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Rx FACTOR

Newsletter by

National Association of Pharmacology & Therapeutics

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National Association of
Pharmacology & Therapeutics

Happy

Independence Day

15th August 2026

On the mark of this 80th Independence day we are proudly committed to redefining the role of medical pharmacologists in building a healthy nation

National Association of Pharmacology & Therapeutics

Promoting Pharmacology & Therapeutics for a better tomorrow

HIGHLIGHTS

01 **Academic Corner**
Current Therapeutics, New Drugs, Banned Drugs, Integrated Approach to Therapeutics

02 **Research Corner**
Trends in Current Research, Areas of Research for UG & PG, Innovations & Techniques in Research

03 **Vigilant Corner**
Adverse Drug Reaction Updates, Widening the Horizon of Safe Therapeutics

04 **Medical education Corner**
Competency Building, Skill Development, OSPE, New Teaching & Learning Methods

05 **Ethics & Regulations**
Current Updates from Regulatory Bodies

06 **Current affairs**
Latest medical news

07 **Cool corner**
Mini quiz, Puzzle, Cartoons, Mnemonics, images

Editorial

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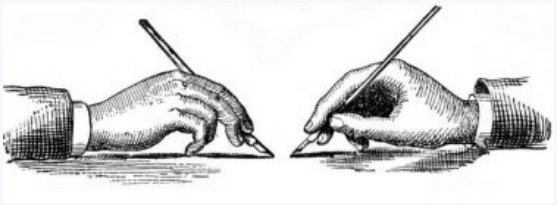

Warm greetings to all,

We are delighted to present the 16th issue of RxFactor from the National Association of Pharmacology and Therapeutics (NPT). This edition reflects the evolving landscape of pharmacology, integrating advances in drug delivery, digital health technologies, pharmacovigilance, and medical education.

This issue highlights emerging innovations that are reshaping pharmacology. We begin with the concept of the "Digital Pulse," where smart wearables and next-generation medical devices enable continuous monitoring of physiological parameters.

Advances in drug delivery are explored through brain shuttle peptides, including CNS-penetrant therapies. We also examine the pharmacological complexity of modern nicotine delivery systems, highlighting how electronic nicotine delivery systems alter pharmacokinetics and contribute to addiction dynamics, necessitating a pharmacological approach that matches the development of anti-craving agents.

In the vigilant corner we have an article discussing causative agents and mechanisms of Drug induced peripheral oedema.

In the Medical Education Corner, we present Prescription Detective™, an innovative teaching–learning approach that transforms real-world health information into interactive pharmacology cases. By integrating clinical scenarios with structured reasoning through the DETECT framework, this initiative promotes critical thinking, evidence-based medicine selection, and rational prescribing among students.

From the perspective of precision medicine, targeted therapies like selpercatinib exemplify the transition toward genetically tailored treatments in oncology. Also, insulin icodec, once-weekly basal insulin therapy, significantly improves adherence and convenience without compromising efficacy.

The issue also includes insightful discussions in clinical pharmacology and showcases contributions from academicians and postgraduate scholars, reflecting the depth and diversity of ongoing work in the field.

We extend our sincere gratitude to all authors and contributors for their valuable work. We remain committed to fostering academic excellence, innovation, and meaningful learning through RxFactor. We hope this issue proves informative and thought-provoking for our readers.

With regards,
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Jai Hind

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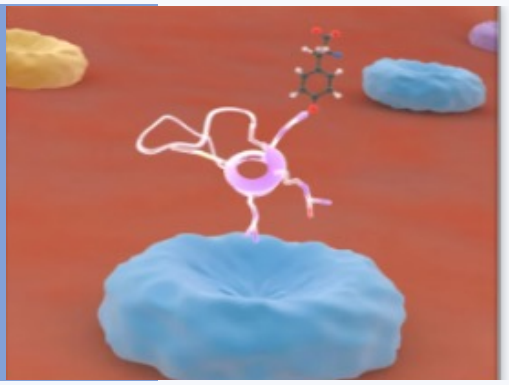
Newer drug delivery technique - Brain shuttle peptides

The brain shuttle peptides are molecules that have the capacity to transport drugs across the blood brain barrier and thus be helpful in treating diseases affecting central nervous system. Standard therapeutic molecules and large antibodies that were used were too big to cross the tight brain junction; they were expensive. The field of **brain shuttle peptides** (BSPs) and receptor-mediated transcytosis (RMT) platforms has reached major clinical, regulatory, and chemical milestones.



TfR1- targeted brain shuttle

1. Denali therapeutics brought a drug delivery breakthrough by launching **AVLAYAH** (Tividenofusp alfa-eknm), which is a CNS-penetrant enzyme replacement therapy. It utilizes a transferrin receptor (TfR) mediated brain shuttle for treatment of Hunter syndrome in children with ⩾ 5kg weight and before advanced neurological impairment

The FDA granted accelerated approval to AVLAYAH in March 2026. This shuttle is a fusion protein combining iduronate -2- sulfatase (IDS), the enzyme deficient in Hunter syndrome, combined with enzyme transport vehicle (ETV) designed to bind with the TfR1 on brain vascular endothelium

Mechanism of action- by binding to TfR1, AVLAYAH triggers receptor-mediated endocytosis across the blood-brain barrier, delivering the enzyme directly to brain parenchyma where standard transport vehicle cannot reach.

Dosage:- This is given as weekly intravenous infusion over 3-4 hours

Efficacy- it has shown 91% reduction in CSF heparan sulphate

Adverse effects:- A black box warning for hypersensitivity and anaphylaxis is present. Other side-effects include fever, upper respiratory tract infections, ear infections, vomiting, diarrhoea, and anaemia.

2. **Trontinemab**:- It is the shuttle that uses an anti-amyloid antibody bound with ETV for the treatment of Alzheimers disease. It targets the aggregated amyloid beta plaques and has shown rapid plaque reduction in phase 1 and phase 2 trials. It has not yet been FDA-approved and is currently under Phase 3 trial

Limitations: Peripheral off-target RBC destruction can lead to anaemia. High-affinity binding traps the drug within cells, while low affinity fails to facilitate effective transport; immunogenicity may also contribute. Additionally, traditional transport pathways may down regulate with ageing or neurodegenerative diseases, reducing efficacy over time. This can be addressed by utilising alternative mechanisms such as caveolae-mediated transcytosis or targeting alternate pathways

Caveolae mediated transcytosis: These pathways do not downregulate with age. Caveolae are tiny flask-shaped bubble pockets found across the cell membrane

Alternative targets:- Instead of competing with normal ligand activity, alternative receptors like IGF1R are targeted to avoid overcrowding and safely preserve CNS transport. One such example is ABL Bio's Grabody B, designed for treating Alzheimers disease. These advances will help us to overcome serum proteolysis and receptor saturation and turn the blood-brain barrier into a routine delivery route.

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The Digital Pulse of Pharmacology: Next-Generation Medical Devices and Smart Wearables

Introduction : Digital health technologies are transforming modern healthcare by enabling continuous monitoring of physiological parameters outside traditional clinical settings.[1] Conventional pharmacology often fails to capture real-time drug responses.[2] Smart wearables and next-generation medical devices bridge this gap by generating continuous digital data that reflect dynamic pharmacological effects.[3]

What is the 'Digital Pulse' in Pharmacology?

- The 'Digital Pulse' refers to the continuous physiological information generated through wearable sensors during monitoring for a disease and drug therapy.[4]
- Unlike conventional assessments, digital monitoring captures real-time pharmacodynamic changes, continuous physiological responses, early detection of therapeutic effects, and the dynamic relationship between pharmacokinetics (PK) and pharmacodynamics (PD).[5]
- This approach allows clinicians to understand drug response as a continuous process rather than isolated clinical observations.[6]

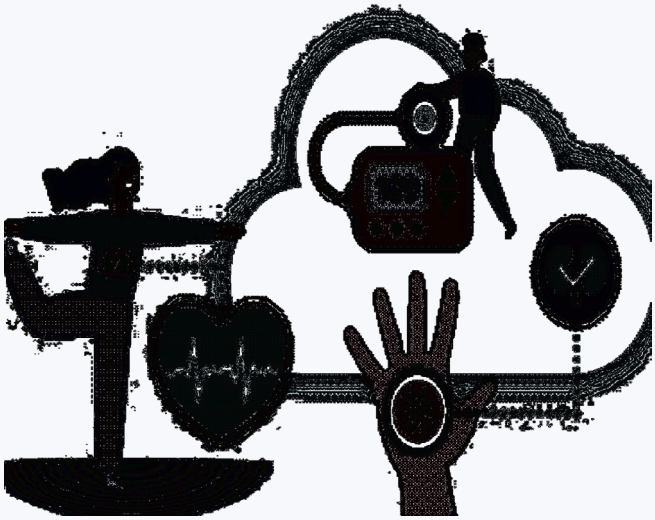
From Physiological Signals to Pharmacological Intelligence

Smart wearables collect physiological signals such as ECG, glucose levels, physical activity, and sleep parameters.[4]

The collected information is transmitted to cloud-based platforms where AI and machine learning algorithms process the data to generate meaningful digital biomarkers.[5]

Next-Generation Medical Devices and Smart Wearables

- Smartwatches and ECG patches: Monitor heart rate, heart rate variability, arrhythmias, and cardiac rhythm.[7]
- Continuous Glucose Monitors (CGM): Provide real-time glucose trends.[4]
- Activity and Sleep Trackers: Measure physical activity, energy expenditure, sleep quality, and functional recovery.[5]
- Wearable biosensor patches : Record electrophysiological and biochemical signals continuously. [5]
- Activity & sleep trackers → Functional outcomes[4-6]

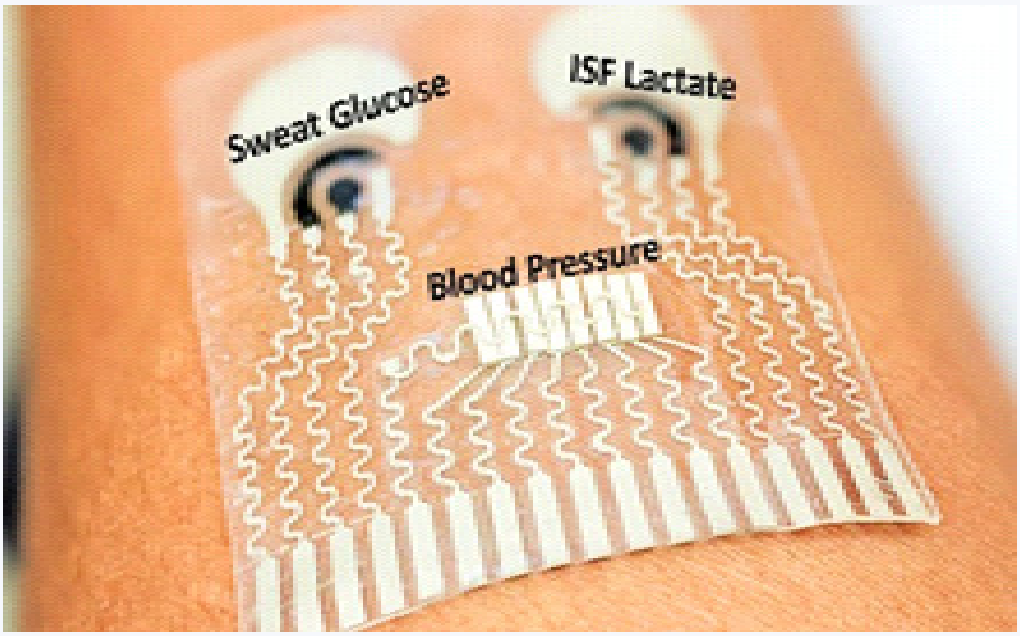


Role in Pharmacovigilance and Precision Medicine

Continuous physiological monitoring strengthens post-marketing surveillance through early detection of arrhythmias, hypoglycaemia, and drug-induced physiological changes while supporting individualized dose optimization and precision therapeutics.[2]

Impact on Drug Development

Smart wearables enable decentralized and hybrid clinical trials, improve patient recruitment and retention, enhance medication adherence monitoring, increase sensitivity of clinical endpoints, generate high-quality real-world evidence, and reduce trial duration and development costs.[7]

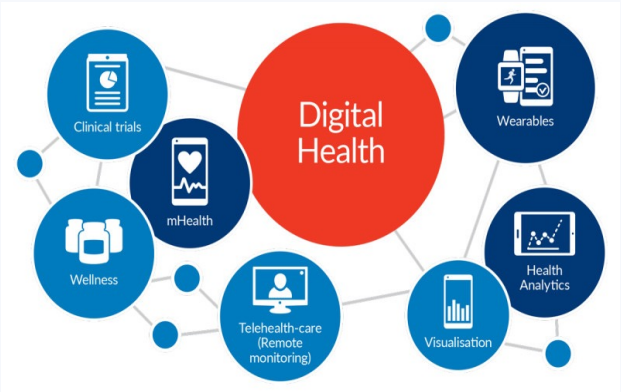


Pharmacological Significance:

Digital health technologies complement classical pharmacology by improving PK–PD modelling, Therapeutic drug monitoring, digital and clinical endpoints, Continuous safety assessment[3,6] ,Digital Health Technologies[3] ,Software as a Medical Device (SaMD)[3]

Regulatory Acceptance

FDA and EMA recognize Digital Health Technologies through guidance on remote data acquisition, Software as a Medical Device (SaMD), digital biomarkers, and real-world evidence integration, encouraging patient-centric drug development.[3]



Conclusion

Smart wearables represent the Digital Pulse of Pharmacology by enabling continuous physiological monitoring throughout drug therapy. AI converts physiological signals into clinically meaningful digital biomarkers, improving drug development, pharmacovigilance, precision medicine, and personalized therapy. Future pharmacology will be digital, continuous, and patient-centric.

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Theme of Nicotine Addiction

Engineered Addiction: Molecular Deception of Nicotine Salts

Introduction

Vaping is not just a trend or a simple public health issue anymore. For us, in clinical pharmacology, the rise of modern Electronic Nicotine Delivery Systems (ENDS) presents an interesting but concerning pharmacological challenge. The addiction we observe in clinics is not just about the physical act of vaping, it comes from deliberate, molecular-level engineering. Manufacturers have discovered that by altering the chemistry of the e-liquid itself, they can evade our natural bodily defences and deliver nicotine quickly to the brain. If we want to improve our cessation strategies, we must look beyond the devices and understand the pharmacokinetic and pharmacodynamic paradox involved.

The Pharmacokinetics of Protonation

In the early days of vaping, most devices were using freebase nicotine, which is naturally alkaline. Inhaling high concentrations of a highly alkaline aerosol triggers significant pharyngeal irritation known as the harsh "throat hit" [1]. Physiologically, this irritation serves as a defence mechanism. It acts as a natural limit, preventing the user from inhaling excessively or consuming too much nicotine at once. However, manufacturers found a chemical workaround, they began incorporating weak organic acids, like benzoic or lactic acid, into their product formulations [1]. This simple addition protonates the nicotine, reducing the pH and generating nicotine salts [1]. The protonated aerosol is smooth, eliminating the airway's natural defence. In the absence of harshness, users can effortlessly take deep, long draws, pulling in substantial amounts of nicotine directly into the pulmonary alveolar beds. Systemic absorption is rapid once it reaches the extensive alveolar surface area. Recent studies show that nicotine salts reach their peak plasma concentration (T_{max}) much faster than older freebase formulations [2]. The resulting kinetic spike, resembles the quick, intense delivery of a traditional cigarette. This rapid T_{max} increases reinforcing behaviours and creates a foundation for severe addiction [2] (Figure 1).

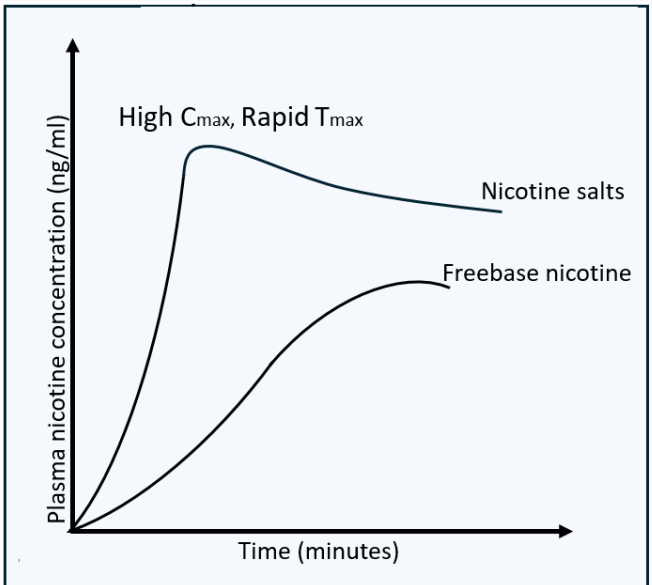

Figure 1: PK profile of nicotine salts vs Freebase nicotine

Pharmacodynamics and Receptor Response

So, what happens when those quick kinetic spikes hit the central nervous system? The high-affinity nicotinic acetylcholine receptors (nAchRs) are the main targets. Nicotine salts bypass the mucosal barriers and enter the blood rapidly; they end up sending high-amplitude waves of nicotine to these receptors. Cells cannot withstand a high level of nicotine for long periods without adapting, chronic exposure to nicotine forces the nAchRs into a state of functional desensitisation, temporarily shutting them down [3]. As a paradoxical response, the brain, in an attempt to maintain homeostasis, upregulates the receptors, thus increasing the density of receptors across the neuronal membrane [3]. This creates a hyperdense receptor environment which is ready to be triggered. When the next dose of nicotine is consumed, it attaches to this multiplied receptor population and triggers a dopamine surge in the ventral tegmental area (VTA) [4]. This vicious cycle of desensitisation-massive upregulation-exaggerated dopaminergic reward is what makes the effects of

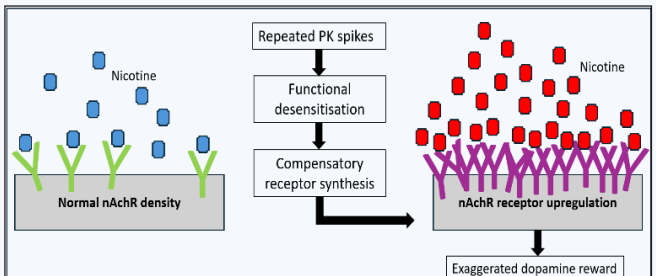

Figure 2: Pharmacodynamic response to nicotine salts

nicotine salts powerful, while also accounting for the withdrawal symptoms users face when the kinetic spike wears off [4] (Figure 2).

Implications for clinical de-addiction

This brings us to the realities we see in the de-addiction clinic. When a patient wants to quit, our typical approach involves steady-state nicotine replacement therapy (NRT) such as a transdermal patch. These are effective in delivering a slow, continuous flow of nicotine to ease baseline cravings [5]. However, they have to compete with a brain that is rewired to expect rapid spikes in nicotine levels and significant dopamine surges. A slow-releasing patch simply cannot match the effects of nicotine salts. This mismatch is a key reason why monotherapy often fails for heavy ENDS users. We need to take a more varied and aggressive approach to counter the heavily upregulated receptor population. We should consider combination therapies such as using the patch with a rapidly acting chewing gum or spray to mimic those spikes or stepping up to pharmacotherapies like Bupropion or Varenicline to better manage cravings at the receptor level [5].

Conclusion

We need to stop looking at vaping as just a bad habit or a sociological trend. It is a complex, engineered pharmacological dilemma, manipulated by kinetics and profound neuroadaptation. As members of the pharmacology fraternity, we need to view ENDS from the perspective of molecular chemistry and receptor density. Only by addressing and targeting the unique kinetic properties of nicotine salt, can we begin to develop cessation strategies that work in the real world.

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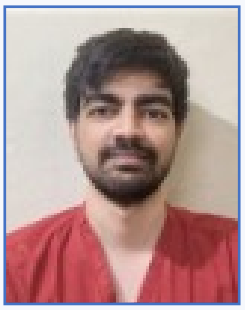

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Traditional to Translational: Reverse Pharmacology of Kalyanaka Ghrita in Cognitive Impairment

Introduction

The Indian elderly population of more than 60 years is projected to expand 2.5-fold over three decades: from 71 million in 2001 to 179 million by 2031. The estimated prevalence of dementia in this population is 7.4%. This acceleration increases the burden of age-related neurological and cognitive conditions, including mild cognitive impairment (MCI). [1,2] As this burden increases, the demand for safe, effective and personalized treatments has grown correspondingly- a gap traditional medicine systems may help address. Combination of Ethnopharmacology and Reverse Pharmacology is a novel approach to drug discovery from traditional medicinal practices using modern scientific methods. Reverse Pharmacology progresses through documented traditional knowledge followed by experimentation for systematic scientific validation. Unlike normal drug discovery, which starts in the laboratory, reverse pharmacology uses a 'Clinics to Laboratories' approach.

Traditional Evidence

One of the world's oldest medical systems i.e. Ayurveda describes several *Medhya Rasayana* formulations for enhancing memory, intellect, and mental well-being. Among these formulations *Kalyanaka Ghrita* (KG) gained considerable research interest, which is a polyherbal formulation made with clarified butter (ghee) as base and extracts from 28 different plants. It is traditionally indicated for *Smriti daurbalya* (poor memory) , psychosomatic disorders like *Unmada* (Schizophrenia), *Bhutonmada* (psychosis) , *Apasmara* (Epilepsy). [3]

Standardization of formulation

Traditional Ayurvedic formulations vary in preparation, ingredient sourcing, and composition—a feature of empirical development but an obstacle to scientific validation. Singh et al. (2024) resolved this by developing and validating a standard manufacturing procedure for the lipid-based formulation of *KG* which is essential for ensuring batch-to-batch consistency, improving quality control, and facilitating regulatory acceptance, strengthening scientific credibility.[3]

Experimental validation in β-amyloid animal model

Diddi et al. (2023) used a β-amyloid-induced memory impairment model in male Wistar rats to test *KG* as a potential Alzheimer's therapy. They assessed behavioural outcomes via Morris water maze, social recognition, and novel object recognition tasks alongside locomotor activity. Molecular endpoints included brain acetylcholinesterase activity, brain-derived neurotrophic factor (BDNF), inflammatory cytokines, oxidative stress markers, antioxidant levels, and histopathological examination. *KG* treatment restored cognitive and memory function, elevated brain BDNF and antioxidant capacity in Aβ1-42-treated animals, suppressed brain acetylcholinesterase activity, reduced oxidative stress and inflammatory cytokine levels, and mitigated neuronal damage. Such preclinical studies support traditional use and explain how *KG* works at the biological level. [4]

Real world Clinical Evidence

At NIMHANS Bengaluru, Ramana et al. (2019) conducted an open-label study in 40 children with cognitive deficit found that *KG* significantly improved cognitive performance on the Binet Kamat test and modified mini-mental status examination ($p < 0.001$), with assessments at days 0,30, 60, and 90. No safety concerns arose from laboratory monitoring. The results suggest *KG* is both safe and effective for managing childhood cognitive deficit. [5]

Precision Medicine through N-of-1 trial

Sharma et al. (2026) at IMS-BHU reported the first single-patient, quasi-randomized, controlled, open-label N-of-1 trial in an elderly patient with MCI tested *KG* over 14 months using alternating two-month treatment and no-treatment blocks. Cognitive function was tracked with Hindi Mental State Examination and Clinical Dementia Rating scales, while functional abilities and mood were assessed using standardized instruments. *KG* improved orientation and recall

consistently during active treatment periods, reduced dementia severity across cognitive domains, decreased depressive symptoms markedly without adverse effects. The pattern of improvement aligned with treatment on and off—when the treatment was given, cognitive gains appeared; when withheld, decline resumed. This cycle-to-cycle response in a single patient provides a proof-of-concept for precision pharmacology, where treatment can be evaluated and refined based on individual response rather than assumed efficacy from broader trials. The findings suggest *KG* holds potential for older adults with early cognitive decline.[6]

Conclusion

Taken together, these studies show a clear path from tradition to clinic for *KG*: traditional use suggests where to look; standardization guarantees the product quality; preclinical studies show biological plausibility; early patient trials confirm therapeutic promise; and N-of-1 trials reveal how well it works for one person at a time. This step-by-step approach, shows how traditional medicine and modern medicine can work together in research. While larger controlled trials are still needed to confirm efficacy, the studies reviewed here suggest that combining reverse pharmacology with precision medicine is a practical way forward for evidence-based medicine. This approach may help traditional medicines gain acceptance globally without losing what makes them valuable.

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Drug Induced Peripheral Oedema

Interstitial fluid accumulation (oedema) results from reduced oncotic pressure (N=25 mmHg), increased capillary pressure due to reduced precapillary sphincteric tone (precapillary dilation), increased capillary permeability (leak), sodium and water retention due to aldosterone or renal damage, and impaired lymphatic drainage due to block or damage. (Pal et al, 2022). The venous side of the capillaries has poor local auto-regulation. As a result, when there are venous changes in pressure (i.e., blood volume expansion or venous blockage), there are also changes in capillary hydraulic pressure that can lead to edema. Drug induced oedema may result from interplay of multiple factors as in case of estrogens (Table-1). Oedema may develop rapidly over days or gradually over weeks and months of therapy. In many cases oedema is mild and self-limiting but occasionally it may be a disturbing symptom and may worsen the underlying CHF.

CCBs: Both DHP-CCBs and Non DHP CCBs may cause pedal oedema. Nifedipine, and amlodipine induced pedal oedema manifests as diffuse and bilateral swelling over dorsa of feet, ankles and sometimes lower legs that worsens throughout the day and improves overnight (Sicca, 2003). DHP receivers are 11 times more prone to develop peripheral edema and may necessitate drug withdrawal in approximately 25% patients. Amazingly, nifedipine and amlodipine have mild diuretic action! Newer lipophilic CCBs (lercanidipine, manidipine) show less likelihood of oedema (7%). The underlying mechanisms are 1: Preferential dilation of pre-capillary over post-capillary arterioles, without venular dilation, 2: Antagonism of the postural sympathetic vasoconstrictor reflex, and 3: Lymphatics dilation: Clinically achievable concentrations of the nifedipine inhibit the spontaneous contractions of isolated human thoracic duct and mesenteric lymph vessels. There are many risk factors for CCB-induced edema such as female gender (chronic venous insufficiency is more common in females), obesity, diabetes, and older age. Oedema is considered to be dose and duration dependent. Diuretics do not relieve this vasodilatory oedema because it is not due to sodium and water retention (Masserli et al, 2002). Interestingly, ACE inhibitors and ARBs are effective. RAAS inhibition by these agents causes post-capillary dilation, normalizing intracapillary pressure and reduces fluid extravasation into the interstitial space. .

Hormonal agents: Glucocorticoids (GCs), anabolic steroids, danazol, estrogens, progestins and Oral contraceptives: Androgens and anabolics cause Na^+ and water retention through ENaC stimulation (Quinkler et al, 2005). Increased formation of angiotensinogen with resultant increased aldosterone activates Na^+-Cl^- reabsorption within the aldosterone-sensitive distal tubules and collecting ducts. Amazingly, chronic aldosterone excess is rarely associated with oedema because of 'aldosterone escape' characterized by a: normalization of Na+ levels b; ANP secretion and c; downregulation of ENaC channels in distal tubules resulting in excretion of excess Na+. GCs cause capillary leakage. Posaconazole, an antifungal azole, may cause pseudoaldosteronism. Estrogen (OCs) have multiple actions such as increased capillary permeability, precapillary arteriolar vasodilatation through Mas receptors activation induced by RAAS, polarization to ACE2 and sodium and water retention. Trayer et al, 2013)

Direct arterial and arteriolar dilators (Hydralazine / Potassium Channel Openers (KCOs): Diazoxide, Minoxidil/Riociguat/Bosentan, Ambrisentan

KCOs (Minoxidil) and diazoxide inhibit the rhythmic contractions of isolated lymph vessels and reduce in-vivo lymph flow and increase in capillary pressure (vasodilation-induced) leading to sodium and water retention through activation of RAAS axis resulting in oedema. Hydralazine that acts in part by opening of K_{ATP} channels in arterioles may cause oedema. K_{ATP} channel blockers (glibenclamide) partially restored the frequency of lymphatic contractions and fractional pump flow in isolated mesenteric lymph vessels (Zawieja et al., 2016). Glibenclamide reduced lymphedema in *Cantu syndrome*, a disease in which K_{ATP} channels exhibit gain-of-function (i.e., high open state probability) associated with a high incidence of lymphedema (Nichols et al., 2013).

Table-1: Other Drugs and Their Mechanisms:

Drug Group/Drugs (incidence %)	Mechanisms
Prazosin(1-4%).	Vascular α1A, α1B, and α1D receptor blockade leads to vasodilation of arteriole and veins, reduction in PVR and fluid retention.
Both Nonselective and COX-2 selective inhibitors (NSAIDs): Dose and duration dependent and necessitates withdrawal of NSAID (~10%)	A: **Vascular effects:** Blockade of vasodilatory prostaglandins (PGIs) that counter-regulate angiotensin-II mediated vasoconstriction B: **Renal effects:** preferential afferent glomerular arteriolar constriction reduces GFR, leading to Na+, water retention, increased blood volume
Gabapentinoids (Freynhagen et al, 2015) Pregabalin (in particular) & gabapentin cause fluid retention, peripheral oedema and weight gain in 17%	Blockade α2δ-1 subunits of vascular calcium channels (CCs) which may cause amlodipine like pedal oedema. Blockade of cardiac CCs reduce cardiac output and worsen CHF. Preferential precapillary arteriolar vasodilatation and inhibition of the myogenic response through Cav1.2 inhibition in small blood vessels results in fluid accumulation. Oedema usually decreases with dose reduction.
Thiazolidinediones (TZDs) (Pioglitazone, Lobeglitazone) (5-10% pioglitazone; 15-20% with insulin) (Scheen 2004)	PPARγ-mediated ↑vascular endothelial permeability, elevated VEGF secretion, and enhanced sodium and fluid retention in renal PCTs and collecting ducts.
Dopamine receptor agonists: pramipexole and ropinirole. Rarely, α-methyldopa and bromocriptine	stimulation of vascular presynaptic α-2 receptors causing reduced sympathetic tone and preferential arteriolar vasodilation
OKT3 monoclonal antibodies ((Muromonab-CD3)	↑ capillary permeability (capillary leak syndrome)
m-TOR inhibitors, sirolimus, Everrlimus	Lymphedema through antilymphangiogenic effects by VEGFR-3/VEGFC pathway inhibition.
Cyclosporine	Renal damage
Anthracyclines (Doxorubicin) and taxens (paclitaxel, docetaxel) (Zhang et al., 2019)	Breast tissue lymphedema. Capillary leak. Inhibition of rhythmic contractions of isolated rat mesenteric lymph vessels (Stolarz et al., 2019)
Insulins (10-20% incidence) more with TZDs (Lalande et al,2019).	↓capillary oncotic pressure, ↑vascular permeability, arteriolardilatation and Na/water retention due to its intrinsic antinatriuretic effect and through RAAS activation, Contributed by chronic hyperglycemia induced vasculopathy
Protein kinase inhibitors: Eritinib, crizotinib, imatinib, dasatinib, nilotinib bosutinib, ponatinib	Increased capillary permeability
Newer antidepressants Escitalopram, mirtazapine, paroxetine, venlafaxine	Arteriolar dilatation through 5-HT_2 receptors antagonism, resulting in oedema (Ravi et al, 2014)
Atypical antipsychotics (Clozapine, olanzapine, paliperidone, quetiapine, risperidone, ziprasidone)	Arteriolar dilatation through α1- and/or by 5-HT_2 receptors antagonism (Umer et al, 2016).
Methadone	Water retention by vasopressin release.
Apalutamide, epoetin alfa, sargramostim, IL-2, IL-11, hGH, Rifaximin, Itraconazole, Letermovir, Ivermectin, Panobinostat, Penicillamine	Capillary leakage and others

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Prescription Detective™: Using the DETECT Framework to Bring Current Affairs into the Pharmacology Classroom

"Today's news can become tomorrow's pharmacology lesson."

Medical students encounter abundant real-world health information, yet pharmacology teaching often remains theory-focused, limiting clinical application. Prescription Detective™ bridges this gap by transforming current health news into interactive cases, where students analyze scenarios, identify drug-related issues, and make rational therapeutic decisions.

THE DETECT FRAMEWORK

Each case follows the DETECT Framework, a six-step instructional model designed to promote contextual learning and clinical reasoning:

D – Discover: Identify an authentic trigger such as a current news report, FDA/CDSCO safety alert, social media claim, or published case report.

E – Evaluate: Analyse the patient's history, clinical presentation, and the prescription placed "under investigation."

T – Think: Apply pharmacological principles to understand the mechanism, diagnosis, adverse drug reaction, or prescribing error.

E – Explore: Search standard pharmacology references and current clinical guidelines to validate reasoning.

C – Choose: Recommend evidence-based management and rational prescribing decisions.

T – Translate: Conclude with a "Pharmacology Pearl" highlighting the key learning point and its application to future clinical practice.

The activity is firmly grounded in established educational principles. By using authentic contemporary cases, it embraces Situated Learning Theory, enabling students to learn within meaningful clinical contexts. Case-Based Learning encourages collaborative discussion and application of pharmacological concepts to patient care, while Constructivist Learning Theory supports active knowledge construction through inquiry and peer interaction. The visually rich poster format also aligns with Dual Coding Theory, integrating text and graphics to enhance understanding and retention.

Recent cases in the Prescription Detective™ series have explored topics such as Cyclospora-associated diarrhoea linked to contaminated food, topical lidocaine toxicity following cosmetic procedures, metoclopramide-induced acute dystonic reactions mistaken for seizures, and misuse of herbal "detox" teas containing stimulant laxatives. Each case concludes with evidence-based prescribing recommendations and key pharmacology pearls, reinforcing the importance of rational therapeutics.

Classroom experience has been highly encouraging. Students actively debate diagnoses, challenge misleading health claims, justify therapeutic choices, and frequently consult current guidelines during discussions. The authentic nature of the cases transforms pharmacology from a theoretical discipline into a dynamic clinical conversation, encouraging learners to appreciate the relevance of pharmacological principles in everyday medical practice.

The Prescription Detective™ series demonstrates that every authentic health headline has the potential to become a meaningful learning opportunity. By integrating current affairs with structured clinical reasoning, the DETECT Framework promotes curiosity, evidence-based thinking, and rational prescribing—competencies that are essential for tomorrow's physicians. We hope this approach encourages other educators to reimagine pharmacology teaching by bringing the world outside the classroom into the learning experience.

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Mentorship vs Patronage: Drawing the Line in Academic Medicine

1. Introduction & Paradigm Definitions

Mentorship and patronage represent distinct relational dynamics in academic medicine. While mentorship nurtures independent, self-sufficient scholars through reflective learning, patronage establishes transactional dependency that restricts intellectual freedom and perpetuates institutional bias.

Mentorship: An experienced guide providing personalized advice and support to facilitate a mentee's professional development and autonomy (Ramani et al., AMEE Guide No. 167).

Patronage / Sponsorship: A transactional arrangement where a senior figure provides opportunities, resources, or protection in exchange for compliance, personal loyalty, and positional status.

Table 1: Comparative Breakdown between Mentorship and Patronage

Feature / Dimension	Mentorship	Patronage
Main Goal	Professional growth & mentee independence	Personal benefit, status, & transactional loyalty
Power Dynamic	Guide → Supported self-reliance	Patron → Subordinate dependency
Selection Basis	Need, developmental goals, & mutual chemistry	Personal favoritism, alignment, or loyalty
Credit Allocation	Shared fairly based on transparent contribution	Preferentially awarded to select favorites
Transparency	High; open criteria and clear boundaries	Low; hidden arrangements & subjective choices
Dependency Risk	Low–Moderate (targets self-sufficiency)	Very high (sustains structural alignment)
Long-Term Effect	Capable, independent healthcare leaders	Inequality & institutional resentment

Institutional Risks and Ethical Boundaries

Academic favoritism damages organisational governance (Seniwoliba & Alhassan, 2023). When patronage dominates decision-making, recruitment, and promotion committees, institutions experience weakened quality assurance, skill-mismatched graduates, demoralisation, and lost global competitiveness. To preserve integrity, mentoring must align with core ethical principles (Shah et al., 2025): beneficence (promoting mentee interest), nonmaleficence (preventing exploitation/abandonment), autonomy (fostering independence), justice (equitable access), and strict boundary maintenance.

Ethical Boundaries and Principles in Mentoring

To prevent mentorship from devolving into patronage, medical educators must anchor their practice in core bioethical and professional principles (Shah, Gupta, & Singh, 2025). Core ethical principles for mentoring are beneficence, transparency, nonmaleficence, autonomy, justice, fidelity & privacy, boundaries & dual roles. The mentee and the mentor should understand the thin line in mentorship as provided below in the table

Table 2: What Mentoring IS vs. What Mentoring IS NOT

Mentoring IS	Mentoring IS NOT
A collaborative, growth-focused relationship	An authoritative dynamic demanding obedience
Active listening tailored to mentee aspirations	Dictating career paths without consultation
Balancing realistic challenge with adequate support	Overwhelming trainees or spoon-feeding solutions
Fostering independent critical thinking & networks	Restricting external collaborations or forcing authorship
Operating within agreed professional boundaries	Solving all personal issues or taking blame for failures
Guiding reflective practice and scholarship	Providing undue favors or bypassing merit criteria
Empowering the mentee to operate self-sufficiently	Sustaining lifelong professional subordination

Theoretical Frameworks & Mentoring Models

Optimal development occurs at the intersection of High Support and High Challenge (Sanford, 1962; Daloz, 1986). Low support with high challenge induces stress and coping failure, while high support with low challenge results in stagnation.

Modern health professions education utilises diverse delivery formats, such as One-on-One, Group, Peer, Reverse, Distance/E-Mentoring, and Speed Mentoring to widen access and mitigate hierarchical dependency.

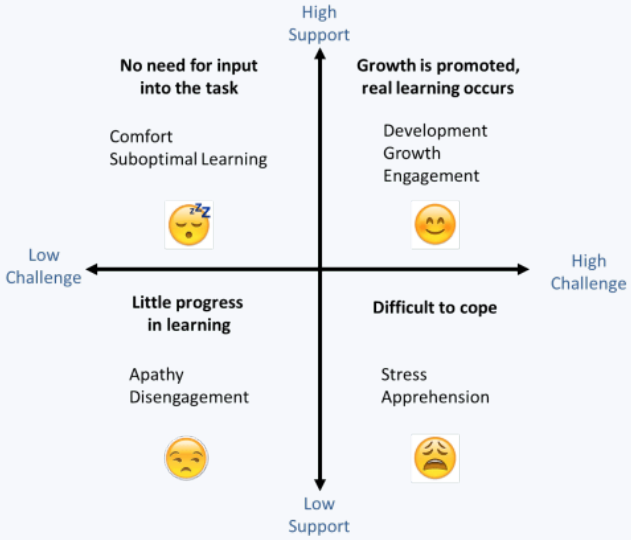

Fig 1: Sanford's Challenge–Support Theory (1962) & Education Daloz's Mentoring Framework (1986)

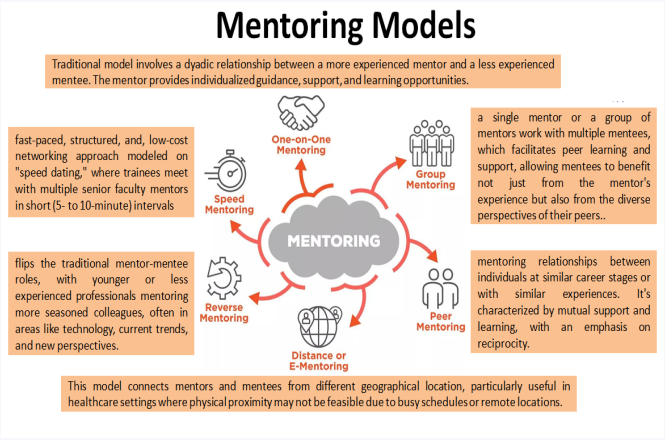

Fig 2: Diverse Mentoring Models in Health Professions

Practical Strategies & Formal Working Agreements for Healthy Mentorship

Formalising a working agreement prevents boundary drift (McCrossan et al., 2020). Key parameters include meeting every 4–6 weeks for 45–60 minutes in professional spaces, defining clear primary contact channels, maintaining confidential mentee-led logs, and establishing a no-fault exit mechanism. Senior faculty should follow the GREAT framework (Davis et al., 2023):

Give opportunities: Provide access to networks and milestones.

Reach out: Proactively identify goals and overcome obstacles.

Encourage by example: Model ethical resilience and integrity.

Advise individually: Recognize individual learning preferences.

Train for independent thinking: Focus on critical problem-solving.

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Selpercatinib: A New Era in Precision Oncology

Introduction

The evolution of precision medicine has transformed cancer treatment from conventional chemotherapy towards targeted therapies. One such breakthrough is Selpercatinib, a highly selective **Rearranged during Transfection (RET) kinase inhibitor** that has considerably improved outcomes in patients with RET-altered cancers. Selpercatinib is a prime example of the potential tailored cancer treatment since it directly targets genetic defects that promote tumor growth.

Understanding RET Alterations

The RET proto-oncogene encodes a receptor tyrosine kinase involved in regulating cell proliferation, differentiation, and survival. Genetic changes such as RET gene fusions and activating RET mutations result in chronic signaling that promotes oncogenesis. While activating RET mutations are typical in medullary thyroid carcinoma (MTC), RET fusions are frequently found in a fraction of non-small cell lung cancer (NSCLC) and papillary thyroid carcinoma. The identification of these molecular changes has enabled the development of highly focused medicines.

Mechanism of Action

Selpercatinib is an oral, highly selective ATP-competitive inhibitor of the RET receptor tyrosine kinase. By binding to the ATP-binding pocket of RET, it inhibits phosphorylation and downstream signaling through the MAPK, PI3K-AKT, and JAK-STAT pathways, thereby reducing tumor cell proliferation and survival.

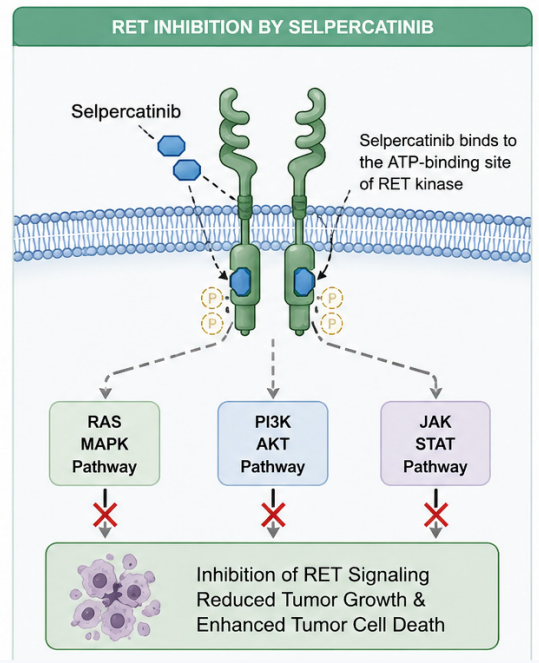

Figure 1: Mechanism of Action - Selpercatinib

Clinical Evidence

The pivotal LIBRETTO-001 clinical trial demonstrated the effectiveness of Selpercatinib. In patients with RET-mutant medullary thyroid carcinoma, RET fusion-positive thyroid malignancies, and RET fusion-positive NSCLC, the study showed high response rates and long-lasting responses. Its efficacy in managing both systemic and central nervous system disease was demonstrated by the significant intracranial responses seen in individuals with brain metastases. Selpercatinib is the first and only selective RET inhibitor approved in India for the treatment of RET-altered locally advanced or metastatic solid tumors upon having received marketing authorization in June 2026 from the CDSCO.

Safety Profile

Selpercatinib is often well tolerated; nevertheless, clinicians should monitor patients for treatment-related adverse effects. Hypertension, increased liver transaminases, dry mouth, diarrhea, constipation, exhaustion, peripheral edema, and QT interval prolongation are typical side effects. Throughout treatment, routine monitoring of blood pressure, liver function, and ECG is advised. Given that the cytochrome P450 (CYP3A) enzyme system is the primary metabolizer of Selpercatinib, it is crucial to assess potential drug-drug interactions carefully.

Role of Pharmacogenomics

The increasing significance of pharmacogenomics in oncology is demonstrated by the clinical success of Selpercatinib. The discovery of RET changes is made possible by molecular diagnostic tools such as next-generation sequencing (NGS), fluorescence in situ hybridization (FISH), and polymerase chain reaction (PCR)-based assays, which guarantee that patients who are most likely to benefit are chosen correctly. This precision-based strategy reduces needless exposure to ineffective medicines while maximizing therapeutic results.

Future Perspectives

Despite Selpercatinib's impressive effectiveness, acquired resistance remains a significant challenge. Understanding resistance mechanisms, creating next-generation RET inhibitors, and assessing combination treatment approaches are the main goals of ongoing research. Additionally, its usage in early stages of the disease and in conjunction with other targeted or immunotherapeutic medicines is being studied in clinical trials.

Conclusion

By offering patients with RET-driven tumors an efficient and well-tolerated treatment, Selpercatinib marks a significant advancement in precision oncology. Its achievement highlights the significance of genomic testing in standard clinical practice and demonstrates the expanding influence of molecularly targeted medicines. Drugs like Selpercatinib will continue to be essential to the future of individualized cancer care as pharmacogenomics advances continue to broaden therapeutic choices. To maximize patient results, pharmacologists and medical professionals must comprehend the pharmacology, clinical uses, and developing evidence around selective RET inhibitors.

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PI3K Inhibitor-Induced Hyperglycemia: Pathophysiology, Clinical Impact, and Management Strategies`

Introduction:

Phosphatidylinositol 3-kinase (PI3K) inhibitors have emerged as vital targeted therapies in oncology, particularly for patients with -mutated hormone receptor-positive (), human epidermal growth factor receptor 2-negative () advanced breast cancer. However, because the PI3K signaling pathway is central to systemic glucose metabolism, pharmacological blockade of PI3K frequently causes on-target hyperglycemia. This review discusses the physiological mechanisms underlying PI3K inhibitor-induced hyperglycemia, clinical prevalence, and management strategies.

FDA-Approved PI3K Inhibitors and availability in India:

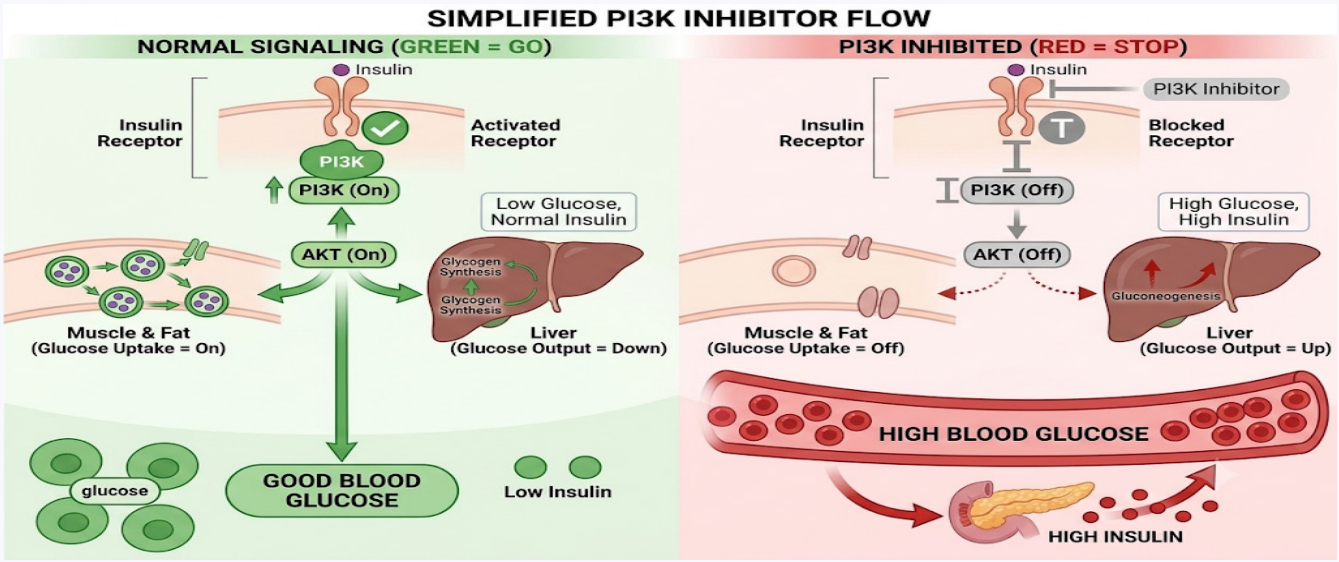
The US FDA has approved several PI3K inhibitors, including -isoform selective agents (Alpelisib, Inavolisib) for advanced breast cancer and others (Idelalisib, Copanlisib, Duvelisib, Umbralisib) for hematologic malignancies. In India, only Alpelisib is CDSCO-approved and commercially available; others are accessed primarily via special import permits, named-patient programs, or clinical trials.

Pathophysiology and mechanism:

The class I PI3K pathway, specifically the catalytic isoform encoded by the gene, serves as a crucial downstream effector of the insulin signaling cascade. Under physiological conditions, insulin binding to its receptor activates PI3K, stimulating downstream phosphorylation of AKT. This pathway promotes the translocation of glucose transporter type 4 (GLUT4) storage vesicles to the plasma membranes of skeletal muscle and adipose tissue, facilitating peripheral glucose uptake. Concurrently, hepatic PI3K activation suppresses gluconeogenesis and glycogenolysis.

When an -selective PI3K inhibitor is administered, this cascade is acutely blocked. The inhibition of GLUT4 translocation prevents peripheral glucose utilization, while the suppression of hepatic glucose output is lifted, driving hepatic gluconeogenesis. The resulting rapid rise in blood glucose levels triggers compensatory hyperinsulinemia via pancreatic -cell secretion. This hyperinsulinemia attempts to overcome peripheral resistance, creating a state of transient insulin resistance.

The -isoform of PI3K drives insulin-mediated glucose transport; its selective inhibitors (e.g., Alpelisib, Inavolisib) cause severe hyperglycemia by blocking GLUT4 translocation and boosting hepatic gluconeogenesis. Conversely, the -isoform plays a secondary role, so its inhibition results in only mild or



negligible hyperglycemia as peripheral glucose uptake remains intact.

Clinical Incidence and Implications:
Real-world observational studies have reported overall hyperglycemia rates exceeding 70–80%. The onset of hyperglycemia is characteristically rapid, with a median time to occurrence of approximately 15 days. If unmanaged, severe hyperglycemia can lead to life-threatening acute metabolic events, such as diabetic ketoacidosis (DKA) or hyperosmolar hyperglycemic state (HHS). Furthermore, uncontrolled glucose elevations frequently necessitate dose reductions or treatment interruptions, potentially compromising cancer control.

Baseline Assessment: Prior to initiating PI3K inhibitor therapy, all patients should undergo baseline glycemic screening, including fasting plasma glucose (FPG) and glycated haemoglobin (). Risk factors for severe hyperglycemia include pre-existing diabetes, prediabetes (5.7–6.4%), elevated body mass index (), and age years.

Management Guidelines for PI3K Inhibitor-Induced Hyperglycemia:
First-Line Pharmacotherapy: Metformin is the preferred initial agent due to its ability to decrease hepatic gluconeogenesis and improve peripheral insulin sensitivity without driving excess insulin release.

Second-Line Pharmacotherapy: Sodium-glucose cotransporter 2 (SGLT2) inhibitors (e.g., Dapagliflozin, Empagliflozin) have shown significant efficacy in clinical and preclinical models. By promoting glucosuria independently of insulin, SGLT2 inhibitors lower blood glucose while mitigating hyperinsulinemia.

Avoidance of Exogenous Insulin: Insulin therapy is generally reserved as a treatment of last resort. Exogenous Insulin can bind to insulin receptors on tumour cells and re-activate residual PI3K or parallel signaling cascades (such as the MAPK pathway), potentially promoting tumour growth and treatment resistance.

Conclusion:
Hyperglycemia is an expected, on-target metabolic consequence of PI3K inhibitor therapy, with the highest severity observed in -isoform specific agents like Alpelisib and Inavolisib compared to -isoform targeters. Early identification of high-risk patients, routine monitoring during the initial weeks of treatment, and prompt initiation of insulin-sparing agents enable effective glycemic control while maintaining therapeutic efficacy against -mutated tumors.

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The ToxiGuide: A Digital Poisoning Information Centre

Scan QR to Access

What is The ToxiGuide?

The ToxiGuide is a comprehensive digital poisoning information centre designed to provide rapid, accurate, and evidence-based information for the identification, assessment, and management of poisoning cases. It serves as a reliable resource for healthcare professionals, students, emergency responders, and the general public, promoting timely and appropriate management of poisoning emergencies.

Need for a Poisoning Information Centre (PIC)

Provides authentic, evidence-based, and up-to-date toxicology information.

Supports clinical decision-making during poisoning emergencies.

Reduces treatment delays by providing immediate access to antidote and management guidelines.

Promotes rational use of antidotes and emergency interventions.

Improves patient safety and clinical outcomes.

Strengthens poison prevention awareness and public education.

Purpose and Scope

Purpose	Scope
Promote early recognition of poisoning	Common household, pharmaceutical, pesticide, plant, animal, and chemical poisonings
Improve patient safety	Poison identification and toxicity information
Provide evidence-based management	First aid measures and emergency treatment protocols
Assist healthcare professionals	Antidote information with indications and dosing
Encourage poison prevention	Poison prevention tips and patient counselling
Support education and training	Clinical toxicology updates, guidelines, and educational resources

Who Can Use The ToxiGuide? Doctors, Pharmacists, Nurses, Emergency Healthcare Professionals, Researchers, General Public seeking reliable poisoning information

Key Features of The ToxiGuide

Easy-to-use digital platform accessible anytime.

First aid recommendations before medical care.

Emergency management and treatment guidelines.

Antidote directory with essential clinical information.

Regularly updated according to current toxicology guidelines.

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Insulin Icodec-First Approval

Type 2 diabetes mellitus (T2DM) is a progressive metabolic condition that often necessitates basal insulin therapy to achieve optimal glycaemic control. Despite the demonstrated efficacy of long-acting basal insulin analogues, the requirement for daily injections frequently contributes to poor adherence. As a result, the development of ultra-long-acting insulin formulations capable of decreasing injection frequency has become an essential treatment goal.

Insulin icodec (AWIQLI) is the first once-weekly basal insulin analogue authorised for people with Type 2 Diabetes. Its once-weekly dosage strategy has the potential to increase treatment adherence, reduce injection burden, and boost patient satisfaction. **The following table summarises the essential pharmacological properties, dose recommendations, safety profile, and regulatory information for insulin icodec.**

Why Is Insulin Icodec Administered Once Weekly?

Ultra-long half-life (Approx 7 days) provides continuous basal insulin coverage.

>99% reversible albumin binding enables slow and sustained insulin release.

Structural modifications delay absorption and clearance.

Stable pharmacokinetics maintain consistent glucose-lowering activity throughout the week.

The introduction of insulin icodec marks a paradigm shift in diabetes management. By providing effective once-weekly basal insulin replacement with a favorable efficacy and safety profile, it has the potential to simplify insulin therapy and improve long-term clinical outcomes.

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Junior Resident(Academic)
Department of Pharmacology
All India Institute of Medical Sciences, Patna, Bihar

Parameter	Details
Generic name	Insulin icodec-abae
Brand name	**AWIQLI**
Alternative names	Insulin icodec
Developer/Manufacturer	Novo Nordisk
Drug class	**Ultra-long-acting once-weekly basal insulin analogue**
Indication	Glycaemic control in adults with Type 2 Diabetes Mellitus
Regulatory approval	US FDA approval (March 2026); marketed as AWIQLI
Mechanism of action	Activates insulin receptors to increase peripheral glucose uptake, suppress hepatic glucose production, inhibit lipolysis and proteolysis, and promote glycogen synthesis
Structural modification	Contains C20 fatty diacid side chain **that enables strong reversible albumin binding and prolonged circulation**
Route of administration	Subcutaneous injection (abdomen, thigh, or upper arm)
Dosage form	Concentration: 700 units/mL (U-700) Administered using U-700 prefilled **FlexTouch** pen
Recommended dosing	☐ Once weekly on the same day each week ☐ Insulin-naïve adults generally initiated with **70 units/week** followed by individualized titration
Missed dose	May be administered within **4 days**; otherwise skip and resume the regular weekly schedule
Protein binding	>99% reversible albumin binding
Time to peak concentration (Tmax)	Approximately 16 hours
Elimination half-life	Approximately **196 hours (~7 days)**
Steady-state concentration	Achieved within **2–3 weeks**
Metabolism	Proteolytic degradation to inactive metabolites
Common adverse effects	Hypoglycaemia, injection-site reactions, lipodystrophy, localized cutaneous amyloidosis, oedema, rash, pruritus, weight gain
Serious warnings	☐ Severe hypoglycaemia ☐ Hypersensitivity reactions ☐ Hypokalaemia, ☐ Worsening heart failure with concomitant thiazolidinediones
Contraindications	☐ Active hypoglycaemia ☐ Hypersensitivity to insulin icodec or formulation components
Storage	1. Refrigeration at **2–8°C** 2. Unopened or in-use pens may be stored for up to **12 weeks** below **30°C**
Clinical significance	First once-weekly basal insulin, representing a major advancement in insulin therapy by reducing annual injections from **365 to approximately 52** while maintaining effective glycaemic control

Enlicitide: A Game Changer in Lipid-Lowering Therapy

Cardiovascular disease (CVD) remains the leading cause of mortality worldwide, with elevated **low-density lipoprotein cholesterol (LDL-C)** being a major modifiable risk factor for **atherosclerotic cardiovascular disease (ASCVD)**. Although **statins** are the cornerstone of lipid-lowering therapy, many high-risk patients fail to achieve recommended LDL-C targets despite maximally tolerated treatment. Injectable **proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors** have transformed dyslipidemia management but are limited by high cost, injection burden, and poor long-term adherence. The recent approval of **enlicitide**, the **first orally administered PCSK9 inhibitor**, represents a major advance in lipid management with the potential to improve cardiovascular prevention. [1]

PCSK9 is a hepatic protein that promotes degradation of **LDL receptors**, thereby reducing hepatic clearance of LDL cholesterol. **Enlicitide**, an orally active **macrocyclic peptide**, selectively inhibits circulating PCSK9, preserving LDL receptor recycling and enhancing hepatic uptake of LDL-C. Unlike statins, which inhibit cholesterol synthesis, enlicitide increases cholesterol clearance, making it an effective adjunct to existing lipid-lowering therapies. [2]

The efficacy of enlicitide has been demonstrated in the **Phase 3 CORALreef programme**. In patients receiving maximally tolerated lipid-lowering therapy, enlicitide reduced **LDL-C by approximately 56–59%** compared with placebo, with sustained efficacy over one year. Significant reductions were also observed in **apolipoprotein B (ApoB)** and **non-high-density lipoprotein cholesterol (non-HDL-C)**. Its LDL-C lowering is comparable to that achieved with injectable PCSK9 monoclonal antibodies. Clinical benefits were consistent in patients with **heterozygous familial hypercholesterolemia (HeFH)** and those at high or very high cardiovascular risk. Current **ACC Expert Consensus** supports PCSK9-targeted therapies for patients requiring additional LDL-C reduction despite maximally tolerated statin therapy. [3]

Clinical trials have demonstrated a **favorable safety profile**, with adverse events comparable to placebo. Mild gastrointestinal symptoms, including diarrhea and nausea, were the most commonly reported adverse effects. Unlike injectable PCSK9 inhibitors, enlicitide eliminates injection-site reactions and provides the convenience of **once-daily oral administration**, potentially improving treatment acceptance and long-term adherence. According to the prescribing information, the drug should be taken **on an empty stomach**, underscoring the importance of patient counseling. [4]

The principal advantage of enlicitide is its **oral formulation**, which addresses an important unmet need for patients reluctant or unable to receive injectable therapy. It may be particularly beneficial for individuals who remain above LDL-C goals despite statins and ezetimibe and for those with **familial hypercholesterolemia**. The **2021 ESC Guidelines** emphasize intensive LDL-C lowering in high- and very high-risk patients, a goal that enlicitide may help achieve. [5]

Although enlicitide produces substantial LDL-C reductions, definitive evidence demonstrating reductions in **major adverse cardiovascular events**, including myocardial infarction, stroke, and cardiovascular mortality, is still awaited from ongoing outcome trials. Furthermore, cost, accessibility, reimbursement, and real-world effectiveness will determine its ultimate impact on clinical practice.

In conclusion, **enlicitide** marks a new era in dyslipidemia management as the **first oral PCSK9 inhibitor**. By combining potent LDL-C reduction with the convenience of oral administration, it addresses key limitations of injectable PCSK9 inhibitors and expands therapeutic options for patients with hypercholesterolemia. If ongoing cardiovascular outcome trials confirm reductions in major adverse cardiovascular events, enlicitide may truly justify its reputation as a **"game changer"** in cardiovascular prevention. [1]

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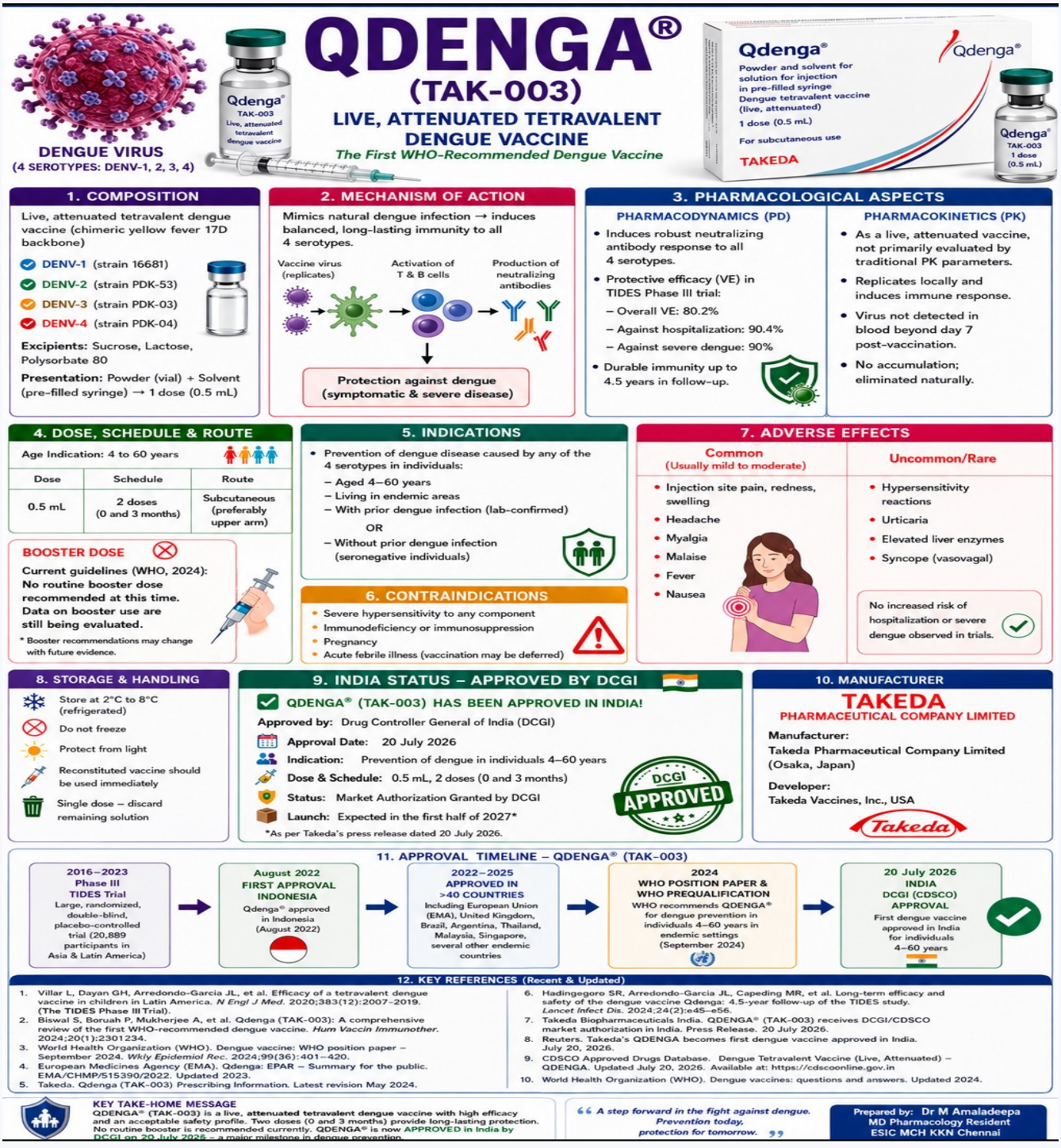

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The Pharmacologist's Challenge

Can you identify the drugs?

COOL CORNER

Challenge 1

Clue 1
I was initially developed as an antianginal drug.

Clue 2
I prolong the action potential by blocking multiple ion channels.

Clue 3
Unlike many antiarrhythmics, I have a very low incidence of torsades de pointes.

Clue 4
My long-term use may cause blue-grey skin pigmentation, thyroid dysfunction and pulmonary fibrosis.

Challenge 2

Clue 1
I am an antineoplastic antibiotic.

Clue 2
My efficacy depends on free radical generation.

Clue 3
I have little bone marrow toxicity.

Clue 4
My dose is limited by potentially irreversible pulmonary fibrosis.

Challenge 3

Clue 1
I am just heavier than hydrogen but lighter than sodium.

Clue 2
I compete with sodium to be eliminated from the body.

Clue 3
NSAIDs and thiazides can increase my serum concentration.

Clue 4
Long-term therapy may produce nephrogenic diabetes insipidus.

Challenge 4

Clue 1
I inhibit xanthine oxidase.

Clue 2
I increase the toxicity of azathioprine.

Clue 3
My dose should be reduced in chronic kidney disease.

Clue 4
I may rarely cause severe hypersensitivity syndrome associated with HLA-B*58:01.

Challenge 5

Clue 1
I inhibit calcineurin.

Clue 2
I do not cause significant bone marrow suppression.

Clue 3
I commonly produce hypertension and gingival hyperplasia.

Clue 4

My dose-limiting toxicity is nephrotoxicity.

Quick One-Liners – Who am I?

I am the only antipsychotic proven to reduce suicide in schizophrenia.

I require leucovorin rescue to limit my toxicity.

I produce purple glove syndrome after intravenous administration.

I cause dose-dependent optic neuritis with red-green color blindness.

I am the only antidepressant approved specifically for premature ejaculation.

I can cause serotonin syndrome despite not being classified as an antidepressant.

I cause a rise in serum creatinine without actually reducing the glomerular filtration rate.

My overdose is uniquely treated with high-dose insulin euglycemia therapy.

I am the only proton pump inhibitor available for intravenous continuous infusion in acute upper gastrointestinal bleeding.

I am the only oral P2Y12 inhibitor that requires CYP2C19-mediated activation.

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Nerchowk HP

What is EXTRACT

Extract are the collections of some important points taken from the discussion in National MD Pharmacology group. NMDP is a group of eminent pharmacologists from all over the country. The head of departments of pharmacology, deans, directors of institutions and people with significant contribution in the field of pharmacology are members of NMDP family. National association of Pharmacology and Therapeutics is promoted by NMDP group.

Shuttle Box: A Classic Tool in Behavioural Pharmacology

The shuttle box is a valuable experimental model for studying learning, memory, avoidance, and anxiety-related behaviours, helping assess CNS drug effects and related neuropharmacological mechanisms.

Clinical Ethics Committees: Strengthening Ethical Decision-Making in India

Clinical Ethics Committees can support multidisciplinary, patient-centred decisions, promoting transparency, accountability, and ethical healthcare practice in India.

The Future of Clinical Trials: Towards 2050

Clinical trials are moving toward AI-enabled, wearable-supported, adaptive, and real-world data-driven models that accelerate patient-centric precision medicine.

Managing Expired Medicines

Effective expired-medicine management through audits, regulatory compliance, and safe disposal reduces medication errors and protects public health and the environment.

INTERASPIRE 2026

INTERASPIRE 2026 emphasizes individualized blood pressure targets in coronary heart disease, where BP below 120/70 mmHg may reflect frailty or comorbidity, emphasizing precision rather than aggressive lowering.

Baxdrostat

Baxdrostat, a selective **aldosterone synthase inhibitor**, offers targeted RAAS blockade for resistant hypertension, with phase III trials defining its long-term role.

NMC–ICMR Initiative: Strengthening Clinical Research in Medical Education

The NMC–ICMR initiative aims to integrate clinical research, ethics, scientific writing, and AI into medical education to strengthen evidence-based practice and research culture.

Remembering Dr. Yellapragada Subbarow

Dr. Yellapragada Subbarow's discoveries in ATP, antifolates, diethylcarbamazine, and tetracyclines continue to shape modern medicine and pharmacology.

Warfarin: A Paradigm of Clinical Pharmacokinetics

From a pharmacologist's perspective, warfarin exemplifies the clinical application of pharmacokinetic principles. Warfarin illustrates precision pharmacotherapy through its narrow therapeutic index, genetic variability, drug interactions, and need for individualized INR-guided dosing.

Affordable Medicines

Affordable medicines require evidence-based prescribing, quality-assured generics, transparent pricing, and strong regulation to improve adherence and outcomes.

Clinical Pharmacology Requires Clinical Competence

As pharmacologists, we believe Competency-Based Medical Education demands educators with formal medical training to teach rational therapeutics, prescribing, pharmacovigilance, and clinical decision-making. While non-medical pharmacologists make invaluable research contributions, the clinical teaching of medical students should remain the responsibility of medically qualified pharmacologists with direct patient-care experience.

Targeted Therapy Shows Promise in Chronic Inducible Urticaria

RemIND study data suggest Rhapsido® may become a targeted therapy for chronic inducible urticaria, advancing precision immunopharmacology.

Banning Irrational Fixed-Dose Combinations

The ban on 16 irrational fixed-dose combinations reinforces the need for scientific validation, patient safety, and evidence-based pharmaceutical regulation.

The Insulin–Potassium Axis

As pharmacologists, we emphasize that insulin exerts physiological effects beyond glycemic control. By stimulating Na^+/K^+-ATPase activity, insulin promotes intracellular potassium sequestration, whereas extracellular potassium influences pancreatic β-cell function and insulin release. Understanding this reciprocal relationship is essential for optimizing therapy in diabetes, hyperkalemia, and critically ill patients

QR Codes on Drug Labels

Effective 1 July 2027 (with antimicrobial provisions effective from 1 July 2028), the amendment of Drug Rules, mandates the inclusion of Quick Response (QR) codes on labels of selected high-priority therapeutic categories, including vaccines, antimicrobials, anticancer medicines, and narcotic/psychotropic drugs. This will strengthen traceability, authentication, pharmacovigilance, and patient safety.

Olezarsen: A New Era in Lipid Pharmacotherapy

Olezarsen, the first RNA-targeted therapy for severe hypertriglyceridemia, reduces triglycerides and pancreatitis risk while advancing precision cardiovascular care.

Beyond Half-Life: Understanding Rational Drug Dosing

As pharmacologists, we emphasize that elimination half-life alone does not determine dosing frequency. Dutasteride is administered once daily to maintain sustained 5α-reductase inhibition, Delamanid twice daily to achieve optimal antimycobacterial exposure, and Amlodipine once daily because its prolonged pharmacodynamic effect ensures effective 24-hour blood pressure control. Rational dosing integrates both pharmacokinetic and pharmacodynamic principles.

Wegovy for MASH

Semaglutide approval for MASH with moderate-to-advanced fibrosis expands GLP-1 receptor agonists' role in hepatic and metabolic pharmacotherapy.

Answer KEY to The Pharmacologist's Challenge

Challenge 1–5

1. Amodarone
2. Bleomycin
3. Lithium
4. Allopurinol
5. Cyclosporine

Quick One-Liners

1. Clozapine
2. Methotrexate
3. Phenytoin
4. Ethambutol
5. Dapoxetine
6. Linezolid
7. Trimethoprim
8. Verapamil
9. Pantoprazole
10. Clopidogrel

IUPHAR and National Association of Pharmacology and Therapeutics (NPT), India

Meeting Minutes
14th July 2026 14:00 – 14:30 local time Melbourne

Attendees:

Caroline Samer **PRESIDENT IUPHAR**	**Emilio Clementi** **SECRETARY GENERAL IUPHAR**	**Alicia Goia** **(OIC) IUPHAR**
Ganesh Dakhale **NATIONAL PRESIDENT NPT**	**C M Kamaal** **NATIONAL COORDINATOR/FOUNDER/CEO/NPT**	Pramod Manjihi **MEMBER NATIONAL COUNCIL NPT**
Alessia Costanzo		

Representatives of the National Association of Pharmacology and Therapeutics. (NPT), India presented their association and explored potential collaboration opportunities with the International Union of Basic and Clinical Pharmacology (IUPHAR).

The Indian organization represents approximately 3,200 MD pharmacologists and positions itself as a patient-centered clinical pharmacology society, distinct from other pharmacology associations in India. Its primary focus is on bridging pharmacology and direct patient care, integrating therapeutics, bedside teaching, and clinical decision-making into pharmacology training and practice. Membership mainly consists of MD Pharmacologists, including academic faculty, researchers, and clinicians.

The delegation highlighted the organization's strong academic and professional infrastructure, including its registration under the Government of India, regular national conferences, publication of conference proceedings, and the development of a professional journal. The society plays an active role in postgraduate medical education through innovative bedside-based pharmacology examinations and clinical training initiatives.

A significant part of the discussion focused on the possibility of establishing a formal relationship with IUPHAR, either through full membership, associate membership or recognition as a special interest group. IUPHAR representatives explained the current membership structure and noted that existing rules generally recognize one primary national society per country. Nevertheless, they expressed interest in exploring mechanisms to strengthen collaboration and increase engagement with the Indian clinical pharmacology community.

The participants identified several opportunities for future cooperation, including:

Joint educational webinars and scientific exchanges.

Participation in IUPHAR working groups, committees, and international projects.

Greater involvement in the World Smart Medication Day initiative and related student poster competitions.

Enhanced engagement of Indian pharmacologists in global pharmacology and therapeutics activities.

The National Association of Pharmacology and Therapeutics (NPT), India representatives also emphasized its extensive experience in clinical research, pharmacovigilance, ethics, and clinical trials. Members are actively involved in national pharmacovigilance programs, Good Clinical Practice (GCP) training, ethics committee oversight, antimicrobial stewardship, clinical trial management, and initiatives related to alternatives to animal testing. The association maintains access to national pharmacovigilance databases and contributes significantly to clinical research and patient safety activities.

IUPHAR representatives acknowledged India's strong tradition in clinical pharmacology and recognized the value of an organization focused on direct patient care and therapeutics.

It was agreed that practical options for future collaboration would be explored, including connecting

the association with relevant IUPHAR contacts and educational programs. Both parties expressed a shared interest in strengthening international cooperation and advancing clinical pharmacology education, research, and patient care.

PRESS RELEASE

National Association of Pharmacology and Therapeutics Strengthens its Global Engagement

NPT Strengthens Global Engagement Through High-Level Strategic Meeting with IUPHAR Leadership at WCP 2026

Melbourne, Australia | 14 July 2026

The National Association of Pharmacology and Therapeutics (NPT), India, achieved a significant milestone in its international journey by holding an official high-level strategic meeting with the leadership of the International Union of Basic and Clinical Pharmacology (IUPHAR) during the 20th World Congress of Basic and Clinical Pharmacology (WCP 2026) in Melbourne, Australia. The meeting provided an important platform for NPT to present its vision, achievements, and unique role in advancing Clinical Pharmacology and Therapeutics, while exploring avenues for future academic collaboration and global engagement.

The meeting was attended by **Prof. Caroline Samer (President, IUPHAR), Prof. Emilio Clementi (Secretary General, IUPHAR), Ms. Alicia Goia (Operations in Charge, IUPHAR), Dr. Ganesh Dakhale (National President, NPT), Dr. C. M. Kamaal (Founder, CEO & National Coordinator, NPT), and Dr. Pramod Manjhi (Member, National Council, NPT).**

The NPT delegation highlighted the Association's distinctive patient-centered approach to Clinical

Pharmacology and Therapeutics, its national academic initiatives, postgraduate education, scientific activities, and contributions to clinical research, pharmacovigilance, Good Clinical Practice (GCP), ethics, and patient safety.

Both organizations discussed opportunities for future collaboration, including **joint educational webinars, scientific exchanges, participation in IUPHAR working groups and international projects, and greater involvement of Indian pharmacologists in global Pharmacology and Therapeutics initiatives.** IUPHAR also acknowledged India's strong tradition in Clinical Pharmacology and recognized the value of an organization dedicated to patient care and therapeutics.

NPT remains committed to expanding national and international academic partnerships that promote excellence in Pharmacology and Therapeutics for the benefit of healthcare professionals, researchers, students, and patient care.

The National Association of Pharmacology and Therapeutics (NPT) expresses its sincere gratitude to Prof. Caroline Samer, President, IUPHAR, and Prof. Emilio Clementi, Secretary General, IUPHAR, for their warm reception, valuable time, and keen interest in understanding the vision, activities, and future aspirations of NPT. The Association also conveys its appreciation to Ms. Alicia Goia and Ms. Alessia Costanzo for their kind support and coordination in facilitating this important interaction. NPT looks forward to strengthening this academic relationship through future collaborative initiatives for the advancement of Clinical Pharmacology and Therapeutics.

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Founder & National Coordinator
National Association of Pharmacology and Therapeutics (NPT), India
Website: www.nationalpharmacology.org
Contact : 9528540756

Enclosure: Copy of minutes of meeting received from IUPHAR

Rx Factor Glimpse
PHARMA PRAXIS-1: Translating Pharmacology into Practice (2026)

The Department of Pharmacology & Therapeutics, AIIMS Nagpur, successfully conducted the four-day national workshop "PHARMA PRAXIS-1: Translating Pharmacology into Practice" from 7–10 April 2026 under the aegis of the National Association of Pharmacology and Therapeutics (NPT). The programme was inaugurated by Dr Prashant P. Joshi, Executive Director & CEO, AIIMS Nagpur, who emphasized the need to bridge the gap between theoretical knowledge and clinical practice through competency-based, skill-oriented learning. The inaugural ceremony was attended by Dr. Ganesh Dakhale (President-NPT & Organizing Chairperson), Dr. Chaitali Chindhalore (Organizing Secretary), and distinguished NPT office bearers, Dr. Parveen Sharma (General Secretary), Dr. C. M. Kamaal (National Coordinator) and MajorDr. Jeetendra Singh (Head of the Academic Board), along with faculty members and delegates.

The organizing committee gratefully acknowledges the constant guidance and encouragement of the NPT leadership and sincerely appreciates the dedicated efforts of the departmental faculty, postgraduate students, and supporting staff whose commitment ensured the successful conduct of the workshop.

The four-day workshop featured interactive, hands-on sessions delivered by departmental faculty—Dr Anant Khot, DrSnehlata Gajbhiye, Dr Khushboo Bisht—along with eminent national experts from premier academic institutions, hospitals, and the pharmaceutical industry, including Dr Puneet Dhamija, Dr Nithya Gogtay, Dr Shoibal Mukherjee, Dr Avinash Turankar, Dr Ashish Kakkar, DrRishikesh Deshpande, Dr Vipul Choudhary, Dr Biswadeep Das, Dr Santosh Taur, Dr Kiran Haridas. Participants received competency-based training spanning the entire drug development continuum, including lead identification, preclinical and clinical research, protocol design, biostatistics, innovative clinical trial methodologies, pharmacoeconomics, pharmacovigilance, pharmacokinetics, therapeutic drug monitoring, real-world evidence, biologics and biosimilars, vaccine trials, research ethics, and career opportunities in academia and the pharmaceutical industry. The program was enriched through protocol development exercises, group activities, case-based discussions, hands-on statistical training, protocol presentations, and demonstrations of drug screening models, thereby strengthening participants' research aptitude and facilitating the translation of pharmacological principles into evidence-based clinical and academic practice. Quiz-o-Pharm was conducted by Dr Mrunalini Kalikar and Dr Chaitali Chindhalore.

The workshop witnessed enthusiastic participation from 56 delegates representing premier medical institutions across India and received highly positive feedback for its academic excellence, practical orientation, and interactive learning environment. PHARMA PRAXIS-1 stands as a testament to AIIMS Nagpur's commitment to advancing competency-based pharmacology education and fostering research-oriented training for the next generation of pharmacologists.















NATIONAL ASSOCIATION OF PHARMACOLOGY AND THERAPEUTICS

Promoting Pharmacology and Therapeutics for a better tomorrow

About the organization

A national organization of medical doctors specialized in pharmacology /clinical pharmacology and therapeutics. Envisaged to provide strong leadership to promote pharmacology and therapeutics for a better tomorrow. The association is fostered by NMDP (National MD Pharmacology), a prestigious group of eminent pharmacologists.

Aims and objectives

Empowering medical doctors specialized in Pharmacology/Clinical Pharmacology and Therapeutics.

Promoting academic and clinical research in Pharmacology/Clinical Pharmacology and Therapeutics.

Enhancing the standard of teaching/training in Pharmacology/Clinical Pharmacology and Therapeutics

Promoting Pharmacology/Clinical Pharmacology and Therapeutics for the benefit of patients and society.

BENEFITS OF LIFE MEMBERS

Receive notifications on of the organization

Keep yourself updated in the world of pharmacology and therapeutics .

Get connected with fellow pharmacologists of the country.

Contest for various posts in the organization.

Receive of the permanent membership e-certificate through email, enhance your profile by writing MNPT

Participate in general body meetings (GBM) to speak and to vote.

Participate in conferences/seminars/workshops/symposiums/training sessions at subscribed charges.

Receive an e-copy of the official publications (i.e. News letter, Journal, academics, research material etc.

NATIONAL ASSOCIATION OF PHARMACOLOGY AND THERAPEUTICS

Promoting Pharmacology and Therapeutics for a better tomorrow

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Rx FACTOR

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