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Editorial

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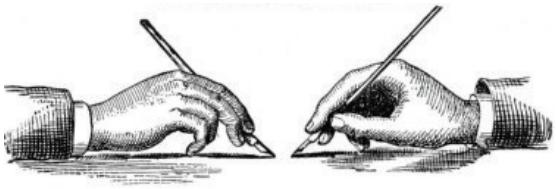

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Warm greetings to all.

Welcome to the Issue 12 of 'RxFactor', from the NATIONAL ASSOCIATION OF PHARMACOLOGY AND THERAPEUTICS (NPT). RxFactor has been designed to encompass the range and breadth of Pharmacology and therapeutics ranging from Medical Education, Pharmaco-vigilance, Research and Therapeutics. Previous editions of Rxfactor have been well received and we thank you all for the words of encouragement and appreciation.

This edition of the Rxfactor newsletter is packed with informative articles. Article titled: Cross Talk Among Cellular Signaling Pathways delves into how various pathways that intersect and influence one another, providing insights into potential clinical applications and treatment strategies.

Advancements in Heart Failure Management reviews the top five clinical trials of 2024 that have shown promising advancements in managing heart failure, highlighting innovative treatments and improved patient outcomes.

The OSCE has revolutionized medical education by providing a structured and objective framework for clinical assessments. The present issue has an article with a very meticulously designed OSCE.

Engaging additions include Impact of Social Media on Medical Students which examines the positive and negative impacts of social media on students shedding light on how it influences their academic performance, mental health, and social interactions. Network pharmacology offers a paradigm shift in drug discovery by focusing on multi-target drugs that can address complex diseases more effectively.

Other notable inclusions are RISDIPLAM: The First In-Utero Treatment for Spinal Muscular Atrophy (SMA), Landiolol an ultra-short acting adrenergic antagonist and extracts from the NMDP WhatsApp Group. The Cool Corner offers crosswords and cartoons for some brain-storming fun.

We extend our heartfelt gratitude to all contributors who have made this issue of RxFactor a grand success. Special thanks to the PG students, whose contributions underscore their potential as future leaders in our specialty. We anticipate a fruitful journey of education and mutual learning with all our readers.

We look forward to a happy education and mutual learning with all our readers.

Jai Hind.

Cross talk among cellular signaling pathways and its clinical relevance

A: What is cross talk?

National Cancer Institute describes cross talk as 'the process inside a cell that occurs when the same signal is shared by two or more signaling pathways. Usually, a signal caused by the binding of a substance to a molecule on or inside a cell is passed from one molecule to another in the same pathway'. A signal transducing system may provide input to another related or unrelated signal system to alter its activity. For example, such communication between c'AMP pathway and MAPK pathway (cross talk) may result in activation or inhibition of MAPK. Other examples are GPCR regulated c'AMP pathway cross talks (integrates) with Ca^{++} channels signaling in excitable cells. In cardiac myocytes, activation of the β1 receptor - Gs-adenylyl cyclase-cyclic AMP–PKA pathway increases cardiac contractility not only by augmenting Ca^{++} entry through voltage-gated Ca^{++} channels but also by Ca^{++} release from sarcoplasmic reticulum via the ryanodine receptor activation. Signals from multiple pathways are frequently integrated within the responsive cell so that ample cross talk occurs among multiple signaling pathways. Gq/11 → Gs cross talk with AT1 receptors couple to Gq/11 to activate the phospholipase (PL) Cβ-IP3-Ca2+ pathway. A cross talk between Gq/11 → Gs results in enhanced c'AMP production.

Glossary

Crosstalk	=	Cross talk or Cross-talk
CBP	:	c'AMP response element binding (CREB)-binding proteins
EGF	:	Epidermal growth factor
ERK	=	Extracellular signal Regulator protein
GEF	:	guanosine nucleotide exchange factor
IGF-1	:	Insulin like growth factor-1
MAPK	:	Mitogen activated protein kinase
MEK	=	MAP kinase kinase
NF-kB	:	nuclear factor kappa B
Nrf2	:	Nuclear Factor Erythroid (NF-E2)-Related Factor 2
Raf	:	A proto-oncogene kinase (Rapid accelerated fibrosarcoma)
Ras	:	Rat Sarcoma virus (GTPase)
ROCK	:	Rho kinase
TGR-5	:	Takeda G-protein coupled receptor-5
ROCK	:	Rho kinase
TGR-5	:	Takeda G-protein coupled receptor-5

B: Cross talk in cells and tissues

1: Gut-Liver Cross talk

Bile acids mediate such cross talk. The gut microbes (Lactobacillus, Clostridium, and Bifidobacterium spp) deconjugate taurine and glycine conjugates (glycocholate and taurocholate) by deconjugating hydrolases. Microbes also convert cholic acid into deoxycholic acid. The resultant bile acids act as signaling molecules as they have ability to bind to nuclear receptors such as the farnesoid X receptor (FXR), and TGR5 (a GPCR activated by bile acids). By binding with FXRs, bile acids alter CYP3A activity and may modify drug metabolism.

2: Target organ-immune system cross talk

The immune system has significant cross talk with other organ systems and chemicals. This cross talk results in alterations in bio activation of immunotoxins that act as Endocrine Disruptor Chemicals (EDCs) that indirectly impair immune function.

3: Ligand independent estrogen receptor (ER) activation

It is now known that ERs are localized not only on nuclear membrane but are also present as membrane ERs. These membrane-localized ERs mediate the rapid activation of some proteins such as MAPK by phosphorylation and this explains rapid increase in cyclic AMP caused by the estrogen. The finding that MAPK is activated by estradiol provides an additional level of cross talk and multiplicity of estrogen signaling. In addition, a cross talk between membrane-bound receptor pathway (i.e., EGF/IGF-1) results in simultaneous activation of MAPK and the nuclear ERs immediate ligand independent target ER genes.

4: Cross talk in cancer cell signaling pathways

Cross talk between cancer cells and the host stroma (including the vasculature and immune cells) is modulated through the release of angiogenic factors and the expression of immune checkpoint proteins.

The signaling cascade of RAS-RAF-MEK-ERK that plays a central role in cell proliferation and differentiation, is activated in many human cancers, and controls the cross talk of cancer cells with the tumor microenvironment. The concept of cross talk is also utilized to develop pathway targeted inhibitors as anticancer drugs such as sotorasib. Histone deacetylase (HDAC) is over expressed in some cancer cells. Impact of HDAC inhibitors on the crosstalk of cancer cells and the immune system is currently evaluated in trials using combination treatments with immune checkpoint inhibitors.

5: Cross talk between arginine-vasopressin (AVP) and oxytocin hormone receptors

AVP and oxytocin activate each other receptors in brain and periphery. In brain receptor cross-talk may be involved in mediating at least some of shared behavioral effects. Peripherally administered oxytocin and AVP produce similar effects in reducing heart rate and body temperature, inducing contractions in ejaculatory tissues, and in promoting seizure susceptibility in male rats and rabbits. These results show that oxytocin and AVP cross talk at receptor levels (Vi receptors mainly).

6: Cross talk among Gs signaling, MAP kinase, integrin and ROCK pathways

G protein–coupled receptors can also stimulate cell proliferation, because their intracellular pathways can connect with the Ras/kinase cascade. The Gα subunit can interact with a guanosine nucleotide exchange factor (GEF), which facilitates GDP–GTP exchange at another GTPase, *Rho*. Activated *Rho*-kinase phosphorylates many proteins and controls a large number of cellular functions, including smooth muscle contraction and proliferation, cell movement and migration, angiogenesis and synaptic remodeling.

7: Cross talk between integrins and growth factor pathways

Integrin activation inhibits apoptosis. Auto-phosphorylation of growth factor receptors is enhanced by integrin activation. In addition, the integrins work together with growth factors to control angiogenesis signal integration connecting integrins and cytokine receptors for interleukins.

C: Implications of Cross talk

1: Cross talk and drug resistance: Drug resistance is a clinical problem with use of targeted treatment of cancer. The Intracellular signaling pathways link the cellular genome to the extracellular microenvironment that enable dynamic modulations of signal transduction network involved in oncogenesis by generating post translational factors for oncogene proliferation.

2: Antiestrogen therapy of breast cancer: The response rate to antiestrogen treatment is in the subset of patients with tumors that are ER+ or PR+ but also positive for human epidermal growth factor receptor (HER2)/neu amplification. This is because of a cross talk between the ER and the HER2/neu pathway that has been implicated in tamoxifen resistance.

3: Cross talk and organ toxicity

3a: Drug induced hepatotoxicity: Nrf-2 is key nuclear mediator of organ defense against oxidant injury and inflammation. The NF-κB family, a regulator of κB light chain expression in mature B- lymphocytes and plasma cells, regulates many genes involved in different cellular processes, such as cell differentiation, proliferation, development, and apoptosis. NF-κB Inhibits the Nrf2 Pathway and Nrf2 Inhibits the NF-κB Pathway. Nrf2 gene has an NF-κB binding site and loss of Nrf2 may induce more aggressive inflammation by activating NF-κB and downstream pro-inflammatory cytokines. In drug induced liver disease (DILI), primary hepatocytes show NF-κB inhibition coinciding with a strong Nrf2 response and enhanced hepatocytic pro-apoptotic signaling cascades. Isoniazid (INH) reduces the phosphorylation of ERK1 and prevents Nrf2 translocation into the nucleus. Increased cytosolic concentration of Nrf-2 leads to loss of hepato-protection of cellular elements and hepatic oxidant injury.

3b: Drug induced nephropathy (cisplatin, gentamicin and contrast media): Nrf2 and NF-κB pathways can be simultaneously regulated in gentamicin-induced nephrotoxicity models, and which provides direct evidence between the cross talk of Nrf2 and NF-κB and drug-induced toxicity. A renal protective drug could increase the activation of the Nrf2 antioxidant defense pathway. Melatonin (MT) restores antioxidant enzyme activity and blocks NF-κB and nitric oxide synthase (iNOS) activation in rat kidneys, thereby preventing gentamicin-induced nephrotoxicity

3c: Doxorubicin cardiotoxicity is mediated by ROS production via Nrf2 NF-kB cross talk. cardamonin (CAR), a flavone found in Alpinia plants, can reduce the NF-

κB signaling pathway and improve Nrf2 signaling to suppress oxidative stress, apoptosis, and inflammatory response

D: Drugs Affecting Cross talks

1: Metformin: Metformin exhibited unexpected indirect anticancer activity. It affects insulin cross talk with GPCR signaling in cancer cells changing Ca++ mobilization, mTOR activation and DNA synthesis. Experimentally, metformin prevents growth of pancreatic cancer in rats and hamsters. Metformin is known to activate AMPK and that activation inhibits mTOR.

2: Ketamine: While its main mechanism of action is inhibition of central NMDA receptors, it can cross talk with Ca++ signaling pathway involved in Ca++ 'turn-on' and 'turn-off' in neurons. It is also known to alter cross talk between pathways involving Receptor Tyrosine kinases in cancer cells and such findings have implication in cancer therapy. The pathways involved are NF-kB, MAPK, P13/mTOR signals. The antidepressant action could be related to ketamine's cross talk with NF-kB pathways.

Conclusions: Exciting developments in signaling pathways triggered by discovery of G-proteins and GPCRs have provided crucial links of communications among intracellular signaling pathways and extracellular microenvironment concerned with mediation of physiological responses (Gp cascade), cell differentiation (ROCK, MAPK) and maintaining integrity (integrins) and functionality. Cross talk has become an increasingly utilized avenue to identify interventions and drugs to reduce toxicity and demote oncogenesis. A dedicated database for cross talk speaks about its pivotal role.

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Advancements in Heart Failure Management: A Review of the Top 5 Trials in 2024

Introduction

Heart failure (HF) remains one of the leading causes of morbidity and mortality worldwide, necessitating continuous advancements in treatment strategies. The past year has seen the publication of several pivotal clinical trials that have reshaped our understanding and management of HF. This review highlights five of the most influential studies in 2024, covering novel pharmacologic treatments, device-based interventions, and advanced monitoring techniques. The selected studies include **FINEARTS-HF, STEP HFpEF-DM, RESHAPE-2HF, MONITOR-HF** and **ELEVATE Registry**. These trials address critical gaps in HF management and provide evidence for improved patient outcomes.

1. Finerenone in HFpEF (FINEARTS-HF Trial) [1]

Background

Heart failure with preserved ejection fraction (HFpEF) presents a significant therapeutic challenge due to the lack of effective guideline-recommended treatments. Finerenone, a novel nonsteroidal mineralocorticoid receptor antagonist (MRA), was evaluated in the FINEARTS-HF trial to assess its efficacy in reducing HF-related events in patients with HFpEF and mildly reduced ejection fraction (HFmrEF).

Study Design & Methods

- Population: Over 6000 patients with HFpEF/HFmrEF.
- Intervention: Finerenone vs. placebo.
- Primary Endpoint: Composite of HF hospitalization or cardiovascular (CV) death.

Key Secondary Endpoints: Changes in serum potassium levels, incidence of hyperkalemia and hypokalemia.

Key Findings

- Finerenone reduced total worsening HF events (P = .006).
- The cardiovascular mortality rate was similar between the two groups (P = .07).

Increased hyperkalemia risk but a lower incidence of hypokalemia was observed.

Clinical Implications

The trial highlights finerenone as a potential treatment for HFpEF, particularly in reducing HF-related hospitalizations, although close electrolyte monitoring is necessary.

2. Semaglutide in HFpEF with Obesity (STEP HFpEF-DM Trial)[2]

Background

Obesity is a major contributor to HFpEF, and lifestyle interventions often fail to achieve sustained weight loss. STEP HFpEF-DM evaluated semaglutide, a GLP-1 receptor agonist, for its role in improving functional capacity and quality of life in obese HFpEF patients.

Study Design & Methods

- Population: Over 600 patients with HFpEF, obesity (BMI ⩾30), and type 2 diabetes (T2DM).
- Intervention: Weekly semaglutide (2.4 mg) vs. placebo.
- Primary Endpoints:
- Change in body weight.
- Change in Kansas City Cardiomyopathy Questionnaire Clinical Summary Score (KCCQ-CSS).
- Secondary Endpoints:
- 6-minute walking distance (6MWD).
- C-reactive protein (CRP) levels as an inflammatory marker.

Key Findings

- Semaglutide significantly reduced body weight (-9.8% vs. -3.4%, P < .001).
- Improved KCCQ-CSS scores (13.7 vs. 6.4 points, P < .001).
- Secondary benefits included increased exercise capacity and reduced systemic inflammation.

Clinical Implications

While the trial was not powered for HF hospitalization outcomes, it suggests semaglutide as a promising therapy for improving symptoms and functional status

in obese HFpEF patients.

3. Transcatheter Mitral Valve Repair in Functional Mitral Regurgitation (RESHAPE-2HF Trial)[3]

Background

Mitral regurgitation (MR) worsens HF symptoms and is associated with higher hospitalization and mortality rates. The RESHAPE-2HF trial examined the impact of transcatheter mitral valve repair (M-TEER) in patients with moderate-to-severe MR and HF.

Study Design & Methods

- Population: 505 patients with HF and moderate-severe MR.
- Intervention: M-TEER + guideline-recommended medical therapy (GRMT) vs. GRMT alone.
- Primary Endpoints:
- Total HF hospitalization or CV death at 24 months.
- Change in KCCQ Overall Summary Score (OS) at 12 months.

Key Findings

- Reduced HF hospitalizations and CV mortality (Rate Ratio: 0.64, $P = .002$).
- KCCQ-OS scores improved significantly in the M-TEER group (+21.6 vs. +8.0, $P < .001$).

Clinical Implications

RESHAPE-2HF confirms the benefit of M-TEER, making it a viable intervention for HF patients with significant MR.

4. Pulmonary Artery Pressure Monitoring in HF (MONITOR-HF Trial) [4]

Background

PA pressure monitoring has been explored as a means of preventing HF hospitalizations, but its benefit in different patient subgroups remained unclear. The MONITOR-HF trial examined its effectiveness across multiple subgroups.

Study Design & Methods

- Population: Patients with NYHA Class III HF.
- Intervention: PA pressure-guided therapy vs. standard care.
- Primary Endpoints:
- Changes in HF hospitalizations.
- Impact on quality of life.

Key Findings

- Benefits of PA monitoring were observed in all subgroups.

  Reduced HF hospitalizations with consistent improvements in quality of life.

Clinical Implications

PA monitoring remains a valuable tool in advanced HF management, particularly in high-risk patients.

5. Long-Term Outcomes of LVADs (ELEVATE Registry)

Background

LVADs provide hemodynamic support for end-stage HF patients. The ELEVATE registry examined 5-year outcomes of HeartMate 3 (HM3) LVADs.

Study Design & Methods

- Population: 463 patients implanted with HM3 LVADs.
- Endpoints:
- Overall survival at 5 years.
- Stroke-free survival.

Functional capacity and quality of life improvements.

Key Findings

- 63.3% overall survival at 5 years.
- 58.1% survival free of stroke.

Substantial improvements in functional capacity and quality of life.

Clinical Implications

The **ELEVATE registry confirms HM3 as an effective long-term therapy for end-stage HF**. (Table 1)

Conclusion:

These five trials have significantly advanced HF management, providing new pharmacologic, device-based, and monitoring strategies. Future guidelines may integrate these findings, improving long-term outcomes for HF patients.

Trial	Condition	Intervention	Primary Endpoint	Key Finding
FINEARTS-HF	HFpEF	Finerenone vs. placebo	CV death/HF hospitalization	↓ HF events (P = .006)
STEP HFpEF-DM	HFpEF + Obesity	Semaglutide vs. placebo	Weight loss, KCCQ score	-9.8% body weight, ↑ KCCQ score
RESHAPE-2HF	HF + MR	M-TEER vs. GRMT	HF hospitalization, CV death	↓ Hospitalizations (P = .002)
MONITOR-HF	HF (NYHA III)	PA pressure monitoring vs. standard care	HF hospitalization, QoL	↓ HF events, QoL benefits
ELEVATE	End-stage HF	HM3 LVAD	Survival, QoL	63.3% survival at 5 years

Table 1: Summary Table of Key Trials

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Objective Specific Clinical Examination (OSCE)

OSCE 1:

Therapeutics OSCE - High-Dose Insulin Therapy in a Non-Diabetic Patient

Case Scenario:

During routine therapeutic rounds, a 76-year-old non-diabetic patient was found to be receiving 190 units of insulin over the last 3 hours. The patient's potassium levels were within normal limits, and the prescription was deemed rational by the medical team.

OSCE Structured Question & Answer

Station Instructions for Examiner:

- Assess the candidate's ability to recognize and manage high-dose insulin therapy (HDI) in a non-diabetic patient.
- Evaluate understanding of the indication, pathophysiology, monitoring, and stepwise management of HDI.
- Ensure clear, structured, and clinically sound responses.

Candidate Questions & Model Answers

1. What is the most likely therapeutic indication for high-dose insulin therapy (HDI) in this patient, given that they are non-diabetic?

- The most likely indication is Calcium Channel Blocker (CCB) Toxicity leading to hemodynamic instability (hypotension, bradycardia, and myocardial depression).
- HDI is used in CCB overdose to enhance myocardial contractility and increase cardiac output.
- It may also be used in β-blocker toxicity, but in this scenario, CCB toxicity is the more probable cause.

2. What are the pathophysiological mechanisms behind the use of HDI in this scenario?

- Counteracts Myocardial Depression: HDI enhances intracellular glucose uptake, fueling cardiac myocytes.
- Positive Inotropic Effect: Insulin increases calcium influx into myocardial cells, improving contractility.
- Reduces Catecholamine Requirement: HDI decreases the need for vasopressors in hemodynamically unstable patients.
- Vasodilation & Perfusion Enhancement: Insulin facilitates vasodilation and tissue perfusion, improving hemodynamic stability.

3. What are the key steps for the safe administration and monitoring of HDI in a non-diabetic patient?

Initiation of Therapy:

- Initial Bolus: 1 unit/kg regular insulin IV bolus.

Continuous Infusion: Start at 1–2 units/kg/hr, titrated based on response.

Concurrent Dextrose Infusion:

- To prevent hypoglycemia, administer:
  50 mL of D50 IV bolus, then

Continuous infusion of D10 or D20, adjusted based on glucose monitoring.

Close Monitoring:

- Blood Glucose: Every 15–30 minutes initially, then hourly (target 100–250 mg/dL).
- **Electrolytes (Potassium, Magnesium, Phosphate):** Every **1–2 hours** (risk of hypokalemia).
- **Cardiac Function:** Continuous ECG monitoring for arrhythmias or conduction delays.
- **Hemodynamic Stability:** Frequent blood pressure and cardiac output monitoring.

4. Considering the patient's age, what are the potential complications of HDI, and how should they be mitigated?

Potential Complications:

1. **Hypoglycemia** – Insulin can **rapidly lower blood glucose**, leading to confusion, seizures, or coma.

- **Mitigation:** Ensure adequate **dextrose infusion** and frequent **glucose checks**.
- **Hypokalemia** – Insulin shifts

potassium **intracellularly**, leading to arrhythmias.

- **Mitigation:** Monitor **serum potassium closely**; replace if <3.0 mEq/L.
- **Volume Overload** – Large fluid volumes from **dextrose infusion** may cause **pulmonary edema**.
- **Mitigation:** Use **diuretics if needed**, and monitor **fluid balance**.
- **Rebound Hypotension or Bradycardia** – Occurs if **HDI is abruptly discontinued**.
- **Mitigation:** Gradual tapering of insulin infusion over **several hours**.

5. If the patient exhibits no improvement or deteriorates despite HDI, what adjunctive therapies should be considered next?

1. Vasopressors:

- Norepinephrine for vasodilatory shock.
- Epinephrine or dopamine if cardiac output remains low.

2. Calcium Therapy:

- Calcium chloride or calcium gluconate IV to enhance myocardial contractility.

3. Lipid Emulsion Therapy (LET):

- Effective in lipophilic CCB toxicity (verapamil, diltiazem).

4. Methylene Blue:

- Used in refractory vasoplegic shock due to CCB toxicity.

5. Extracorporeal Membrane Oxygenation (ECMO):

- Considered in cardiogenic shock if all other treatments fail.

6. What specific factors must be considered before discontinuing HDI in such a patient?

- Ensure Hemodynamic Stability:
- Mean arterial pressure (MAP) > 65 mmHg.
- Heart rate > 50 bpm.
- Systolic BP > 100 mmHg.
- Gradual Weaning of Insulin Infusion:
- Reduce insulin infusion gradually over 12–24 hours to prevent rebound hypotension.
- Continued Monitoring for Delayed Toxicity:
- Some CCB formulations are extended-release; continued close monitoring is essential.
- Electrolyte and Glucose Normalization:

Ensure glucose, potassium, and magnesium are stable before stopping insulin therapy.

Assessment Criteria for OSCE Examiner:

Competency Checklist:

- Correct identification of CCB toxicity as the indication for HDI.
- Clear explanation of HDI mechanism and rationale.
- Stepwise administration and monitoring plan.
- Identification of complications and risk mitigation strategies.
- Proper escalation plan if HDI fails.
- Safe discontinuation strategy for HDI.

Final Notes for OSCE Candidates:

This station evaluates critical thinking in managing HDI therapy, focusing on safe administration, monitoring, and escalation of therapy.

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Impact of Social Media on Medical Students at BRD Medical College, Gorakhpur, Uttar Pradesh

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INTRODUCTION- Social media plays a crucial role in the lives of medical students, influencing their academic performance, mental health, and professional development. Social media has significantly transformed communication and learning methodologies. In medical education, it provides opportunities for resource sharing, professional networking, and interactive learning. However, concerns arise regarding potential distractions, social media addiction, and its impact on academic performance and mental health. This study explores the impact of social media usage among medical students in Uttar Pradesh at BRD Medical College, Gorakhpur.

Objectives

- To analyze social media usage patterns among medical students at BRD Medical College, Gorakhpur.
- To assess its benefits and drawbacks in medical education and student well-being.
- To present statistical trends based on recent studies conducted in Uttar Pradesh.

Methodology

Study Design: Cross-sectional survey.

Participants: 300 medical students from BRDMC

Data Collection: Structured questionnaires (online& offline) and secondary analysis of recent studies.

Variables Assessed: Duration and purpose of social media use, academic performance, and health effects.

Results

Social Media Usage Trends:

- **Average Usage:** 72% of students use social media for 2-4 hours daily.
- **Primary Platforms Used:** WhatsApp (95%), YouTube (89%), Facebook (65%), Instagram (75%), Twitter and Telegram (30%).
- **Purpose of Use:** Academic Learning (68%), Social Networking (82%), Research & Case Studies(45%), Entertainment(76%)

Impact on Academic Performance:

Positive Effects:

- 60% found it useful for accessing easy concepts and medical discussions.
- 52% improved conceptual understanding through medical YouTube channels.

Negative Effects:

- 40% experienced decreased study efficiency due to

distractions.
- 30% reported lower grades correlated with excessive usage.

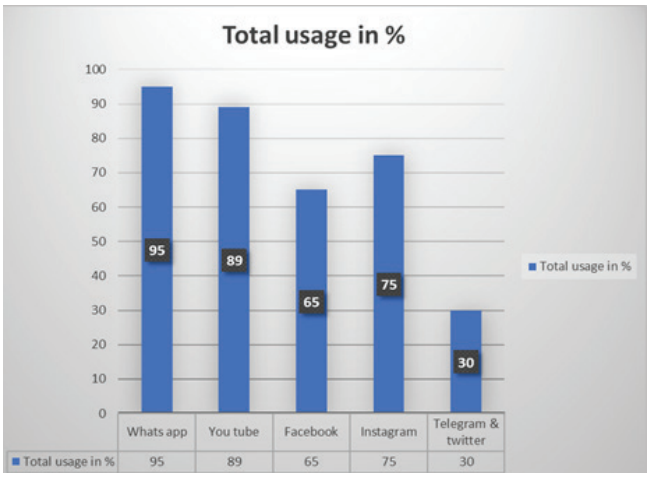
Misinformation: Spread of unverified medical information.

Mental and Physical Health Effects:

- Sleep Disturbances: Reported by 58% of students who use social media late at night.
- Increased Stress and Anxiety: Found in 48% of students using social media excessively.
- Screen Time-Related Issues:
- Eye Strain: 39%
- Neck/Back Pain: **31%**
- Reduced Physical Activity: **45%**

Recommendations:

- **Regulated Usage:** Implementing structured screen time schedules.
- **Educational Integration:** Encouraging the use of verified medical platforms such as PubMed, Medscape etc.
- **Awareness Campaigns:** Conducting workshops on digital well-being and social media literacy.
- **Institutional Guidelines:** Establishing policies for ethical and professional social media use.



Discussion

Social media is a double-edged sword in medical education. While it enhances learning, networking, and accessibility to medical resources, excessive use results in reduced study efficiency, stress, and health issues. A balanced approach incorporating time management strategies, institutional guideline, digital detox, and awareness programs can help mitigate negative effects.

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Network Pharmacology: One Drug Multiple Targets

Dr. Nidhi Mahlawat
Junior Resident DRPGMC, Tanda (Kangra)

Network Pharmacology: One Drug Multiple Targets

A game-changing method in drug discovery, network pharmacology (NP) successfully connects reductionist models with all-encompassing treatment approaches. For multi-factorial diseases like cancer, neurodegenerative disorders, and infectious diseases, NP promotes a multi-target, system-wide approach instead of the traditional one-drug, one-target model.

How Network Pharmacology is Changing the Game

The foundation of network pharmacology is the aim that diseases result from interrelated biological networks rather than from malfunctions in a single molecular structure. This approach systematically maps the interactions between drugs, proteins, genes, and disease pathways. NP offers a more comprehensive understanding of drug action and disease progression by utilizing large-scale databases, computational biology, and omics technologies. In recent years, network pharmacology has advanced significantly due to advanced technology and a greater comprehension of intricate biological systems.

Combining machine learning and artificial intelligence (AI)

Drug discovery is being transformed by AI-powered

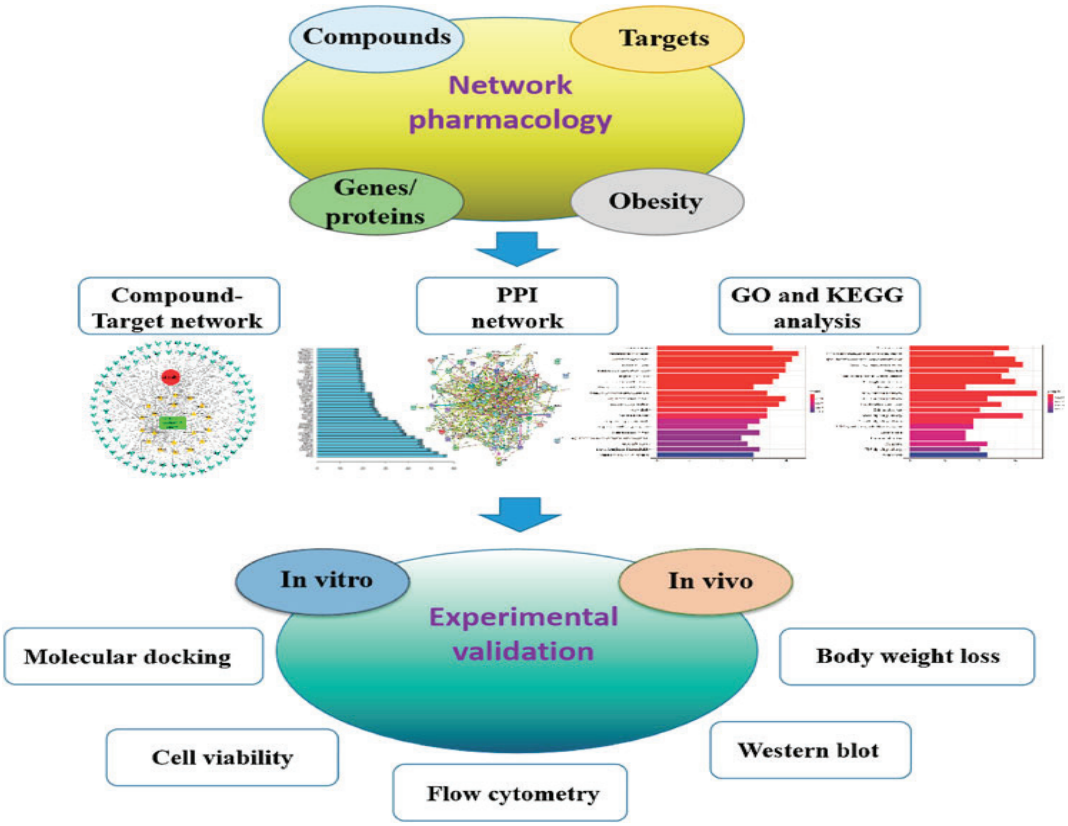

Image Courtesy:
doi: 10.3389/fphar.2020.572387

network pharmacology, which analyses enormous biological datasets with previously unheard-of speed and accuracy. Nowadays, artificial intelligence algorithms are used to model complex biological networks to understand disease mechanisms.

Emerging Computational Tools in NP

In NP research, several AI-powered tools are being employed, such as:

DeepChem: A deep learning framework for predicting molecules and finding new drugs.

STITCH: A database that incorporates chemical-protein interactions.

BindingDB: A publicly available online database of measured binding affinities for interactions between drugs and their targets. These tools accelerate the identification of potential drug candidates, strengthening NP's ability to predict therapeutic outcomes with greater accuracy.

Network Pharmacology in Drug Combination Therapy

Deep generative models have allowed, scientists to create the best possible drug combinations that target several disease pathways. This is particularly important for treating cancer and antibiotic resistance, as drug resistance frequently causes single-target therapies to fail.

Immunotherapy Advancements

By mapping immune system interactions, network pharmacology (NP) has helped discover PD-1/PD-L1 inhibitors other than traditional monoclonal antibodies, which has been a significant factor in the success of new checkpoint inhibitors for cancer immunotherapy.

Network Ethnopharmacology and Traditional Medicine

Network Ethnopharmacology is essential to the evidence of traditional medical systems such as TCM and Ayurveda. Nowadays, researchers are:

- Mapping the bioactive ingredients of herbal remedies to particular disease targets using NP.
- Find new uses for natural products that already exist.

Drug Discovered Through Network Pharmacology

Berberine, an alkaloid derived from plants, discovered through network pharmacology analysis for its multi-target effects in treating cancer, cardiovascular diseases, and metabolic disorders, is one of the most recent advances in NP-based drug discovery. Since NP has made its interactions with several signaling pathways more apparent, it is a viable option for repurposing therapeutic applications.

Network Pharmacology vs. Other Computational Approaches

Computational systems biology focuses on cellular reactions and disease modeling. It aids in the understanding of disease mechanisms, but unlike NP, it does not incorporate drug-target interactions.

Cheminformatics and Bioinformatics use structural analysis, QSAR models, and molecular docking to perform virtual screening. In contrast to NP, which charts drug interactions in biological networks, this method mainly concentrates on chemical characteristics.

Conclusion

Network pharmacology (NP) transformed drug discovery by enabling precision medicine, drug repurposing, and personalized therapeutics. By integrating AI, multi-omics data, and computational modeling, NP shifts the focus from a linear, single-target approach to a dynamic, systems-based paradigm. As we advance into 2025, NP will continue to drive innovations in pharmaceutical sciences, facilitating the development of more effective, safer, and targeted therapies for complex diseases. As AI and big data progress to evolve, network pharmacology is poised to reshape medicine.

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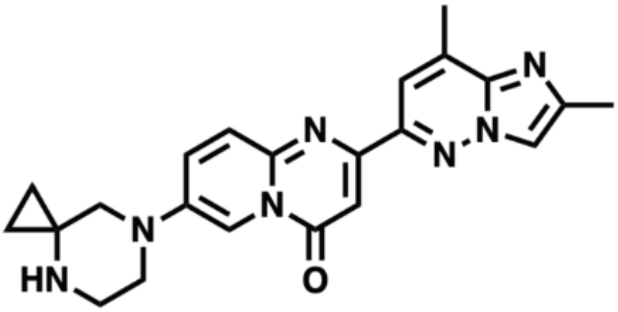
Risdiplam: The First In-Utero Treatment for Spinal Muscular Atrophy(Sma)

INTRODUCTION

Scientists at St. Jude Children's Research Hospital have developed the first in-utero treatment for spinal muscular atrophy (SMA).Risdiplam, an oral drug, administered to the expectant mother during the final six weeks of pregnancy gave birth to a baby with no signs of SMA even after two years of birth. SMA, the principal genetic cause of infant death, is a neurodegenerative disease of the anterior horn of the spinal cord and lower brainstem neurons. Loss of these motor neurons results in the typical non-progressive weakness of SMA.Spinal muscular atrophy is inherited in an autosomal recessive pattern, resulting from most classically homozygous deletions of the survival motor neuron 1 (SMN1) on chromosome 5q13.2 in 92% of cases.

SMN2 mRNA splicing modulator



- First approved small molecule splicing modulator for Spinal Muscular Atrophy
- Induces splicing to include exon 7 of *SMN2* to elevate levels of functional *SMN1*

SMA is caused by a lack of survival motor neuron protein and occurs in around 1 in every 11,000 births in the United States. If not treated, SMA type 1 (SMA-1), the most common and severe form, results in progressive muscle weakness that leads to death. Currently, treatments for SMA-1 have demonstrated improved survival and motor function in infants, especially if administered shortly after birth before symptoms begin, but is not a cure

EARLY SMA TREATMENT IN THE WOMB

Survival motor neuron protein is most needed in the third trimester of foetal development and the first three months of life after birth. Symptom severity is, therefore, closely linked with the intervention time point. The St. Jude researchers, as part of the Paediatric Translational Neuroscience Initiative, launched a unique clinical protocol to study Risdiplam in a single patient. The goal was to determine the likelihood of starting treatment of SMA-1 *in-utero*. The parents in this case were both known carriers of SMA genetic variants and had a prior infant born with SMA-1 before current treatments became available, who died at 16 months of age. Genetic testing conducted by amniocentesis confirmed the foetus had no copies of the survival motor neuron gene, which, shared with

the family history and other genetic information, was highly predictive of the infant being born with SMA-1. Risdiplam was administered to the expectant mother during the final six weeks of pregnancy.

CHILD BORN SMA-FREE AFTER PRENATAL TREATMENT

Shortly after birth, the infant was diagnosed with three developmental abnormalities: ventricular septal defect ,optic nerve hypoplasia,and a brainstem asymmetry,with related delays in vision and overall development. These abnormalities are considered to have occurred early in foetal development before exposure to Risdiplam. Now two-and-a-half years old, the child continues to be monitored periodically at St. Jude Children's Research Hospital and there were no signs of SMA found. The findings were published on February 19 in a letter to the *New England Journal of Medicine*.

Dr. Mitra Bhattacharyya,
Assistant Professor,
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LANDIOLOL: Ultra-short-acting adrenergic receptor antagonist.

FDA Approved: First approved November 22, 2024
Brand name: Rapiblyk
Generic name: Landiolol
Dosage form: Lyophilized Powder for Injection

Landiolol is an ultra-short-acting adrenergic receptor antagonist. It is characterized by a fast onset of action and rapid reduction in heart rate without significantly lowering blood pressure.

It is approved for the short-term reduction of ventricular rate in adults with supraventricular tachycardia including atrial fibrillation and atrial flutter.

Mechanism of action-

Landiolol is a selective beta-1-adrenoreceptor antagonist that inhibits the positive chronotropic effects of the catecholamines, epinephrine and norepinephrine on the heart where beta-1-receptors are predominantly located.

A negative bathmotropic effect—decreased cellular excitability (by inhibiting the effects of catecholamines on beta adrenergic receptors, which reduces the concentration of Ca^{2+} ions in the cytoplasm);

A negative dromotropic effect (it decreases the speed of impulse conduction through the AV node by blocking IKs channels most notably);

A negative chronotropic effect (blocking the β-1 receptors leading to a decrease in Ca^{2+} influx into cardiomyocytes during action potential, which is thus responsible for the negative inotropic effect);

A negative inotropic effect—Due to its blockade of the β-1 receptors in the myocardium and in this way blocking the release of calcium, necessary for cardiomyocyte contraction;

An antiarrhythmic effect—Due to its suppression of triggered activity and the prolongation of a refractory period by blocking IKs channels. In this way, it may help prevent the re-entry of electrical impulses that can lead to certain types of arrhythmias such as AF, AFL, atrioventricular re-entry tachycardia, or atrioventricular nodal reentry tachycardia.

Current Channel	If	IKs	INa	INaK
Role	A mixed cation current is carried by both Na+ and K+. It is mainly involved in the pacemaker activity of the sinoatrial node, contributing to the diastolic depolarization and spontaneous firing of action potentials.	A delayed rectifier potassium current plays a role in repolarizing the cardiac action potential. It contributes to the plateau phase of the action potential in cardiac myocytes.	The fast inward sodium current is responsible for the rapid depolarization phase of the cardiac action potential. It plays a critical role in initiating and propagating action potentials in cardiac myocytes.	The sodium–potassium pump (Na+/K+-ATPase) plays a crucial role in maintaining the resting membrane potential of cardiac myocytes by actively transporting 3 atoms of sodium out of the cell and 2 atoms of potassium into the cell.
The influence of landiolol	Indirect—by blocking β-1 receptors, landiolol reduces the stimulatory effects of endogenous catecholamines, leading to a decrease in If activity and a subsequent decrease in heart rate.	May have minor effects on IKs, primarily through downstream signaling pathways influenced by the blockade of β-1 adrenergic receptors.	Indirect—by blocking β-1 receptors, landiolol reduces the stimulatory effects of catecholamines on INa, leading to a decrease in the rate of rise of the action potential and a reduction in myocardial excitability.	Not well-documented, its effects on intracellular calcium levels and ion handling in cardiac myocytes, mediated through β-1 receptor blockade, may indirectly influence the activity of the sodium–potassium pump.

Property	Landiolol	Esmolol	Metoprolol	Nebivolol	Bisoprolol	Atenolol
Drug class	Ultra-short acting selective β-1 blocker	Short-acting selective β-1 blocker	Selective β-1 blocker	Highly cardio selective β-1 blocker with vasodilator properties	Highly cardio selective β-1 blocker	Cardio selective β-1 blocker
Half-life	Very short (about 4 min)	Very short (about 9 min)	3-7 hr	10-12 hr	10-12 hr	6-7 hr
Pharmacokinetics	Rapid onset and offset of action	Rapid onset and offset of action	Rapidly and completely absorbed	Absorbed rapidly and extensively metabolized	Slowly and completely absorbed	Absorbed slowly but almost completely

Comparison with other beta blocker drugs

In terms of cardioselectivity, landiolol hydrochloride demonstrates a high degree (β1/β2 = 255) in comparison to esmolol (β1/β2 = 33) or propranolol (β1/β2 = 0.68).

Study Design-

Five randomized, double-blind, placebo-controlled studies were conducted to test landiolol efficacy and safety in patients with supraventricular tachycardia (including atrial fibrillation and atrial flutter). A total of 317 adults were treated with landiolol: heart rate in patients treated with landiolol decreased of 40-90% vs 0-11% of patients who received placebo. The infused dose of landiolol in these studies ranged from 9.3 to 74.6 mcg/kg/min. Adverse events were observed in 9,9% of landiolol treated patients (main adverse event was hypotension) versus 1 % in patients treated with placebo.

Adverse reactions-

The most important and common adverse reaction is hypotension, which in clinical trials occurred in 9.9% of patients receiving Rapiblyk vs. 1% in those receiving placebo.

Dr.Nishi, Senior resident,
Department of Pharmacology, AIIMS Patna

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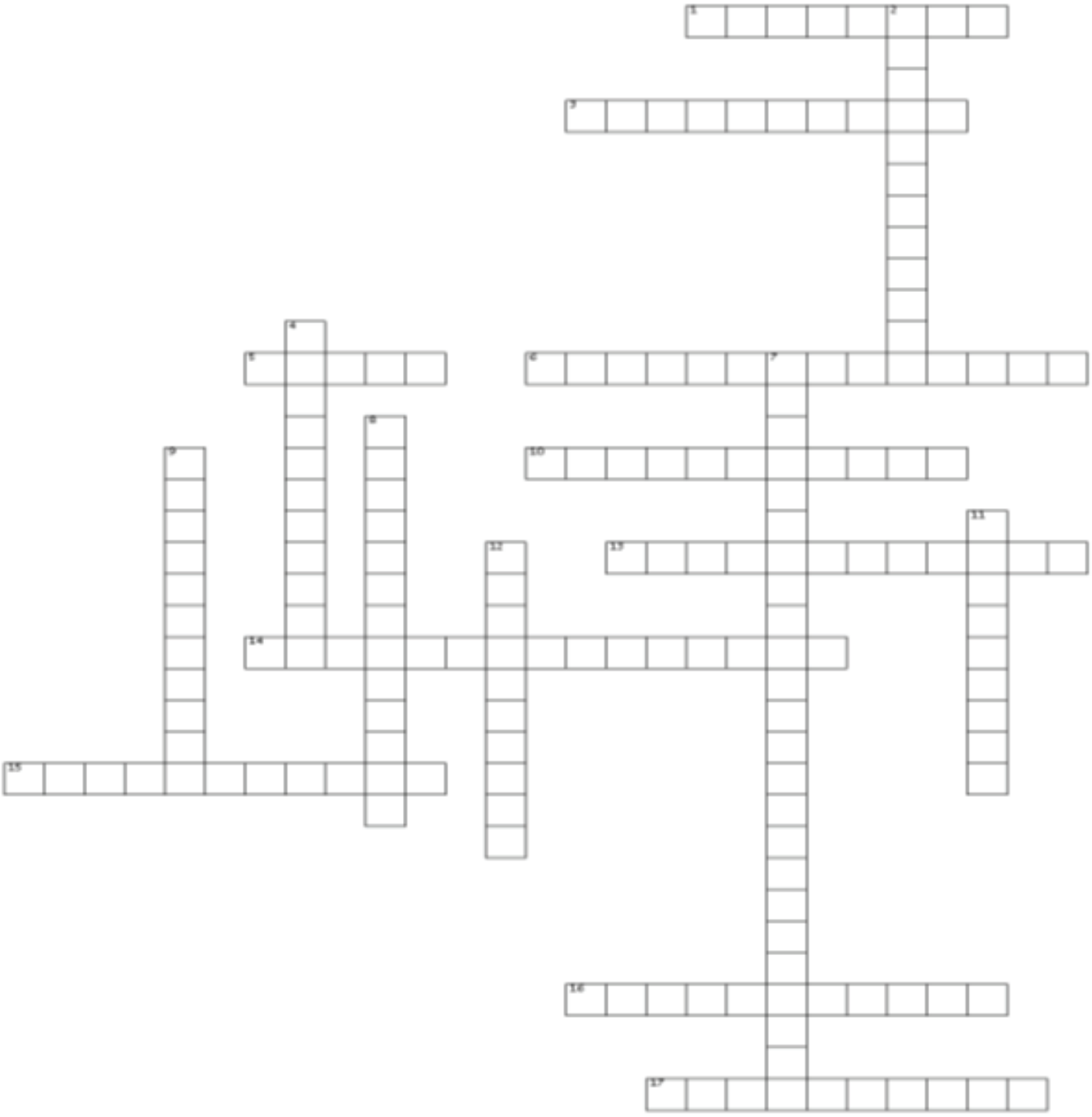
Crossword
General anaesthetics

COOL CORNER



ACROSS

1.- A tiny dose, yet strong as steel an opioid with deadly appeal

3. - Once e dream for painless sleep, later banned, its risks too deep.

5.- A noble gas, so rare, so right, like Etomidate, but out of sight. ,

6. - A gas discovered long ago, his work made nitrous oxide glow."

10. - A prodrug form, smooth to flow; no painful burn -just nice and slow.

13 - For ECI it acts so fast, a barbiturate that doesn't last.

14. - A gentle touch, both calm and slow, sedation strong without the low.

15. - Milk of anaesthesia, white and pure, for deep sedation it's the cure.

16 - He found a gas that made folks bright then laughed and wrote with pure delight.

17. - Blocks NMDA, lifts your m i nd, for pain and mood, it's well-designed.

DOWN

2. - A gas so Light, it makes you grin, d tills the pain but keeps you in.

4. - A smell so sweet no fear, no pain, the perfect start for your sleep train

7. - PRIS can strike when doses climb, a deadly risk in too much time.

8. - In history's books, he took his place, for showing ether's pa in -free grace

9. - A 'truth serum" in past display used for death and seizures today

11. - Keeps the heart both calm and steady hut adrenal shock— be ready!

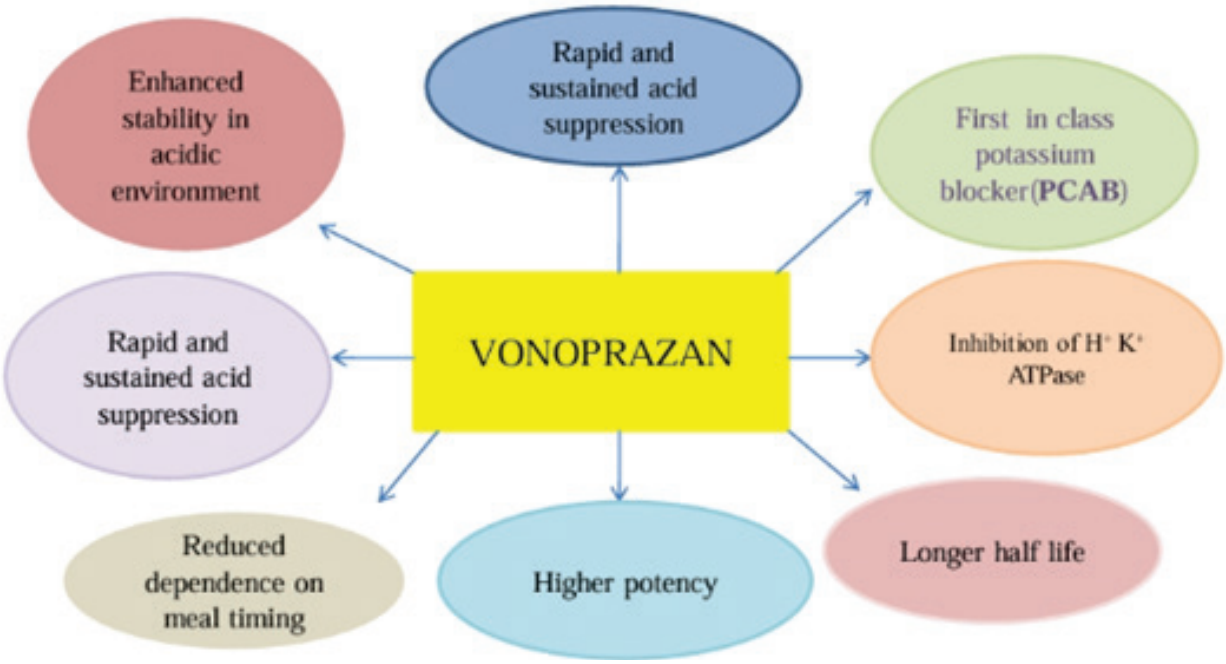
12 - Shivering stops when this is in, a post- op drug that's always been.

Precision Medicine : The Right Drug, The Right Dose, For The Right Patient!

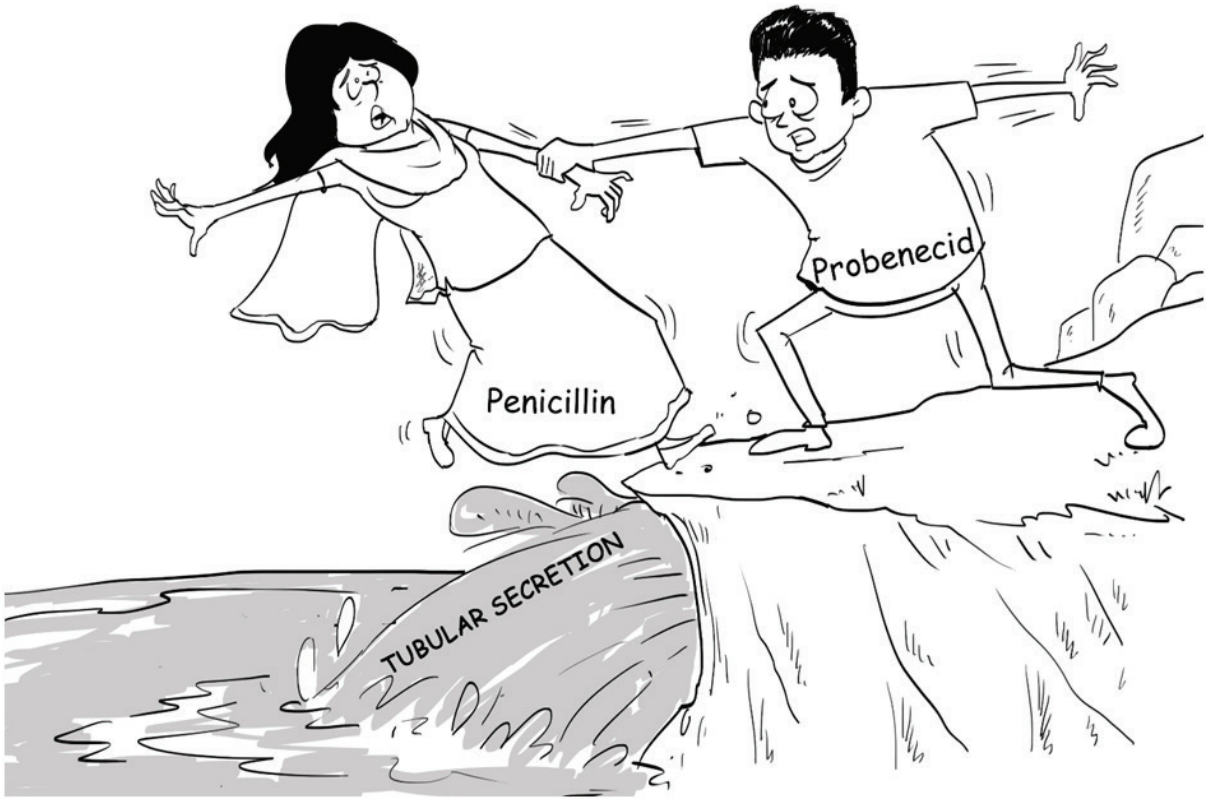
Prepared by
Dr Parvathy PR,
Assistant professor,
Believers Church Medical College Hospital, Thiruvalla, Kerala

Conquering GERD with novel anti -secretory agent VONOPRAZAN

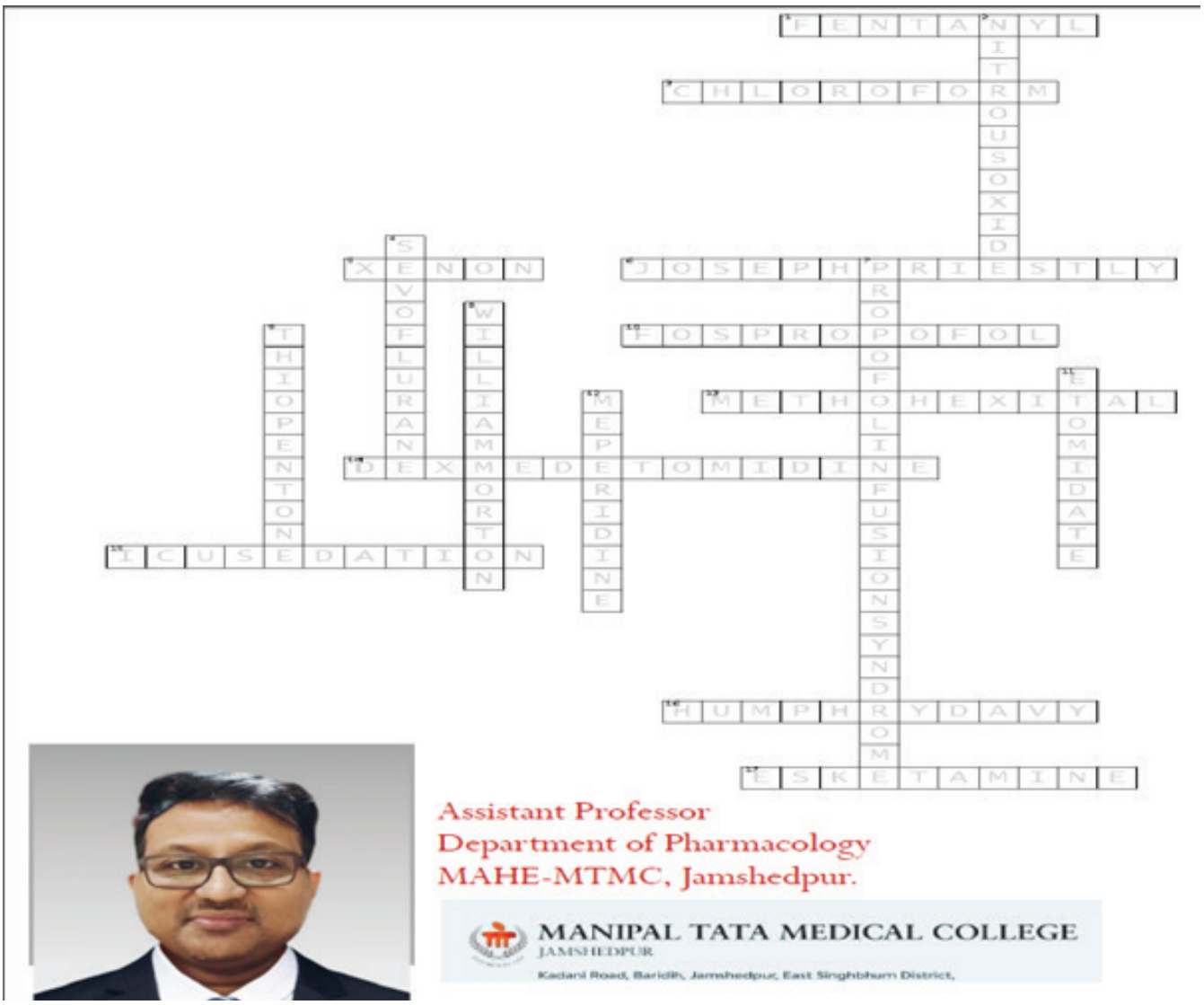
Dr. Divea Sharma
Senior Resident
Dr. Rajendera Prasad
Government Medical College, Tanda at Kangra (HP)



Cartoon Corner



Excerpt from the book "Drug Autobiographies in Pharmacology" by Dr. Sushil Sharma


FENTANYL
CHLOROFORM
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JOSEPH PRIESTLY
THIOPENTONE
SEVOFLURAN
WILLIAM HERSCHEL
METHOHEXITAL
DEXMEDETOMIDINE
ISOFLURANE
PROPOFOL
SUCCINYLCHOLINE
HUMPHRY DAVY
ESKETAMINE
Assistant Professor
Department of Pharmacology
MAHE-MTMC, Jamshedpur.
MANIPAL TATA MEDICAL COLLEGE
JAMSHEDPUR
Kadarai Road, Baridih, Jamshedpur, East Singhbhum District,

Extract

Dr Ruchi Baghel
Professor
Department of Pharmacology
Ruxmaniben Deepchand Gardi Medical College, Ujjain,
Madhya Pradesh

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.

#PGMER-23: A post-graduate student of a degree course in broad specialty/super specialty will do at least one of the following to make him/her eligible to appear in his/her final examination:

a. Poster presentation at a National/Zonal/State conference of his/her speciality;

b. Podium presentation at a National/Zonal/State conference of his/her speciality;

c. Have one research paper published/accepted for publication in journal of his/her speciality as first author.

#Soft drug effect: It refers to the concept of designing drugs that are intentionally less potent, less toxic, and more easily metabolized by the body. Advantages of Soft Drugs are Improved patient compliance, Reduced risk of adverse effects, Increased flexibility in dosing : Examples of Soft Drugs: Remifentanil, Esmolol, Landiolol.

#Opininons on centralized online teaching proposed by NMC: Most of the faculties opined that online classes can never be a replacement of physical interactive class by a good teacher. Some faculties said that students are already watching lectures on youtube and other platforms. They won't be any more interested in centralized online lectures.

#Counter receptors: Discovered on cell surfaces. These molecules interact with ligands and modify/ modulate the ligand activity and / or the actions of the related cells. They can regulate / modulate signalling pathways and immune responses. This discovery is an opening for the new drugs which may be useful for cancer chemotherapy, autoimmune conditions, and may be certain infectious diseases. Examples of drugs acting through counter receptors. PD-L1 - Atezolizumab, Avelumab & Immune modulators -Belatacept, Abatacept

#Homing receptors: Cell surface receptors that recognize and bind to specific ligands or addressins on endothelial cells, facilitating the migration and homing of cells to specific tissues or organs. Examples CD34-CD62L, CXCR4-SDF-1. Homing receptors play a crucial role in various physiological processes, including: Hematopoiesis, Immune responses & Tissue repair.

#A **slow learner** is a student who exhibits a significantly slower rate of cognitive processing and academic achievement compared to their age-matched peers. These students are not considered to have intellectual disabilities, but their learning pace is considerably slower than average.

SNACS: Students Needing Additional Curricular Support.

It refers to students who require extra academic assistance to keep pace with the medical curriculum. These students may struggle with specific subjects, learning methods, or the overall volume of information.

SNAPS: Students Needing Additional Psychological Support.

It highlights the importance of addressing the emotional and mental well-being of medical students. Medical training is demanding, and some students may experience stress, anxiety, or other psychological challenges that require professional support.

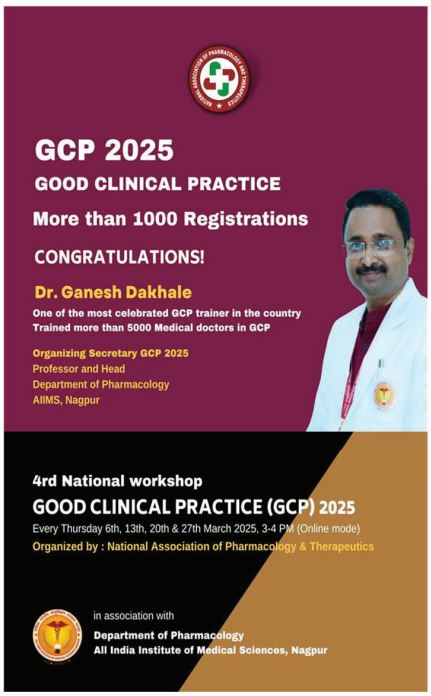
 # Polypill containing statin, ACE inhibitor, B-blocker, Thiazide& aspirin: Rationality of use of these kind of poly pills was discussed. Following are the highlights-

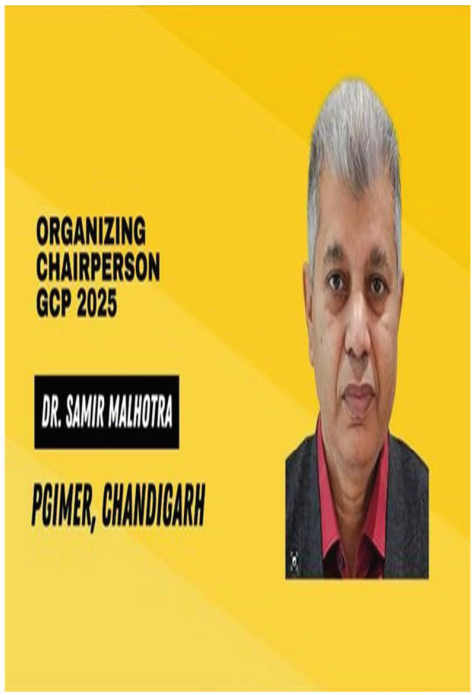
It has been approved in some developed countries, but they strictly ensure that it is used only for patients who are prescribed these drugs separately. Unlike the scenario in India where there is high chance of unregulated prescribing due to various undesirable factors influencing prescribing. If used indiscriminately it's no doubt irrational while if used appropriately after due consideration for the correct patient, it can prove beneficial. Risk-Benefit ratio should always be considered before prescribing.

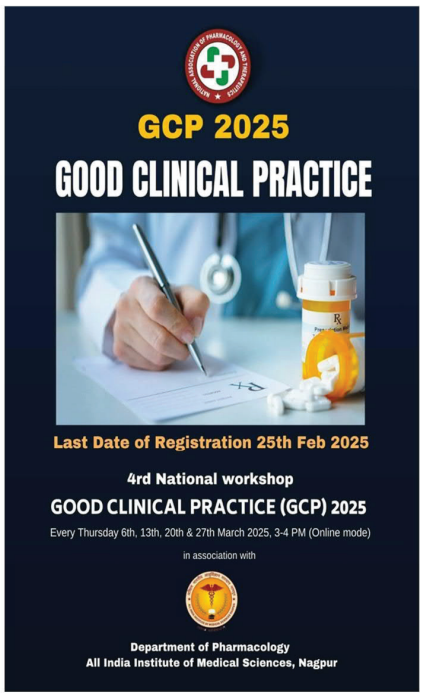
#Popular weight loss drug launched in India: Mounjaro, A anti-diabetic and obesity drug containing the active ingredient tirzepatide is now available in India in a single dose vial. Tirzepatide has been approved by USFDA for obstructive sleep apnoea.

#Digital Personal Data Protection Rules: DPDP rules bring India closer to international data protection standards making the clinical research industry more globally competitive however adopting to these rules will require a proactive approach investing in technology training and collaboration with legal and IT experts to ensure seamless compliance without compromising research efficiency.

#Deffciency of Faculty: Concerns were raised that NMC has reduced number of faculty requirement and on the other hand it is promoting small group learning through CBME. This issue needs to be addressed


GCP 2025
GOOD CLINICAL PRACTICE
More than 1000 Registrations
CONGRATULATIONS!
Dr. Ganesh Dakhale
One of the most celebrated GCP trainer in the country
Trained more than 5000 Medical doctors in GCP
Organizing Secretary GCP 2025
Professor and Head
Department of Pharmacology
AIIMS, Nagpur
4rd National workshop
GOOD CLINICAL PRACTICE (GCP) 2025
Every Thursday 6th, 13th, 20th & 27th March 2025, 3-4 PM (Online mode)
Organized by : National Association of Pharmacology & Therapeutics
in association with
Department of Pharmacology
All India Institute of Medical Sciences, Nagpur


ORGANIZING
CHAIRPERSON
GCP 2025
DR. SAMIR MALHOTRA
PGIMER, CHANDIGARH


GCP 2025
GOOD CLINICAL PRACTICE
Last Date of Registration 25th Feb 2025
4rd National workshop
GOOD CLINICAL PRACTICE (GCP) 2025
Every Thursday 6th, 13th, 20th & 27th March 2025, 3-4 PM (Online mode)
in association with
Department of Pharmacology
All India Institute of Medical Sciences, Nagpur


Thursday, 6th March, 2025
RESOURCE PERSONS
Topic
Overview of Indian GCP and ICH GCP
Time
3:00 PM-3:30 PM
Speaker
Dr. Usharani Pingali
Professor and Head
Department of Clinical Pharmacology
Nizam's Institute of Medical Sciences, Hyderabad
Moderator
Dr. Nusrat Shafeeq
Professor
Department of Pharmacology
Postgraduate Institute of Medical Education and Research (PGIMER), Chandigarh
Topic
Responsibilities of Investigators, Sponsors and monitors
Time
3:30 PM-4:00 PM
Speaker
Dr. Debashish Hota
Professor and Head
Department of Pharmacology
All India Institute of Medical Sciences Bhubaneswar
Moderator
Dr. Vipul Chaudhry
Professor and Head (Pharmacology)
GCS Medical College Hospital & Research Centre Ahmedabad


Thursday, 20th March, 2025
RESOURCE PERSONS
Topic
Ethics Committee-Composition & Individual responsibilities of members
Time
3:00 PM-3:30 PM
Speaker
Dr. Mohd. Ziauddin
Sr.,Clinical Pharmacologist
Apollo Hospitals, Hyderabad
Moderator
Dr. Alok Dixit
Professor and Head
Department of Pharmacology
UPUMS, Saifai, Uttar Pradesh
Topic
National Ethical guideline for biomedical & health research involving human participants-ICMR 2017
Time
3:30 PM-4:00 PM
Speaker
Dr. Amrita Sil
Professor
Department of Pharmacology, Medical College and Hospital Kolkata
Moderator
Dr. Rani Indira Sinha
Professor of Pharmacology
Netaji Subhas Medical College, Bihta Bihar


Thursday, 13th March, 2025
RESOURCE PERSONS
Topic
Clinical trial protocol, IB and Essential documents
Time
3:00 PM-3:30 PM
Speaker
Dr. Hira Bhalla
Professor
Department of Pharmacology
All India Institute of Medical Sciences Gorakhpur
Moderator
Dr. Sanjay Gaur
Professor and Head (Pharmacology)
Govt. Doon Medical College Dehradun
Topic
Informed consent process
Time
3:30 PM-4:00 PM
Speaker
Major Dr. Jeetendra Singh
Professor and Head (Pharmacology)
Govt. Medical College and Maharashtra Post Graduate Institute of Medical education and research
Maharashtra University of Health Sciences, Nashik.
Moderator
Dr. Naresh Jyoti
Professor and Head
Department of Pharmacology, Adesh Medical College, Shahabad, Haryana


Thursday, 27th March, 2025
RESOURCE PERSONS
Topic
SAE and Compensation Guidelines in India as per NDCT 2019
Time
3:00 PM-3:30 PM
Speaker
Dr. Ashok Shenoy
Professor
Department of Pharmacology
Kasturba Medical College
Mangalore, Karnataka
Moderator
Dr. Jayanthi M,
Professor of Pharmacology
JIPMER, Puducherry
Topic
Salient Features of NDCT 2019
Time
3:30 PM-4:00 PM
Speaker
Dr. Renuka Munshi
Professor and Head
Department of Clinical Pharmacology
TNMC and BYL Nair Hospital
Mumbai
Moderator
Dr. Yogendra Keche
Professor
Department of Pharmacology
All India Institute of Medical Sciences
Raipur, Chattisgarh

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