medications are selected based on proven benefit, safety, and cost-effectiveness.

The Digital Transformation of Prescribing

The rapid advancement of information technology has further revolutionized prescribing practices in recent decades. Electronic Health Records (EHRs) and computerized prescribing systems have replaced handwritten prescriptions in many healthcare settings, improving both efficiency and patient safety.

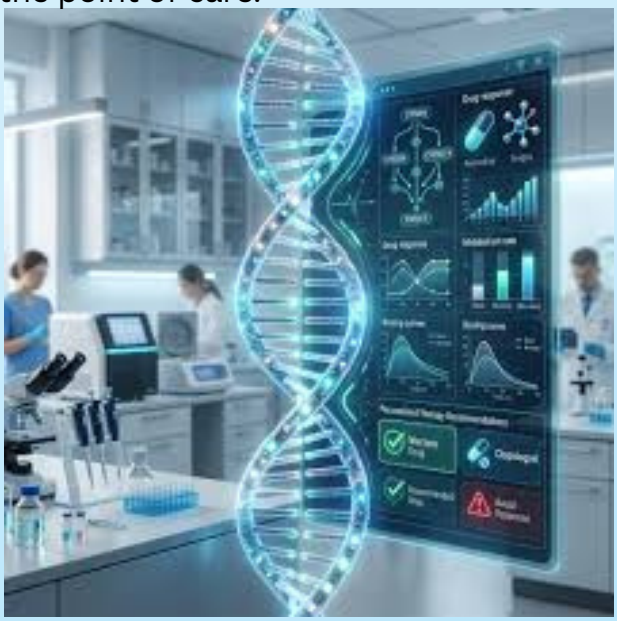


Modern clinical decision support systems can now:

- Detect drug-drug interactions
- Alert clinicians to allergies and contraindications
- Recommend dose adjustments based on renal or hepatic function
- Identify duplicate therapies
- Suggest evidence-based treatment pathways

These systems help reduce medication errors, which have historically been a major source of preventable harm in healthcare.

Additionally, access to real-time clinical guidelines and digital medical databases enables clinicians to remain updated with the latest evidence. Mobile applications and online prescribing resources have made accurate therapeutic information instantly available at the point of care.



The Emergence of Precision and Personalized Medicine

Perhaps the most exciting development in contemporary prescribing is the move toward precision medicine. Unlike traditional “one-size-fits-all” approaches, precision medicine recognizes that patients may respond differently to the same medication due to genetic, environmental, and lifestyle factors.

Pharmacogenomics: Prescribing Based on Genetics

Pharmacogenomics studies how genetic variations influence an individual’s response to drugs.

Certain genetic profiles can affect:

- Drug metabolism
- Therapeutic effectiveness
- Risk of adverse drug reactions

For example, variations in cytochrome P450 enzymes may determine whether a patient metabolizes a medication too quickly, too slowly, or normally. This knowledge allows clinicians to tailor drug selection and dosing to maximize benefit while minimizing toxicity.

Pharmacogenomic-guided therapy is already influencing prescribing in fields such as oncology, cardiology, psychiatry, and infectious diseases.

Artificial Intelligence and the Future of Prescribing

Artificial Intelligence (AI) is now beginning to shape the next frontier of prescribing. AI-driven systems can analyze vast amounts of clinical data to support therapeutic decision-making. These technologies have the potential to:

Predict individual treatment responses
Identify high-risk patients
Optimize drug combinations
Enhance medication adherence
Detect patterns of adverse effects earlier

Machine learning algorithms may eventually assist clinicians in selecting the most appropriate therapy for each patient based on millions of comparable clinical scenarios.

Although AI is unlikely to replace clinical judgment, it is increasingly becoming a powerful tool that complements physician expertise and supports safer, more effective prescribing practices.

Balancing Technology with Human-Centered Care

Despite these technological advances, the human element of prescribing remains essential. Effective prescribing is not solely about selecting the correct medication; it also involves understanding the patient's values, concerns, financial circumstances, and preferences.

Communication, empathy, shared decision-making, and clinical reasoning continue to be fundamental components of rational therapeutics. Modern medicine increasingly recognizes that the best prescribing decisions emerge from combining scientific evidence with individualized patient care.

Conclusion

The evolution of prescribing reflects the broader evolution of medicine itself—from ancient theories and anecdotal traditions to evidence-driven, technology-enhanced, and increasingly personalized healthcare.

Today's prescribing practices are safer, more precise, and more scientifically grounded than ever before. The integration of evidence-based medicine, digital health technologies, pharmacogenomics, and artificial intelligence continues to redefine therapeutic decision-making and improve patient outcomes.

As healthcare moves further into the era of precision medicine, the future of prescribing promises not only greater accuracy and safety, but also truly individualized care tailored to the unique biology and needs of every patient.

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Capacity Building Workshop Series

The SLACPT Capacity Building Workshop aims to enhance the knowledge and practice of clinical pharmacologists regarding their professional responsibilities. Third session of this series was held on the 27th February 2026 via zoom as an online session. Professor Priyanga Ranasinghe conducted an impactful session on **"AI and Modern Medical Tools for Creating Medical Illustrations".**

Prescribing During Ramadan: Key Pharmacological Challenges and Clinical Pearls

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Ramadan presents a unique and important prescribing challenge for clinicians, because fasting from dawn to sunset alters not only meal timing but also patterns of drug administration, adherence, hydration, sleep, and symptom recognition. For many patients, the issue is not simply whether a medicine can be taken during fasting, but whether its pharmacokinetic profile, dosing schedule, food-related absorption, or adverse-effect risk remains acceptable under these altered physiological and behavioural conditions.

Drugs that may ordinarily be used safely can become problematic during Ramadan because of hypoglycaemia, dehydration, postural symptoms, reduced daytime oral intake, or difficulty maintaining the required timing of administration. At the same time, many patients are highly motivated to fast and may continue treatment without seeking medical advice, placing them at risk of preventable complications. A sound understanding of clinical pharmacology is therefore essential during Ramadan, enabling physicians to individualize therapy, anticipate problems, and provide practical, culturally sensitive guidance that supports both patient safety and informed religious observance.[1,2]

Levothyroxine presents a distinctive Ramadan challenge because its efficacy depends heavily on consistent absorption. International thyroid guidance recommends that levothyroxine be taken on an empty stomach, ideally 30–60 minutes before food, or at bedtime after an adequate gap from the last meal, because food, coffee, calcium, iron, fibre, soy, and some other drugs can reduce absorption and lead to variability in TSH control.

During Ramadan, this becomes difficult because the two main meals are clustered around iftar and suhoor, and patients often struggle to find a reliable fasting window. Recent Ramadan-specific studies suggest that taking levothyroxine 30 minutes before iftar or 3–4 hours after iftar can be practical alternatives, provided patients are consistent and avoid food for the recommended interval. Even so, biochemical control may drift during Ramadan, and one recent study found a significant rise in TSH in some previously euthyroid patients after the fasting month.

The key clinical pearl is therefore not necessarily to change the dose pre-emptively, but to emphasize strict timing, separation from interacting agents, and post-Ramadan reassessment of thyroid function when clinically indicated. This is especially important in patients with thyroid cancer, pregnancy, central hypothyroidism, or those who are highly sensitive to small changes in replacement.[3–6]

Diuretics are another important group because Ramadan fasting may increase the risk of dehydration, postural hypotension, renal dysfunction, and electrolyte imbalance, particularly in hot climates, elderly patients, and those with heart failure or chronic kidney disease. Contemporary cardiovascular guidance for Ramadan advises that all fasting patients should undergo pre-Ramadan assessment and counseling regarding the risks of dehydration and the need to break the fast if they develop significant symptoms. In general, morning diuretic dosing should be avoided during fasting hours, and where possible the regimen should be shifted to non-fasting hours such as after iftar, while carefully balancing congestion risk in patients with heart failure.

A recent narrative review also advises avoiding the initiation of diuretics immediately before Ramadan where possible, because early treatment phases and dose escalation can precipitate volume depletion and intolerance during fasting. Clinicians should individualize therapy: a stable patient on a low-dose diuretic may fast safely with education and monitoring, whereas a frail patient with advanced heart failure, symptomatic hypotension, recurrent acute kidney injury, or major electrolyte disturbance may be better advised not to fast.[2,7,8]

Type 1 diabetes and insulin require especially careful discussion during Ramadan because fasting in these patients carries a real risk of hypoglycaemia, hyperglycaemia, and diabetic ketoacidosis. Current guidance emphasizes structured pre-Ramadan risk assessment, and many people with type 1 diabetes, particularly those with poor glycaemic control, recurrent hypoglycaemia, impaired awareness, recent diabetic ketoacidosis, pregnancy, or major comorbidity, should generally be advised not to fast. Nevertheless, many still choose to do so, making harm-reduction counselling essential.

Insulin should never be omitted, as interruption of exogenous insulin delivery can rapidly accelerate ketogenesis. For those who insist on fasting, insulin analogues are preferred, glucose monitoring must be intensified, and ketone monitoring should also be addressed. Basal insulin usually needs to be reduced by about 25–35% when fasting begins, while the first prandial insulin dose taken with iftar often requires a reduction of about 35–50%, with later titration guided by glucose values.

Patients with type 1 diabetes should ideally perform blood glucose monitoring at least 3–4 times daily or use continuous glucose monitoring, and they must be clearly instructed to break the fast if hypoglycaemia, marked hyperglycaemia, or symptoms of ketosis occur.[1,9–11]

Type 2 diabetes and insulin present a different but equally important prescribing challenge. Many patients with type 2 diabetes can fast safely with careful preparation, but insulin therapy substantially increases the risk of daytime hypoglycaemia, especially if usual pre-Ramadan doses are continued despite reduced daytime caloric intake. Guidance therefore recommends formal assessment before Ramadan, individualized dose adjustment, and reinforcement of self-monitoring. If glycaemic control is otherwise stable, the basal insulin dose is typically reduced by around 25–35% once fasting begins. Long-acting basal insulin such as U-100 glargine is usually timed just before iftar, whereas ultra-long-acting preparations such as degludec or U-300 glargine may not require a change in administration time.

For patients using prandial insulin, the first meal at iftar often requires a dose reduction of around 35–50%, while later doses can be adjusted according to meal content and glucose readings. In people with type 2 diabetes, the frequency of glucose monitoring should reflect the estimated fasting risk, with high-risk individuals checking at least 2–4 times daily. The key pharmacological principle is that Ramadan is not simply a matter of shifting insulin timing; it requires active matching of insulin pharmacokinetics to altered feeding patterns, close monitoring, and readiness to modify doses promptly.[1,9,11,12]

Sulfonylureas deserve separate attention during Ramadan because, as a class, they remain associated with fasting-related hypoglycaemia, particularly when usual doses are continued despite prolonged daytime caloric restriction. Current guidance recommends that in patients who are already at glycaemic target, the sulfonylurea dose should be empirically reduced during fasting, with further titration guided by glucose monitoring.

Among the sulfonylureas, gliclazide and glimepiride appear to be better tolerated and carry a lower risk of hypoglycaemia than longer-acting agents such as glibenclamide, making them more suitable choices when sulfonylurea therapy is continued during Ramadan. Gliclazide modified release is a practical option, as observational data suggest that it may be associated with low hypoglycaemia rates among sulfonylureas during Ramadan fasting. Nevertheless, sulfonylureas should still be used cautiously, because their insulin-secretagogue effect persists even when the patient is not eating during the day. The key clinical message is that gliclazide may be one of the safer sulfonylurea options during Ramadan, but it is not risk-free, and patients require dose adjustment, education, and regular glucose monitoring.[1,11,12]

Steroids in adrenal insufficiency are particularly important because this is one situation in which fasting may become genuinely unsafe. Patients with primary or secondary adrenal insufficiency depend on exogenous glucocorticoid replacement, and fasting can increase the risk of undertreatment, hypotension, fatigue, hypoglycaemia, and adrenal crisis, both because oral medication timing is restricted and because fasting itself may impose mild physiological stress.

Practical international guidance recommends that these patients undergo assessment before Ramadan, understand sick-day rules, and be clearly advised that the fast must be broken immediately if symptoms of cortisol deficiency develop. A major pharmacological challenge is that standard short-acting hydrocortisone regimens are designed to mimic circadian cortisol secretion and may not fit well with Ramadan meal timings, leading to prolonged dosing intervals during the daytime fast. In clinical practice, some patients on thrice-daily hydrocortisone such as 10 mg/5 mg/5 mg may have their regimen simplified to twice-daily dosing, for example 15 mg at suhoor and 5 mg at iftar, to reduce the daytime gap; however, this should be individualized.

Published Ramadan guidance more strongly supports switching suitable patients from short-acting hydrocortisone to prednisolone 5 mg once daily before the fast, with the dose taken immediately before dawn or suhoor, to provide more sustained daytime glucocorticoid coverage and avoid the risks associated with extended hydrocortisone dosing intervals. Patients should also receive counselling on adequate hydration, sick-day rules, when to terminate the fast urgently, and when to administer emergency parenteral glucocorticoid. Those at high risk of complications should be advised not to fast, while patients with primary adrenal insufficiency can usually continue their usual dose of fludrocortisone, taken once daily before the fast. The overriding priority should always be prevention of adrenal crisis rather than strict continuation of fasting at all costs.[13,14]

In conclusion, prescribing during Ramadan requires more than simple dose rescheduling; it demands an appreciation of how fasting alters drug absorption, glycaemic risk, hydration status, and the safety margins of essential therapies. While many patients can fast safely with appropriate planning, others, particularly those with type 1 diabetes, insulin-treated diabetes at high risk of hypoglycaemia, or adrenal insufficiency, require careful counselling. The clinician’s role is therefore to undertake pre-Ramadan risk assessment, individualize drug regimens, anticipate predictable complications, and reinforce practical measures such as glucose monitoring, correct levothyroxine timing, hydration strategies, and sick-day rules. A culturally sensitive but medically sound approach helps patients observe Ramadan as safely as possible while minimizing preventable harm.

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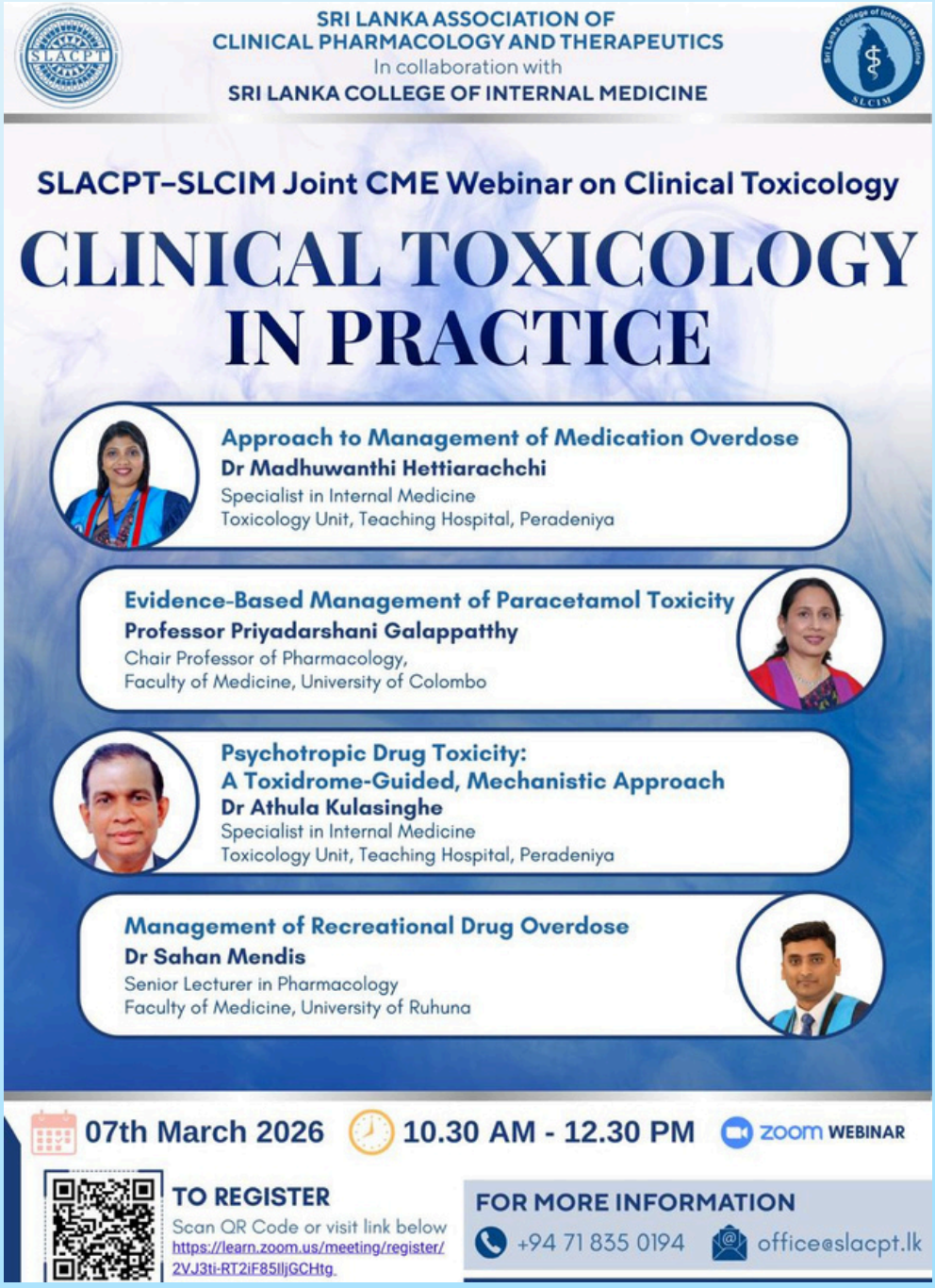
SLACPT/SLCIM Joint CME Webinar on Clinical Toxicology

The Sri Lanka Association of Clinical Pharmacology and Therapeutics (SLACPT), in collaboration with the Sri Lanka College of Internal Medicine (SLCIM), successfully conducted a Joint CME Webinar on Clinical Toxicology titled **"Clinical Toxicology in Practice"** on 7th March 2026 via Zoom.

The webinar featured a distinguished panel of experts who delivered evidence-based and clinically relevant updates on the management of common toxicological emergencies encountered in medical practice.

Sessions included the management of medication overdose, evidence-based management of paracetamol toxicity, psychotropic drug toxicity using a toxidrome-guided mechanistic approach, and the management of recreational drug overdose.



Drug Information Summary

Rimegepant

Rimegepant is a novel oral medication used for the treatment and prevention of migraine. It belongs to a class of drugs known as calcitonin gene-related peptide (CGRP) receptor antagonists, commonly called “gepants.” CGRP is a neuropeptide that plays a major role in the pathophysiology of migraine by promoting neurogenic inflammation, vasodilation, and pain transmission within the trigeminovascular system. By blocking CGRP receptors, rimegepant helps relieve migraine symptoms and reduce the frequency of attacks.

Mechanism of action

Rimegepant acts by selectively binding to and blocking the calcitonin gene-related peptide (CGRP) receptor, which plays a key role in the development of migraine. By antagonizing CGRP receptor activity, rimegepant helps reduce migraine symptoms, although the exact relationship between this mechanism and its full clinical effects is not completely understood.

The efficacy of rimegepant for the acute treatment of migraine, with or without aura, was evaluated in three randomized, double-blind, placebo-controlled clinical trials. Adult patients with moderate to severe migraine attacks were treated with a single 75 mg dose. Rescue medications such as NSAIDs, paracetamol, or antiemetics were allowed after 2 hours if needed, but triptans were prohibited within 48 hours.

Pharmacokinetic properties

Rimegepant is well absorbed after oral administration, reaching peak plasma concentration approximately 1.5 hours after dosing. Its oral bioavailability is about 64% following a 300 mg dose. Food delays absorption by around 1–1.5 hours and reduces overall drug exposure, although clinical studies showed it can be taken without regard to meals.

Rimegepant has a large volume of distribution (120 L) and is highly protein bound (about 96%). It is primarily metabolized in the liver by CYP3A4 and, to a lesser extent, by CYP2C9. Most circulating drug remains unchanged, with no major active metabolites identified. Rimegepant has minimal effects on most cytochrome enzymes, although it acts as a weak inhibitor of CYP3A4.

The elimination half-life is approximately 11 hours. Drug excretion occurs mainly through feces (78%) and urine (24%), with a significant proportion eliminated unchanged. Rimegepant is also a substrate of P-glycoprotein (P-gp) and BCRP transporters, meaning inhibitors of these transporters may increase plasma drug concentrations.

In patients with mild to moderate renal or hepatic impairment, exposure changes were not clinically significant. However, drug exposure increased substantially in severe renal impairment and severe hepatic impairment (Child-Pugh C), indicating caution or avoidance in these populations. Rimegepant has not been adequately studied in patients with end-stage renal disease.

Posology and method of administration

Acute treatment of migraine The recommended dose is 75 mg rimegepant, as needed, once daily.
Prophylaxis of migraine The recommended dose is 75 mg rimegepant every other day.

The maximum dose per day is 75 mg rimegepant which can be taken with or without meals.
Concomitant medicinal products
Another dose of rimegepant should be avoided within 48 hours when it is concomitantly administered with moderate inhibitors of CYP3A4 or with strong inhibitors of P-gp

Special populations
Elderly (aged 65 and over) - There is limited experience with rimegepant in patients aged 65 years or older. No dose adjustment is required as the pharmacokinetics of rimegepant are not affected by age (see section 5.2).

Renal impairment - No dose adjustment is required in patients with mild, moderate, or severe renal impairment. Severe renal impairment resulted in a > 2-fold increase in unbound AUC but less than a 50% increase in total AUC.
Caution should be exercised during frequent use in patients with severe renal impairment. Rimegepant has not been studied in patients with end-stage renal disease and in patients on dialysis. Use of rimegepant in patients with end-stage renal disease (CLcr < 15 ml/min) should be avoided.

Hepatic impairment - No dose adjustment is required in patients with mild (Child-Pugh A) or moderate (Child-Pugh B) hepatic impairment. Plasma concentrations (unbound AUC) of rimegepant were significantly higher in subjects with severe (Child-Pugh C) hepatic impairment. The use of rimegepant in patients with severe hepatic impairment should be avoided.

Paediatric population - The safety and efficacy of rimegepant in paediatric patients (< 18 years of age) have not been established. No data are available.

Method of administration
This drug is for oral use. The oral rimegepant should be placed on the tongue or under the tongue. It will disintegrate in the mouth and can be taken without liquid.

Patients should be advised to use dry hands when opening the blister.

Contraindications
Hypersensitivity to the active substance or to any of the excipients

Special warnings and precautions for use
Hypersensitivity reactions, including dyspnoea and rash, have occurred in less than 1% of patients treated with rimegepant in clinical studies.
Hypersensitivity reactions, including serious hypersensitivity such as anaphylactic reaction, have been reported in the clinical and post-marketing settings. Some hypersensitivity reactions can occur days after administration. If a hypersensitivity reaction occurs, rimegepant should be discontinued and appropriate therapy should be initiated.
Rimegepant is not recommended: - in patients with severe hepatic impairment, in patients with end-stage renal disease (CLcr < 15 ml/min), for concomitant use with strong inhibitors of CYP3A4, for concomitant use with strong or moderate inducers of CYP3A4.

Medication overuse headache (MOH) Overuse of any type of medicinal products for headaches can make them worse. If this situation is experienced or suspected, medical advice should be obtained, and treatment should be discontinued. The diagnosis of MOH should be suspected in patients who have frequent or daily headaches despite (or because of) the regular use of medicinal products for acute headache.

Interaction with other medicinal products and other forms of interaction
Rimegepant is a substrate of CYP3A4, P-glycoprotein (P-gp) and breast cancer resistance protein (BCRP) efflux transporters.

Inhibitors of CYP3A4 increase plasma concentrations of rimegepant. Concomitant administration of rimegepant with strong CYP3A4 inhibitors (e.g., clarithromycin, itraconazole, ritonavir) is not recommended. Concomitant administration of rimegepant with itraconazole resulted in a significant increase in rimegepant exposure (AUC by 4-fold and Cmax 1.5-fold). Concomitant administration of rimegepant with medicinal products that moderately inhibit CYP3A4 (e.g., diltiazem, erythromycin, fluconazole) may increase exposure to rimegepant. Concomitant administration of rimegepant with fluconazole resulted in increased exposures of rimegepant (AUC by 1.8-fold) with no relevant effect on Cmax. Another dose of rimegepant within 48 hours should be avoided when it is concomitantly administered with moderate inhibitors of CYP3A4 (e.g., fluconazole).

CYP3A4 inducers Inducers of CYP3A4 decrease plasma concentrations of rimegepant. Concomitant administration of VYDURA with strong CYP3A4 inducers (e.g., phenobarbital, rifampicin, St John's wort (Hypericum perforatum)) or moderate CYP3A4 inducers (e.g., bosentan, efavirenz, modafinil) is not recommended. The effect of CYP3A4 induction may last for up to 2 weeks after discontinuation of the strong or moderate CYP3A4 inducer.

Concomitant administration of rimegepant with rifampicin resulted in a significant decrease (AUC reduced by 80% and Cmax by 64%) in rimegepant exposure, which may lead to loss of efficacy. P-gp and BCRP only inhibitors Inhibitors of P-gp and BCRP efflux transporters may increase plasma concentrations of rimegepant.

Another dose of rimegepant within 48 hours should be avoided when it is concomitantly administered with strong inhibitors of P-gp (e.g., cyclosporine, verapamil, quinidine). Concomitant administration of rimegepant with cyclosporine (a potent P-gp and BCRP inhibitor) or with quinidine (a selective P-gp inhibitor) resulted in a significant increase of similar magnitude in rimegepant exposure (AUC and Cmax by > 50%, but less than two-fold).

Fertility, Pregnancy and Lactation

Pregnancy - There are limited data from the use of rimegepant in pregnant women. Animal studies demonstrate that rimegepant is not embryocidal, and no teratogenic potential has been observed at clinically relevant exposures. Adverse effects on embryo-foetal development (decreased foetal body weight and increased skeletal variations in rats) were only observed at exposure levels associated with maternal toxicity (approximately 200 times greater than clinical exposures) following administration of rimegepant during pregnancy. As a precautionary measure, it is preferable to avoid the use of rimegepant during pregnancy.

Breast-feeding - In a single center study of 12 breast-feeding women treated with a single dose of rimegepant 75 mg, minimal concentrations of rimegepant were observed in breast milk. The relative percentage of a maternal dose estimated to reach the infant is less than 1%. There are no data on the effects on milk production. The developmental and health benefits of breast-feeding should be considered along with the mother's clinical need for rimegepant and any potential adverse reactions on the breastfed infant from rimegepant or from the underlying maternal condition.

Fertility - Animal studies showed no clinically relevant impact on female and male fertility.

Adverse Drug Reactions

Rimegepant is generally well tolerated, with most adverse effects being mild to moderate in severity. The most commonly reported adverse reaction in both acute migraine treatment and prophylactic use is nausea, occurring in approximately 1–1.4% of patients. Hypersensitivity reactions, including dyspnoea and severe rash, have been reported in less than 1% of treated patients. Rare cases of anaphylactic reactions have also been observed, particularly during post-marketing surveillance. Importantly, hypersensitivity reactions may occur immediately or be delayed by several days after administration.

Long-term safety studies extending up to one year demonstrated an overall favourable safety profile, with more than 1,600 patients exposed for at least 6 months and 740 patients treated for 12 months. Ongoing pharmacovigilance and adverse event reporting remain important to monitor the long-term benefit-risk balance of the drug.

Overdose

Clinical experience with overdose of Rimegepant is currently limited, and no characteristic overdose symptoms have been specifically identified. Management of overdose is mainly supportive and includes monitoring vital signs, clinical observation, and symptomatic treatment as required.

There is no specific antidote available for rimegepant overdose. Due to its high plasma protein binding, the drug is unlikely to be effectively removed by dialysis. Therefore, treatment focuses on supportive care until the patient recovers clinically.

Source:

Summary of Product Characteristics of Rimegepant, European Medicines Agency (EMA)

Detailed information of this medicine is available on the European Medicines Agency web site: https://www.ema.europa.eu/.

Drug Information Summary

Resmetirom

Resmetirom is indicated in conjunction with diet and exercise for the treatment of adults with noncirrhotic metabolic dysfunction-associated steatohepatitis (MASH) with moderate to advanced liver fibrosis (fibrosis stages F2 to F3).

Mechanism of Action

Resmetirom is a liver-directed partial agonist for the thyroid hormone receptor beta (THR-β). Resmetirom produced 83.8% of the maximum response compared to triiodothyronine (T3), with an EC50 of 0.21 μM in an in vitro functional assay for THR-β activation. The same functional assay for thyroid hormone receptor alpha (THR-α) agonism showed 48.6% efficacy for resmetirom relative to T3, with an EC50 of 3.74 μM. THR-β is the predominant form of THR in the liver. Stimulation of THR-β in the liver improves mitochondrial function and lipid metabolism, and increases fatty acid β oxidation, thereby reducing lipotoxic liver fat, inflammation and liver fibrosis. Resmetirom's liver 7 directed THR-β agonism is particularly relevant in the treatment of MASH and leads to minimal off target activity on THR-α in tissues such as heart and bone.

Pharmacodynamic effects

- Liver fat content Resmetirom decreases liver fat content as measured by magnetic resonance imaging-proton density fat fraction (MRI-PDFF) or FibroScan controlled attenuation parameter (CAP).
- Reductions in liver fat content by MRI-PDFF were observed at 16 (the first assessment) and 52 weeks of treatment.
- Reductions in liver fat content by CAP were observed at 52 weeks of treatment. Reductions in lipids Resmetirom reduces blood low density lipoprotein (LDL) cholesterol, apolipoprotein B, lipoprotein (a) and triglyceride levels.
- Reductions in all lipid endpoints were observed initially after 4 weeks (the first assessment) and sustained at 24 and through 52 weeks of treatment. Prohormone FT4 Decreased concentrations of prohormone FT4 were observed at the first assessment at 4 weeks of treatment. Similar decreases in FT4 were observed during treatment.
- Sex hormone binding globulin (SHBG) Resmetirom increased concentrations of sex hormone binding globulin (SHBG) at the first assessment at 4 weeks of treatment and at longer durations of treatment; by week 52, SHBG increased from baseline 145% (95% CI: 128, 160%) for 80 mg, 205% (95% CI: 182, 229%) for 100 mg and -0.4% (95% CI: -4, 2%) for placebo.
- No known adverse reactions were associated with SHBG elevations.
- Cardiac electrophysiology At a dose of 200 mg given for 7 days, resmetirom did not prolong the QT interval, PR interval, QRS interval, or alter heart rate in a study in healthy subjects.

Pharmacokinetic Properties

Resmetirom demonstrates dose-proportional pharmacokinetics at therapeutic doses, with steady-state concentrations achieved within 3–6 days of once-daily dosing. Peak plasma concentrations occur approximately 4 hours after administration. Food has no clinically significant effect on overall bioavailability, although high-fat meals reduce peak concentration and delay absorption slightly.

Resmetirom is highly protein bound (>99%) and has a volume of distribution influenced by body weight. It is partially metabolized by CYP2C8 and is a substrate of transporters including OATP1B1, OATP1B3, and BCRP. Two metabolites are formed, including MGL-3623, which has substantially lower pharmacological activity and does not accumulate with repeated dosing.

The drug has a relatively short terminal half-life of about 4.5 hours and is primarily cleared through non-renal pathways. Renal excretion plays only a minor role in elimination.

In patients with mild hepatic impairment, pharmacokinetics are largely unchanged and dose adjustment is not required. However, moderate and severe hepatic impairment significantly increase drug exposure, and use should be avoided in patients with decompensated cirrhosis. The safety and efficacy of resmetirom have not yet been established in patients with MASH cirrhosis.

Renal impairment has minimal clinical impact on resmetirom pharmacokinetics. Even in severe renal impairment, increases in drug exposure were modest and not associated with clinically meaningful safety concerns or pharmacodynamic changes; therefore, dose adjustment is generally unnecessary.

Pharmacokinetics are not significantly influenced by age, sex, or race, although higher body weight is associated with faster drug clearance.

Posology and Method of Administration

The posology is based on the patient's body weight:

- For patients weighing < 100 kg, the recommended dose is 80 mg taken orally once daily.
- For patients weighing ≥ 100 kg, the recommended dose is 100 mg taken orally once daily.

Missed or delayed doses If a dose of resmetirom is missed, the patient should take the next dose at the scheduled time. A double dose should not be taken to make up for the missed dose.

Method of administration

Oral use - Resmetirom may be taken with or without food

Special populations

Hepatic impairment - Mild hepatic impairment No dose adjustment is required in patients with mild hepatic impairment (Child-Pugh A). Safety and efficacy have not been demonstrated in Child-Pugh A cirrhosis. Moderate or severe hepatic impairment Increased exposure (Cmax and AUC) of resmetirom has been observed in patients with moderate or severe hepatic impairment. Resmetirom should not be used in patients with moderate or severe (Child-Pugh B or C) hepatic impairment.

Renal impairment - Mild renal impairment (eGFR 60 to 89 mL/min), moderate renal impairment (eGFR 30 to 59 mL/min) and severe renal impairment (eGFR 15 to 29 mL/min).
No dose adjustment of resmetirom is recommended in patients with mild, moderate or severe renal impairment. In moderate or severe renal impairment the increase in exposure is within the expected variability of exposure.

Elderly - No dose adjustment is required in patients aged ≥ 65 years.

Paediatric population - The safety and efficacy of resmetirom in children and adolescents less than 18 years old have not yet been established. No data are available.

Contraindications
Hypersensitivity to the active substance or to any of the excipients

Special warnings and precautions for use
Gallbladder events - In clinical studies, cholecystitis was observed more often in resmetirom-treated patients than in placebo-treated patients. If cholelithiasis is suspected, gallbladder diagnostic studies and appropriate clinical follow-up are indicated.

Use in other liver diseases
Resmetirom has not been studied in patients with other underlying liver diseases. Resmetirom should be used with caution in MASH patients with other underlying liver diseases such as autoimmune liver diseases or active viral hepatitis. Resmetirom should be used with caution in patients with alcohol related liver disease. Liver enzymes Conduct liver enzyme monitoring periodically during treatment.
If hepatotoxicity is suspected, discontinue resmetirom treatment and continue to monitor liver chemistry.

Interaction with other medicinal products and other forms of interaction
Resmetirom is partially metabolised by CYP2C8.
Effects of other medicinal products on resmetirom CYP2C8 inhibitors
Clopidogrel, a moderate CYP2C8 inhibitor, increased resmetirom exposure (1.3-fold in Cmax and 1.7 fold in AUC) in healthy adult subjects. Dose adjustment of resmetirom is recommended when used concomitantly with moderate CYP2C8 inhibitors. As greater increases are expected with strong inhibitors (e.g. gemfibrozil), their use is not recommended.

HMG-CoA reductase inhibitors (statins) - The dose of rosuvastatin and simvastatin should be limited to a daily dose of 20 mg and the dose of pravastatin and atorvastatin should be limited to a daily dose of 40 mg.

Warfarin In healthy subjects with stable INR, administration of resmetirom (100 mg/day) for multiple days had minimal effect on the AUC and Cmax of R- and S-warfarin. No dose adjustment is required.

Fertility, Pregnancy and Lactation

Pregnancy - There are no data from the use of resmetirom in pregnant women. Animal studies do not indicate direct or indirect harmful effects with respect to reproductive toxicity. As a precautionary measure, it is preferable to avoid the use of resmetirom during pregnancy.

Breast-feeding - It is unknown whether resmetirom and/or its metabolites are excreted in human milk. A risk to newborns/infants cannot be excluded. A decision must be made whether to discontinue breast-feeding or to abstain from resmetirom therapy, taking into account the benefit of breast-feeding for the child and the benefit of therapy for the woman.

Fertility - No fertility data are available in humans. Animal studies do not indicate any direct or indirect effects on fertility

Adverse Drug Reactions

The most frequently reported adverse reactions are diarrhoea, nausea, and pruritus. Diarrhoea typically occurred at treatment initiation, was mild to moderate and self-limiting, resolving on average in 2 to 3 weeks. The median time to complete resolution of diarrhoea was 3 to 4 weeks. Nausea occurred at treatment initiation and was mild to moderate, occurring more commonly in female patients.
Other rare events included cholecystitis and urticaria.

Treatment was associated with mild transient elevations in liver enzymes (ALT and AST), particularly during the first 4 weeks of therapy and more commonly among patients receiving concomitant statin therapy. These increases were generally less than 1.5 times baseline values and returned to baseline by approximately 8 weeks after treatment initiation.
Resmetirom also caused modest reductions in free T4 levels over 12 months, particularly with higher doses, while T3 and TSH levels remained largely unchanged. Importantly, these laboratory changes were not associated with clinically significant thyroid-related symptoms or findings.

Overdose

In clinical studies in healthy subjects, the highest dose of resmetirom tested was 200 mg once daily for up to 14 days, without reports of any additional adverse reactions. If overdose occurs the patient should be monitored for signs or symptoms of adverse reactions, and supportive care should be provided as needed. The medicinal product is unlikely to be removed by haemodialysis due to high protein binding.

Source:

Summary of Product Characteristics of Resmetirom, European Medicines Agency (EMA)
Detailed information of this medicine is available on the European Medicines Agency web site: https://www.ema.europa.eu/.

SLACPT MCQ Course in Pharmacology for MD Selection Examinations

SLACPT MCQ course targeting the MD selection examinations was meticulously organised by Dr Asanka Eriyawa and Dr Supun Wedasinghe, which was held on the 14th and 21st of March 2026 as an online programme.

SLACPT members contributed as resource persons in this two day MCQ programme.

MCQ Course Targeting MD Selection Exams Organized by the

SRI LANKA ASSOCIATION OF CLINICAL PHARMACOLOGY & THERAPEUTICS (SLACPT)

14th & 21st March 2026

14th March 2026		
09.00 – 10.30 am	Basic Pharmacology	Prof Pradeepa Jayawardane
10.45 AM – 12.15 PM	Respiratory and Autonomic Nervous System Pharmacology	Dr Ruwanthi Jayasekara
01.00 PM – 02.30 PM	Antidiabetic Agents and other Drugs in Endocrine Disorders	Dr Gayana Amiyangoda
02.45 PM - 04.15 PM	Pharmacology in Special Populations	Prof Shalini Sri Ranganathan
21st March 2026		
09.00 AM – 10.30 AM	Prescribing in Rheumatology	Dr Sujeevani Kurukulasuriya
10.45 AM – 12.15 PM	Pharmacology of Anti Infectives	Dr Sahan Mendis
01.00 PM – 02.30 PM	Cardiovascular Pharmacology	Prof Priyanga Ranasinghe
02.45 PM - 04.15 PM	Pharmacology of Gastrointestinal and liver diseases	Prof Anuradha Dissanayake

Actue Pancreatitis: Adverse Drug Reaction of GLP 1 Agonsits

Key Points

GLP-1 receptor agonists are an increasingly important class of medications used in the management of type 2 diabetes mellitus and obesity. Examples include Dulaglutide, Liraglutide, Semaglutide, and Tirzepatide. Although generally safe and effective, these agents have been associated, in rare cases, with acute pancreatitis.

Acute pancreatitis typically presents with:

- Severe upper abdominal pain radiating to the back
- Nausea and vomiting
- Fever
- Abdominal tenderness

Patients experiencing these symptoms while on GLP-1 agonist therapy should seek urgent medical assessment.

When Does It Occur?

Reported cases suggest that pancreatitis usually develops between one month and one year after starting therapy, although onset may range from a few days to several years after initiation.

Who Is at Higher Risk?

The risk appears to be greater in patients with pre-existing risk factors, including:

- Previous history of pancreatitis
- Gallstones or biliary disease
- Smoking
- Coronary artery disease or other vascular diseases
- Obesity and type 2 diabetes themselves, which independently increase pancreatitis risk

Current evidence suggests that in patients without risk factors, GLP-1 agonists may not significantly increase the risk of pancreatitis.

Management Recommendations

If pancreatitis develops during treatment:

The GLP-1 agonist should be discontinued immediately.

Rechallenge with the same or another GLP-1 agonist is generally not recommended.

Recurrence of pancreatitis has been reported after restarting therapy.

Importantly, available evidence does not show a meaningful difference in pancreatitis risk among the different GLP-1 agonists.

Possible Mechanisms

The exact mechanism by which GLP-1 agonists may contribute to pancreatitis remains uncertain.

Proposed explanations include:

- Rapid weight loss leading to gallstone formation, similar to what is observed after bariatric surgery
- Excessive stimulation of GLP-1 receptors causing pancreatic tissue overactivity and inflammation

However, current studies do not demonstrate that GLP-1 agonists cause chronic pancreatitis.

Clinical Take-Home Message

GLP-1 receptor agonists remain valuable therapies for diabetes and obesity management, offering substantial metabolic and cardiovascular benefits. Nevertheless, clinicians should remain vigilant for symptoms suggestive of acute pancreatitis, especially in patients with established risk factors. Early recognition, prompt discontinuation of the medication, and appropriate medical evaluation are essential to minimize complications.

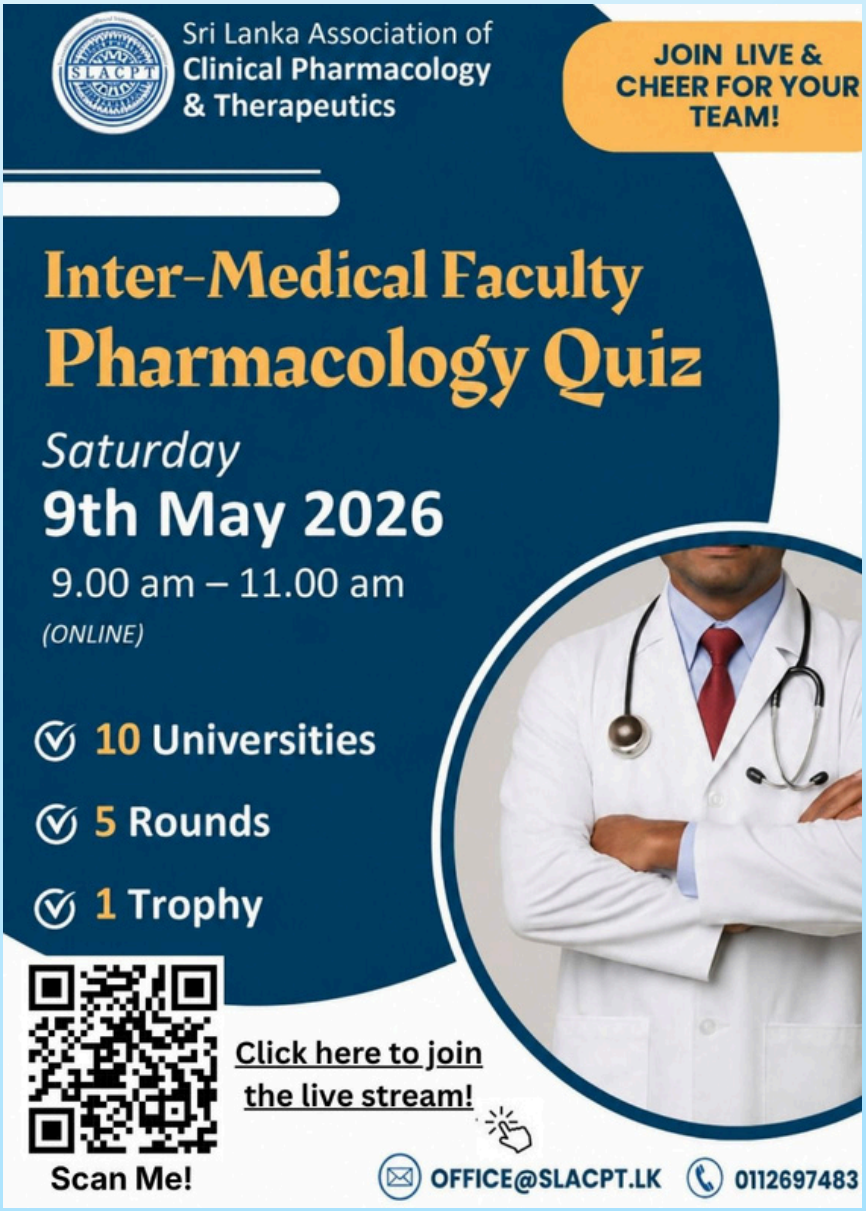
Reference

Dutch Pharmacovigilance Centre Lareb. Pancreatitis and GLP-1 agonists (Alvleesklierontsteking en GLP1 agonisten). Netherlands Pharmacovigilance Information Service. Available at: Lareb Official Website

SLACPT Inter-Medical Faculty Pharmacology Quiz 2026

The Sri Lanka Association of Clinical Pharmacology and Therapeutics (SLACPT) successfully conducted the Inter-Medical Faculty Pharmacology Quiz 2026 on 9th May 2026 as an online academic competition bringing together medical undergraduates from universities across Sri Lanka. The event featured participation from 10 medical faculties and consisted of five competitive rounds designed to assess and strengthen knowledge in clinical pharmacology and therapeutics.

The quiz created an engaging platform for undergraduate medical students to enhance academic collaboration, critical thinking, and enthusiasm towards pharmacology. The event also provided an opportunity for students from different universities to interact in a spirit of healthy competition and academic excellence. SLACPT extends its appreciation to Dr Ruwanthi Jayasekera, the coordinator and the quiz master who meticulously organised and conducted the quiz, all participating universities, organizing committee members, moderators, and students who contributed to the success of this national academic event.



Winners

Sir John Kotalawela Defence University

1st Runners-Up

University of Colombo

2nd Runners-Up

University of Peradeniya

"ClinPharm 2026" - SLACPT Scientific Sessions

"ClinPharm 2026" - SLACPT Scientific Sessions Programme

SLACPT upcoming events

SLACPT/SLCP Joint CME Webinar – July 2026

Next SLACPT joint CME webinar will be conducted in collaboration with Sri Lanka College of Paediatricians.

Wall of Fame

Professor Sujeevani Kurukulasuriya

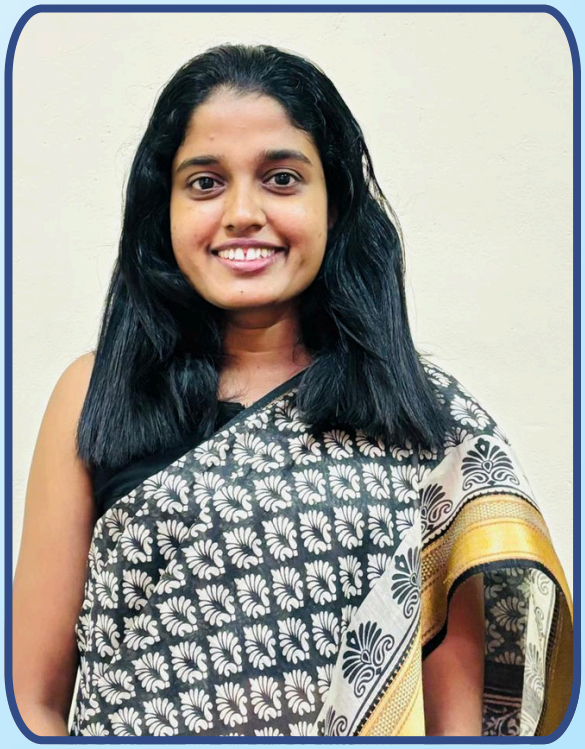
Senior Lecturer in the Department of Pharmacology, Faculty of Medicine, University of Kelaniya has been promoted to Professor in Pharmacology

Professor Roshini Murugupillai

Senior Lecturer in the Department of Clinical Sciences, Faculty of Health-care Sciences, Eastern University of Sri Lanka has been promoted to Professor in Pharmacology

Wall of Fame

Dr. Kalani Mithunika
Lecturer in Pharmacology, Faculty of Medicine, University of Ruhuna was awarded the IUPHAR scholarship to attend the 20th World Congress of Basic and Clinical Pharmacology (WCP 2026) in Melbourne, Australia, representing the SLACPT.
Dr. Mithunika will be presenting a poster titled “ST2 (IL-33 receptor): a potential host-directed therapeutic target in severe leptospirosis” at the WCP 2026.



The President, the Council and the members of the Sri Lanka Association of Clinical Pharmacology and Therapeutics congratulate and rejoice in the achievements of Professor Sujeevani Kurukulasuriya, Professor Roshini Murugupillai and Dr Kalani Mithunika and wish them all success.

SLACPT NEWS

The Official Newsletter of
the Sri Lanka Association of
Clinical Pharmacology and Therapeutics

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Write to Us!

SLACPT welcomes suggestions from readers towards improving the image of the Association and the newsletter.

Please send your suggestions to:
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