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Promoting Pharmacology & Therapeutics for a better tomorrow

HIGHLIGHTS

Editorial

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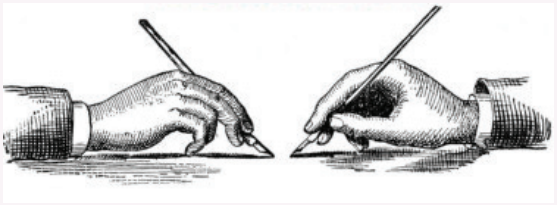

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Warm greetings to all.
Welcome to the Issue 14 of 'RxFactor', from the NATIONAL ASSOCIATION OF PHARMACOLOGY AND THERAPEUTICS (NPT). This issue continues to showcase the dynamic scope of Pharmacology and Therapeutics, encompassing developments in Medical Education, Pharmacovigilance, Research, and Clinical Practice.

The response to prior editions has been overwhelmingly positive, and we remain grateful for the appreciation and support received from our readers. Your encouragement drives our commitment to delivering content that informs, inspires, and advances the field.

This issue brings together a dynamic mix of ideas shaping the future of pharmacology and therapeutics. We begin with an article on designer microbiota—engineered microbial allies that are redefining how we think about drug delivery and disease modulation. Our feature on ferroptosis, its promise in tackling treatment-resistant cancers. We explore the expanding potential of CAR-T technology beyond oncology, including its emerging role in cardiac repair. Our article on AI-driven therapeutics reflects how intelligent systems are accelerating discovery and personalizing treatment like never before.

In the realm of global health, we address new frontiers in AMR, reviewing the evolving microbial landscape and the innovative strategies rising to meet it. We also highlight recent advances in dermatology with icotrokinra for psoriatic care and discuss the growing momentum of second-generation senolytics in age-related diseases.

From the pharmacovigilance desk, we underscore the importance of direct patient reporting and present a striking case of sitagliptin-associated bullous pemphigoid. This issue introduces, the Pharmaguide, a step toward more accessible and reliable drug information.

This issue winds up with an overview of contemporary clinical trial designs, along with our regular crossword and extract sections in the cool corner.

We extend our heartfelt thanks to all authors and contributors whose scholarly work forms the foundation of this issue. A special mention to our Postgraduate students, whose diligence and insights reflect their emerging leadership in the discipline.

We remain committed to advancing mutual learning and professional excellence through RxFactor. We hope you find this issue both informative and thought-provoking.

With best regards,
Editorial Team National Association of Pharmacology and Therapeutics (NPT)

Designer Microbiota: Engineered Allies in the Pharmacology Arsenal

Introduction

The human microbiota constitutes of a vast ecosystem of microorganisms that interacts with various immune mechanisms and biochemical functions in the host. Dysbiosis has long been implicated in the pathophysiology of numerous conditions such as inflammatory bowel disease (IBD), metabolic syndrome, autoimmune diseases, cancer, etc. The modulation of the human microbiota for therapeutic purposes is increasingly moving from an empirical approach to that of one in precision engineering. Traditional approaches of dealing with microbiota including the use of probiotics and faecal microbiota transplantation (FMT), have exhibited efficacy on one side but it often comes with issues of reproducibility, mechanistic clarity and limited regulatory guidelines.

Advances in synthetic biology, genome editing and microbial metabolic engineering now permit the designing of microbiota with pre-defined therapeutic functions: a concept that is coined as designer microbiota. Designer microbiota is defined as microbial strains or consortia deliberately selected and/or genetically modified to perform defined therapeutic functions. They offer unprecedented opportunities in pharmacology, spanning metabolic disorders, inflammatory diseases, oncology, and drug-metabolism modulation.

This article in brief, presents the current state of the field including the designing of these microbiota, key pharmacological applications, highlights translational challenges, and discusses a roadmap for approval and integration into clinical practice.

Concepts in designing therapeutically useful microbiota

Designer microbiota can be simple engineered probiotic strains or complex synthetic microbial consortia, also known as Synthetic Microbial Community. The essential tools that help us in designing a therapeutically useful Synthetic Microbial Community include:

i) selection of safe microbial chassis (e.g., non-pathogenic commensals);
ii) genome editing using CRISPR/Cas technology to insert, delete or modulate its functional components;
iii) metabolic engineering to enable production or degradation of key metabolites;
iv) biosensing circuits to enable an environment-responsive activity;
v) defined consortia establishment and ecological modelling to ensure persistence and function in vivo.

Potential Applications in Clinical Pharmacology:

1. Metabolic disease: Engineered strains have been developed to degrade excess amino acids (e.g., phenylalanine) or consume metabolites (e.g., trimethylamine) linked to disease. A landmark study described engineered Escherichia coli Nissle 1917 to degrade phenylalanine in a phenylketonuria model.
2. Inflammation and immune modulation: Microbial strains engineered to secrete cytokines like IL-10 or nanobodies against TNF have been tested in colitis animal models. The capacity of designer microbiota to deliver therapeutics at mucosal surfaces offers novel drug-delivery systems.
3. Gastrointestinal diseases: Synthetic microbial community design frameworks are being increasingly applied to gut microbiota research.
4. Oncology and tumour targeting: Certain engineered microbes can localize to hypoxic tumour niches and deliver pro-drug activating enzymes or immunomodulators, constituting a living drug delivery vehicle.
5. Drug metabolism: The gut microbiota modulates drug absorption and metabolism. They could potentially be used to control systemic exposure of drugs via modulation of microbial metabolic pathways.
6. Diagnostics and biosensing: Engineerable microbes equipped with biosensors can be used to detect pathologic signals in the body and deliver responses accordingly.
7. Bioremediation: by promoting specific strains of these microbiota with certain metabolic pathways, they can serve as a potential solution for degrading persistent pharmaceutical compounds as a part of

Ecopharmacovigilance initiative.

Approaches in Translation Pharmacology:

The following data can be considered in order to benchmark a designer microbiota as a therapeutically useful one-

1. Preclinical pharmacodynamic and pharmacokinetic studies of engineered strains of microbiota (colonisation, persistence, functional output, etc);
2. Standardised endpoints in early-phase clinical trials (e.g: biomarker modulation, metabolite kinetics, microbial load, etc);
3. Pharmacovigilance & Ecopharmacovigilance frameworks for living therapeutics, including microbial 'kill-switch' systems or biocontainment methods;
4. Integration with precision medicine to match engineered microbiota to patient microbiome profiles, host genomics and disease phenotype;
5. Regulatory harmonisation to define whether engineered microbiota are biologics, drugs or devices, fostering Good Manufacturing Practices;
6. Transparency of negative data reports in public domain.

From a clinical vantage point, a combination of therapies may also be suggested like combining designer microbiota plus small molecule drugs or biologics, for synergistic or adjunctive modulation of host–microbe–drug axis.

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Considerations & Challenges from a Clinical Pharmacologist perspective:

The following considerations and challenges for including these engineerable microbes into our current therapeutic armamentarium are:

1. Pharmacokinetics of microbial effectors: timing of activity, persistence, clearance; off-target and host-microbe interactions; microbial metabolism of drugs (which may alter efficacy or toxicity);
2. Dosing and colonisation kinetics: what 'dose' of designer microbiota corresponds to functional mass in vivo;
3. Monitoring &delivering the required therapy: the same engineered microbe can assess the condition of the patient and respond accordingly;
4. Adaptability: Sensor circuits can be adapted for other biomarkers (metabolic, cancer-related, infection markers, etc).
5. Precision medicine: Therapy is delivered when and where needed but it is triggered by a disease marker rather than continuously;
6. Safety concerns of the engineered probiotics or genetically modified organisms (GMOs) include potential acquisition of virulence traits, immune dysregulation or metabolic imbalance, containment of GMO in human host, etc.
7. Ecological stability of engineered strains and risk of horizontal gene transfer into the environment thereby preventing their harmful impact of ecosystems.
8. Regulatory frameworks still remain nascent and fragmented globally.

Conclusion

Designer microbiota represents a paradigm shift in therapeutics: moving from passive modulation toward active and programmed microbial therapies. While many challenges can be foreseen on safety, regulatory, and ecological aspects in pharmacology, the convergence of synthetic biology, microbiome science and pharmacology opens the door to living

therapeutics with precise, host-tailored functions. For the clinical pharmacologist and bioethicist alike, this domain demands rigorous mechanistic studies, risk-benefit analysis and ethical frameworks for deployment. Ultimately, the pharmacology community is poised to embrace microbiota not just as passive bystanders, but as engineered allies in drug therapy.

References

1. Aggarwal N, et al. Microbiome and human health: current understanding and future perspectives. Chem Rev. 2022;122(12):9774–81.
2. Ainsworth C. Therapeutic microbes to tackle disease. Nature, 2020,577: S20-S22.
3. Charbonneau MR, et al. Developing a new class of engineered live bacterial therapeutics to treat human diseases. Nat Commun. 2020;11:1738.
4. Duhan P, et al. Gut microbiome engineering with probiotics: current trends and future prospects. SN Appl Sci. 2025;7
5. Sadhu S, Paul T, Yadav N. Therapeutic engineering of the gut microbiome using synthetic biology and metabolic tools: a comprehensive review with E. coli Nissle 1917 as a model case study. Arch Microbiol. 2025;207(9):213

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Ferroptosis: A Pharmacological Breakthrough in Targeting Resistant Cancers

Resistance to conventional cancer therapies remains a critical challenge in oncology. Many tumours evade apoptosis by modifying signalling pathways, overexpressing anti-apoptotic proteins, altering metabolic dependencies, or undergoing epithelial–mesenchymal transition (EMT). These changes permit malignant cells to survive standard treatments such as chemotherapy, radiotherapy, targeted therapy, or immunotherapy. In this context, ferroptosis, a regulated cell death modality characterised by iron-catalysed lipid peroxidation, emerges as a promising alternative therapeutic avenue.

Ferroptosis was first defined as a distinct form of regulated cell death by Scott J. Dixon and colleagues in 2012. Unlike classical apoptotic, necroptotic, pyroptotic, or autophagic death, ferroptosis exhibits unique morphological, biochemical and genetic features. Ferroptosis operates independently of apoptosis. Importantly, experimental and preclinical evidence suggest that ferroptosis has potent tumour-suppressive functions, especially in cancers that have developed resistance mechanisms against conventional therapies. Given its reliance on specific metabolic vulnerabilities such as cystine dependency, oxidative stress, and presence of polyunsaturated fatty-acid–rich (PUFA-rich) membranes, ferroptosis represents a novel pharmacological target with high potential to address refractory malignancies.

Mechanism of Ferroptosis

Ferroptosis occurs primarily via uncontrolled lipid peroxidation in the presence of iron, involving at least three central processes: iron overload, peroxidation of membrane lipids, and failure of antioxidant defences.

A. Iron Overload (Increased Labile Iron Pool): Cancer cells often exhibit a high demand for iron, sometimes described as 'iron addiction.' This increased intracellular iron (particularly free ferrous iron, Fe^{2+}) can catalyse the Fenton reaction, producing free radicals, which react with polyunsaturated fatty acids (PUFAs) in membrane phospholipids and initiate lipid peroxidation, generating lipid hydroperoxides. When generated in excess, these lipid peroxides compromise membrane integrity, ultimately leading to cell death by ferroptosis.

B. Lipid Peroxidation: Ferroptosis depends on the oxidation of PUFAs incorporated into membrane phospholipids. Enzymes such as long-chain acyl-CoA synthetase 4 (ACSL4), lysophosphatidylcholine acyltransferase 3 (LPCAT3), and lipoxygenases (ALOX family) promote the biosynthesis and oxygenation of PUFA-containing phospholipids, generating peroxidized lipid species. Once the level of lipid peroxides exceeds a cytotoxic threshold, membrane damage ensues, culminating in ferroptotic cell death.

C. Antioxidant System Failure — GPX4 & GSH Depletion: Under normal conditions, cells rely on antioxidant defences to detoxify lipid peroxides. A critical player is Glutathione peroxidase 4 (GPX4), which uses the tripeptide glutathione (GSH) to reduce lipid hydroperoxides to non-toxic lipid alcohols.

Pharmacological Induction of Ferroptosis

Ferroptosis can be pharmacologically induced by disabling this antioxidant system. For instance:

- The small molecule Erastin inhibits the system cystine/glutamate antiporter, reducing cystine uptake → GSH levels fall → GPX4 becomes inactive.
- The compound RSL3 (a small molecule) directly inactivates GPX4 → lipid peroxides accumulate rapidly → ferroptosis ensues.
- Thus, the convergence of increased iron, enhanced lipid peroxidation and compromised antioxidant r

pair establishes ferroptosis:

Iron ↑ + Lipid peroxidation ↑ + GPX4/GSH ↓ → Ferroptosis

Beyond Erastin and RSL3, several other experimental strategies and molecules have been developed to induce ferroptosis, including FIN56, ferroptosis sensitisers, etc

Cancers Most Likely to Respond to Ferroptosis

Based on accumulating data, certain tumour types

appear particularly vulnerable to ferroptosis due to their metabolic characteristics (iron-rich, PUFA-rich membranes, low antioxidant capacity). These include:

- **Pancreatic ductal adenocarcinoma (PDAC):** among the most therapy-resistant cancers; high iron demand and low antioxidant defenses make them strong ferroptosis candidates.
- **Triple-negative breast cancer (TNBC):** depends heavily on GPX4 for survival — making it a high-yield ferroptosis target.
- **Non–small cell lung cancer (NSCLC):** especially KRAS-mutant or EGFR-resistant subtypes; preclinical experiments suggest sensitivity to erastin/RSL3–mediated ferroptosis.
- **Prostate cancer:** therapy-resistant prostate tumors often accumulate PUFA lipids — rendering them vulnerable to lipid peroxidation–driven death.
- **Hepatocellular carcinoma (HCC):** some standard drugs (e.g. Sorafenib) may exert part of their anti-tumor effect via ferroptosis; thus combining ferroptosis inducers might improve response in resistant HCC.
- **Melanoma, glioblastoma, leukemia, and others:** emerging data suggest that dedifferentiated, mesenchymal, or stem-cell–like tumors with altered lipid/iron metabolism may be especially susceptible.

Conclusion

Ferroptosis has emerged as a promising therapeutic avenue, especially for cancers resistant to apoptosis and standard treatments. Its unique mechanism : iron-dependent lipid peroxidation coupled with failure of antioxidant defences, exploits metabolic vulnerabilities that are often pronounced in therapy-resistant, dedifferentiated, mesenchymal, or RAS-mutant tumours. Preclinical evidence across a variety of cancer types supports the potential of ferroptosis induction, alone or in combination with existing therapies. Although obstacles remain, notably regarding toxicity, immune interactions, and biomarker identification, ongoing advances in small-molecule inducers, delivery systems, and mechanistic understanding suggest a high likelihood that ferroptosis-based cancer therapy will become a reality in the foreseeable future.

References:

1. Li, J., Cao, F., Yin, Hl. *et al.* Ferroptosis: past, present and future. *Cell Death Dis* **11**, 88 (2020). https://doi.org/10.1038/s41419-020-2298-2
2. Chen, F., Kang, R., Tang, D. *et al.* Ferroptosis: principles and significance in health and disease. *J Hematol Oncol* **17**, 41 (2024). https://doi.org/10.1186/s13045-024-01564-3
3. Zhou Q, Meng Y, Le J, Sun Y, Dian Y, Yao L, Xiong Y, Zeng F, Chen X, Deng G. Ferroptosis: mechanisms and therapeutic targets. MedComm (2020). 2024 Nov 20;5(12):e70010. doi: 10.1002/mco2.70010. PMID: 39568772; PMCID: PMC11577302.
4. Jin, X., Tang, J., Qiu, X. *et al.* Ferroptosis: Emerging mechanisms, biological function, and therapeutic potential in cancer and inflammation. *Cell Death Discov.* **10**, 45 (2024). https://doi.org/10.1038/s41420-024-01825-7

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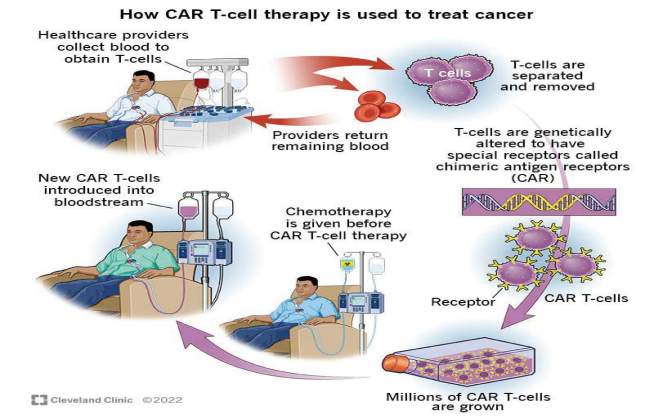
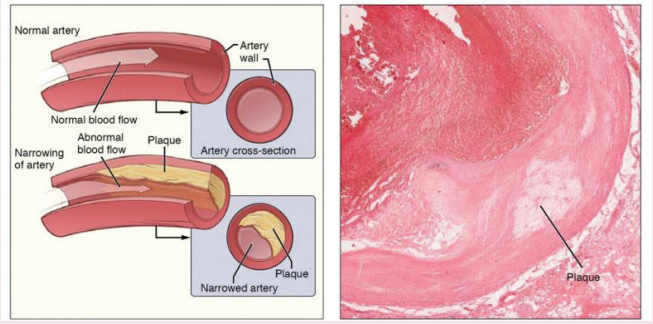
CAR-T cell Therapy in Heart Disease

What is CAR T cell therapy?

Chimeric antigen receptor (CAR) T-cell therapy is an advanced form of immunotherapy used to treat certain blood cancers. The approach involves genetically modifying a patient's own T lymphocytes so that they become more potent at recognizing and destroying cancer cells. By inserting a specially designed gene into these T cells, clinicians equip them with enhanced ability to identify and eliminate malignant cells. For some individuals, CAR T-cell therapy can lead to a complete cure, while for others it significantly extends survival and improves treatment outcomes.





Can it fail?

The success of CAR T-cell therapy varies based on the type of cancer being treated. According to a 2017 review, this approach achieved complete remission in up to 90% of patients with a particular type of leukaemia. Despite these promising outcomes, CAR T-cell therapy remains a relatively recent advancement, with the U.S. Food and Drug Administration (FDA) granting approval for the first therapy only in 2017. As a result, ongoing research is needed to fully understand its long-term effectiveness and broader clinical potential.

Innovating Anti-Inflammatory Pathways in Cardiovascular Research

A groundbreaking preclinical investigation has revealed that CAR T-cell therapy—best known as a personalized immunotherapy for cancer—may also offer powerful benefits in treating atherosclerosis. The study suggests that this engineered immune-cell approach could effectively target the arterial plaque build-up that narrows blood vessels and contributes to heart attacks and strokes.

In mouse studies, the experimental CAR T cells were shown to suppress arterial inflammation, preventing more than two-thirds of the plaque accumulation observed in untreated animals. The research, conducted by scientists at the Perelman School of Medicine at the University of Pennsylvania and published in *Circulation*, marks a significant advance in applying CAR T-cell technology beyond oncology. "Our study shows for the first time how CAR T-cell technology could be used to treat the underlying cause of the most common form of heart disease, which remains the leading cause of death worldwide," said senior author Avery Posey, Ph.D., an assistant professor of Pharmacology.

"These preclinical findings represent an important step toward expanding the therapeutic reach of CAR T cells to widespread diseases outside of cancer". Co-author Daniel J. Rader, MD, a leading expert in lipid biology and atherosclerosis, noted that using CAR T cells to target the pro-inflammatory molecule oxidized LDL (oxLDL) could offer an important complementary strategy for patients who remain at high risk even with optimal cholesterol-lowering therapy.

Researchers are expanding the use of CAR T-cell technology beyond cancer to conditions such as autoimmune disease, fibrosis, and cardiovascular disorders. In this study,

investigators engineered regulatory T cells (Tregs)—immune-suppressing cells recognized in the **2025 Nobel Prize in Physiology or Medicine**—to express a CAR targeting oxidized LDL (OxLDL), a key pro-inflammatory driver of atherosclerosis[1]. These CAR Tregs effectively reduced OxLDL-induced inflammation in human cell assays and, in mouse models predisposed to hypercholesterolemia, lowered atherosclerotic plaque burden by nearly **70%** after twelve weeks of treatment[2]. Importantly, the therapy did not disrupt general immune function. To advance this approach toward clinical translation, the researchers founded **Cartio Therapeutics**, which aims to test OxLDL-targeted CAR Tregs in human trials. This work builds on the established connection between cancer, inflammation, and cardiovascular disease, emphasizing the potential for immunotherapy to address residual cardiovascular risk in cancer survivors[3].

If ultimately proven safe and effective in clinical trials, researchers do not expect CAR T-cell therapy to replace existing treatments. Instead, it may serve as an additional option for individuals who require further intervention beyond standard heart disease therapies.

REFERENCES

1. Steinberg, D., & Witztum, J. L. (2010). Oxidized LDL and atherosclerosis. *Arteriosclerosis, Thrombosis, and Vascular Biology*, 30(12), 2311–2316.
2. Schwab, R. et al. (2024). CAR Treg therapy targeting oxidized LDL reduces atherosclerotic inflammation in preclinical models. *Circulation*.
3. Mehta, L. S. et al. (2018). Cardiovascular disease and cancer: Understanding the shared biology. *Circulation*, 138(7), 734–742.

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"When Medicines Think: The New Era" Advances at the Intersection of Intelligence and Therapeutics

Artificial Intelligence (AI) has quickly shifted from being a simple computer-based aid to becoming a major driving force in drug discovery and development. With the growth of detailed biological, chemical, and clinical datasets, AI now helps researchers study complex biological systems, design new drug molecules, and speed up many steps in the drug development process with greater accuracy and efficiency. By using machine learning, deep learning, natural language processing (NLP), and advanced generative models, AI is reshaping modern pharmaceutical research. It plays an important role in identifying drug targets, improving molecules, predicting safety, and supporting various stages of clinical research.

Success stories that Redefined Modern Medicine:

- HIV/AIDS: once a fatal disease, it is now a manageable chronic condition.
- Imatinib (Gleevec): This targeted drug transformed chronic myeloid leukaemias from a deadly cancer into a largely controllable disease.
- Hepatitis C: Development of curative treatments.
- CAR-T cell Therapy: Engineered immune cells now offer life-saving treatment for certain otherwise refractory blood cancers.

1. Transforming Target Identification and Biology Understanding:

Traditionally, finding the right biological target for a drug takes many years of experiments involving genetics, biochemistry, and disease studies. AI speeds up this process by examining large amounts of data- from omics information to biological pathway networks – to quickly identify targets linked to diseases. Deep learning models can spot patterns in gene expression, protein interactions, and disease characteristics that humans may not easily notice. Tools such as knowledge graphs and network–based AI systems are now commonly used to better understand how diseases work, discover new biomarkers, and select the most promising drug targets. This greatly reduces early uncertainty and supports more efficient and logical drug design.

AI's strengths in Target Discovery:

- Faster Discovery of Novel Targets
- AI Detects Hidden Biological Patterns
- Knowledge Graphs connect Complex Biological Data
- AI improves Target Prioritisation & Validation
- AI accelerates Biomarker Discovery
- Rapid Identification During Infectious Disease Outbreaks

2. Revolutionizing Molecular Design and Lead Discovery:

Generative AI is reshaping early drug discovery by rapidly designing and evaluating novel molecules. Tools like Variational autoencoders (VAEs), Generative adversarial networks (GANs), diffusion models, and Graph neural networks (GNNs) allow researchers to create drug-ready structures and screen them in hours rather than months. These advances are helping companies identify strong hit candidates faster, more accurately, and at a fraction of traditional discovery costs.

Case Study: AI Uncovers a Hidden Target for Fibrosis:

- Insilico Medicine used a GAN-based system and its Panda Omics platform to identify a new Idiopathic Pulmonary Fibrosis target (initially *Target 3084*).
- The AI-designed molecule INS018_055 progressed rapidly into clinical trials—one of the world's first AI-discovered and AI-generated drug candidates.

3. Advancements in Preclinical Prediction: ADMET and Toxicology:

Before a drug reaches human trials, it must show safe ADMET properties. This usually requires extensive lab and animal testing.

AI now predicts ADMET features early by analysing molecular structures to detect toxicity risks, metabolic issues, and drug–drug interactions. This reduces animal testing, cuts costs, and improves safety decisions.

Case study: AI predicts Toxicity for a Cardiovascular Drug Candidate

- A Novartis team used AI toxicology tools to screen early candidates.

- AI flagged a molecule with high liver toxicity – later confirmed in lab tests. This prevented costly downstream failures and redirected efforts towards safer alternatives.

4. Enhancing Clinical Development Through AI :

AI is becoming essential in improving clinical trials by tackling key challenges such as poor patient selection and unexpected side effects. By analysing real-world data, electronic health records, and genomic information, AI helps identify the most suitable patient groups for a study.

Tools like digital twins enable researchers to simulate patient responses, optimise dosing, and identify potential safety issues before trials begin. AI also enables adaptive trial designs and real-time data analysis, helping reduce trial duration and increasing the likelihood of success.

Case study highlights:

- Bristol Myers Squibb (BMS): AI-powered matching improved oncology patient recruitment and trial efficiency.
- Lilly and Evidation Health: Digital twins guided clinical planning for diabetes therapy, improving dose selection and trial design.

5. **Ethical, Regulatory, and Practical Challenges:**

Despite its benefits, AI presents challenges:

- Requires large, high-quality datasets
- Risk of bias and inaccurate predictions
- Need for transparent, explainable AI decision-making

Regulators like the FDA and EMA are creating guidelines focusing on validation, reproducibility, explainability, and continuous monitoring.

Ethical issues such as data privacy, fairness, and equitable access must be addressed to ensure responsible use.

6. The Future of AI-Enabled Drug Discovery:

Drug discovery is moving towards fully automated, AI-driven ecosystems. Future workflows will integrate:

- Autonomous robotic labs
- AI-guided synthesis
- Continuous learning models
- Real-time experimental feedback

These advances will shorten development timelines, lower costs, and enable breakthroughs against diseases once considered "undruggable"

AI will not just accelerate drug discovery – it will redefine it.

References:

1. Hughes JP, Rees S, Kalindjian SB, Philpott KL. Principles of early drug discovery. *Br J Pharmacol.* 2011;162(6):1239–49. doi:10.1111/j.1476-5381.2010.01127.x.
2. Nature Biotechnology. Insilico Medicine's AI-identified fibrosis target. *Nat Biotechnol.* 2023.
3. Jumper J, Hassabis D, et al. AlphaFold and RoseTTAFold enable rapid insights into protein structures and networks. *Nature.* 2021–2022.
4. AstraZeneca R&D. Using AI knowledge graphs to enhance target selection and reduce project risk. *AstraZeneca R&D Blog.* 2022.
5. Brown A, et al. BenevolentAI identifies rheumatoid arthritis targets using graph-based AI. *Lancet.* 2019.
6. Tempus; Flatiron Health. AI-based biomarker discovery for oncology decision-making. *JAMA Oncol.* 2021.
7. Gordon DE, et al. AI platforms flag SARS-CoV-2 protein targets using structural and network models. *Cell.* 2020.
8. Schyman P, Liu R, Desai V, Wallqvist A. *In silico* toxicology: machine learning models for drug toxicity prediction at Novartis. *J Chem Inf Model.* 2020;60(3):1122–32.
9. Bristol Myers Squibb. AI-driven patient matching in oncology trials. *BMS Innovation Report.* 2022.
10. Bruining H, et al. Digital twins for diabetes management and clinical trial simulation. *Nat Med.* 2022.
11. U.S. Food and Drug Administration. Artificial Intelligence & Machine Learning for Drug Safety Surveillance: Pilot Evaluation Report. FDA; 2021.

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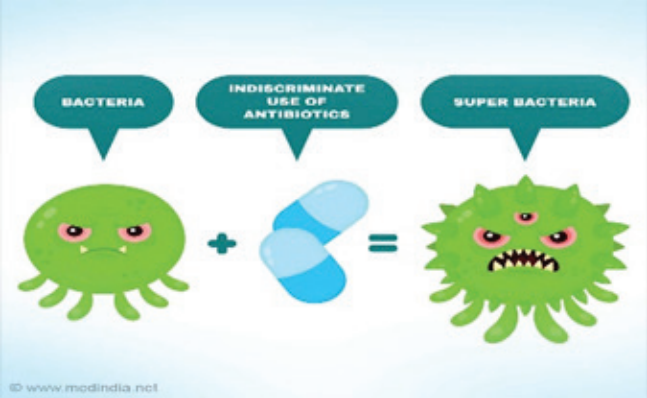
New Frontiers in Antimicrobial Resistance (AMR): Emerging Threats and Innovative Solutions



Health crises such as viral pandemics arise suddenly and require immediate actions, others emerge more slowly and are more unnoticeable and intractable. An example of the latter is antimicrobial resistance (AMR), which has been declared as one of the top ten global public health threats facing humanity by WHO in 2019 [1] . This threat is aggravated by the drying pipeline of new antimicrobials. In the dearth of newer antibiotics, the best possible approach is to efficiently handle the existing antimicrobials. This narrative review highlights the key emerging frontiers in AMR relevant for clinicians in 2025.

One of the most significant shifts in AMR involves the rise of **highly resistant Gram-negative organisms**, particularly carbapenem-resistant *Enterobacterales* (CRE), *Pseudomonas aeruginosa*, and *Acinetobacter baumannii*. Carbapenem-resistant Enterobacterales (CRE), especially strains producing New Delhi metallo-β-lactamase (NDM),KPC and OXA-48 like

remain a major challenge in India and worldwide[2]. The epidemiology of AMR is changing due to plasmid-mediated gene transfer and increased global travel. This increasing complexity underscores the need for rapid diagnostics and targeted therapeutics.

NEXT GENERATION APPROACHES TO TACKLE ANTIMICROBIAL RESISTANCE

RAPID DIAGNOSTICS:

Rapid AMR Diagnostic Tests include MALDITOF MS (Matrix-Assisted Laser Desorption/Ionization – Time-of-Flight Mass Spectrometry), qRT-PCR assays (Quantitative Real-Time PCR), nucleic-acid amplification tests (NAATs), Multiplex / molecular PCR-based panels. These tests can identify species and resistance genes in a matter of minutes compared to conventional methods. AI-enabled tools are increasingly used to predict hospital outbreaks, optimize antibiograms, and guide empirical therapy[3]. When integrated with AMS programs, rapid diagnostics significantly reduce inappropriate antibiotic use and improve clinical outcomes.

TARGETED THERAPEUTICS:

Advanced BL/BLIs (Betalactam/Betalactamase Inhibitors) represent one of the most significant therapeutic developments. Molecules such as **ceftazidime–avibactam**, **meropenem–vaborbactam**, and **imipenem–cilastatin–relebactam** show strong activity against KPC and OXA-48 producers. **Cefiderocol**, a siderophore cephalosporin that hijacks bacterial iron-uptake pathways, provides potent activity against carbapenem-resistant *Pseudomonas* and *Acinetobacter*[4].

Bacteriophage therapy has re-emerged as a promising adjunct for MDR infections. Phages are highly specific, capable of disrupting biofilms, and often synergistic with antibiotics. Phage cocktails were used to treat a **life-threatening MDR** A. baumannii **infection** in patients with a bloodstream infection[5].

CRISPR-Cas systems (antimicrobials) use programmable gene-editing tools by precise elimination of resistance genes or selective killing of resistant bacteria. An Ongoing research project (started 2023) at ICMR-National Institute of Pathology (New Delhi) is exploring the role of CRISPRCas systems in the pathogen Acinetobacter baumannii[6].

Microbiome Modifying Interventions : Antibiotic-induced dysbiosis fuels selection and persistence of resistant organisms. Interventions such as fecal microbiota transplantation (FMT), next-generation probiotics, and microbiome-sparing antibiotics aim to restore microbial balance[7].

Drug Repurposing : The re-investigation of the previously known drug as a new treatment option for another disease is known as Drug Repurposing. Initiatives like Medicines ofMalariaVenture (MMV), Drugs forNeglected Diseases Initiative (DNDi) provide researchers across the globe with drug screening libraries to screen at a far greater pace. Drug repurposing and multidrug therapy have good potential to combat against AMR infections[8].

Nanoparticle based antimicrobials : Nanobiotics are nanoscale formulations of antimicrobial agents designed to improve the delivery, stability, and efficacy of antibiotics, particularly against **multidrug-resistant (MDR) pathogens**. They penetrate biofilms more effectively, target pathogens selectively and overcome bacterial resistance mechanisms such as efflux pumps or enzymatic degradation. Include Gold Nanoparticles (AuNPs), Silver Nanoparticles (AgNPs), Liposome-Encapsulated Antibiotics *(Liposome-vancomycin* or liposome-daptomycin*)* [9].

Antimicrobial Stewardship (AMS) Activities : They integrate **rapid diagnostics, antibiograms, and clinical guidelines** to guide clinicians in making informed treatment decisions. By limiting overuse and misuse of antibiotics, AMS helps **slow the emergence of multidrug-resistant organisms**[10].

Conclusion

AMR demands a multifaceted response that integrates innovative therapies, precision diagnostics, microbiome-centered approaches, and strong One Health collaboration. While newer agents such as cefiderocol, BL/BLIs, phage therapy, and CRISPR-based antimicrobials offer encouraging possibilities, judicious antibiotic use and antimicrobial stewardship practices remains the cornerstone of AMR containment. Continuous investment, policy support, and clinician engagement will be essential to safeguard the future of infectious disease management.

References

1. Antimicrobial resistance: a top ten global public health threat,eClinicalMedicine, Volume 41,2021,101221,ISSN 2589-5370,
2. Logan, L. K., & Weinstein, R. A. (2017). The Epidemiology of Carbapenem-Resistant Enterobacteriaceae: The Impact and Evolution of a Global Menace. *The Journal of Infectious Diseases*, *215*(suppl_1), S28–S36
3. O'Neill J. *Tackling Drug-Resistant Infections Globally: Final Report and Recommendations*. Review on Antimicrobial Resistance. HM Government & Wellcome Trust; 2016.
4. Wunderink RG, Matsunaga Y, Ariyasu M et al . Cefiderocol versus high-dose, extended-infusion meropenem for the treatment of Gram-negative nosocomial pneumonia (APEKS-NP): a randomised, double-blind, phase 3, non-inferiority trial. Lancet Infect Dis. 2021 Feb;21(2):213-225
5. Schooley RT, Biswas B, Gill JJ, et al. Development and Use of Personalized Bacteriophage-Based Therapeutic Cocktails To Treat a Patient with a Disseminated Resistant *Acinetobacter baumannii* Infection. *Antimicrob Agents Chemother.* 2017;61(10):e00954-17.
6. CRISPR-Cas systems in Acinetobacter baumannii: role in bacterial fitness, antibiotic resistance and implications for antibacterial therapy — A research project (2023–2026) at ICMR-National Institute of Pathology aiming to explore the role of CRISPR-Cas systems in *A. baumannii*.
7. Woodworth MH, Hayden MK, Young VB, Kwon JH. The Role of Fecal Microbiota Transplantation in Reducing Intestinal Colonization With Antibiotic-Resistant Organisms: The Current Landscape and Future Directions. Open Forum Infect Dis. 2019 Jul 1;6(7):ofz288.
8. Bhandari V, Suresh A. Next-Generation Approaches Needed to Tackle Antimicrobial Resistance for the Development of Novel Therapies Against the Deadly Pathogens. Front Pharmacol. 2022 Jun 2;13:838092
9. Huh AJ, Kwon YJ. "Nanoantibiotics": a new paradigm for treating infectious diseases using nanomaterials in the antibiotics resistant era. J Control Release. 2011 Dec 10;156(2):128-45
10. Darwish, R.M., Matar, S.G., Snaineh, A.A.A. *et al.* Impact of antimicrobial stewardship on antibiogram, consumption and incidence of multi drug resistance. *BMC Infect Dis* 22, 916 (2022)

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Translating Cytokine Blockade into a Pill: The Pharmacological Promise of Icotrokinra in Psoriasis Management.

Immune-mediated inflammatory diseases (IMIDs), including psoriasis, psoriatic arthritis, and inflammatory bowel disease (IBD), which encompasses Crohn's disease and ulcerative colitis, represent a diverse group of conditions characterized by chronic inflammation (1). Psoriasis is a chronic inflammatory condition mediated by the immune system, frequently linked to various other long-term health disorders, including psoriatic arthritis, metabolic syndrome, cardiovascular disease, (2). The pathogenic mechanism behind these is pro-inflammatory cytokines like Interleukin (IL)-12, IL-17 and IL-23 (3). Growing evidence indicates that IL-23 plays a crucial role in the pathogenesis of psoriasis (4). Biologic therapies are highly effective for treating immune-mediated inflammatory diseases; however, they are administered as injections, which can pose challenges for individuals who experience anxiety or discomfort with injectables. This issue is particularly significant for children and adolescents, as well as for anyone who prefers oral medications. While there are oral treatments available, such as apremilast and deucravacitinib, these tend to be less effective than biologics and may come with side effects. This highlights the need for new oral options that are both effective and easier to take than injections (3).

Icotrokinra (JNJ-77242113) in clinical trials:

Peptides generally are highly specific, have better tissue penetration and bioavailability compared to monoclonal antibodies (mAbs) and small molecule inhibitors (1). Icotrokinra the first-in-class targeted oral polypeptide that binds to Interleukin-23-receptor and inhibits IL-23 signaling (5). Inhibiting the interleukin-23 pathway and the subsequent production of IL-17A, IL-17F, and IL-22 has been proven to be an effective strategy for managing moderate-to-severe plaque psoriasis (3). In the FRONTIER 1 Phase 2b trial, all icotrokinra dose groups achieved ⩾75% reduction in Psoriasis Area and Severity Index (PASI 75) at 16 weeks, with 12–40% reaching complete clearance (PASI 100) versus placebo. The long-term FRONTIER 2 extension showed 76% of patients on 100 mg twice daily maintained PASI 75 at 52 weeks. These results led to the ICONIC-LEAD Phase 3 trial, 65% of those receiving icotrokinra reached an Investigator's Global Assessment (IGA) score of 0/1 (clear or almost clear skin), compared to 8% with placebo. Adverse effect rates were similar between icotrokinra and placebo groups. The common adverse effects of icotrokinra were nasopharyngitis and upper respiratory tract infection (3). Serious adverse events were observed in 1% of the study group and 3% in the placebo group (3).

Results were poisitive in Phase 3 ICONIC-ADVANCE 1 and 2 trials for moderate-to-severe plaque psoriasis. These studies demonstrated icotrokinra's superior efficacy over both placebo and the oral TYK2 inhibitor deucravacitinib with significant improvements in PASI and IGA scores. The treatment maintained a safety profile similar to placebo and offered the convenience of once-daily oral dosing.Based on these results New Drug Application (NDA) has been submitted to the U.S. Food and Drug Administration (FDA) seeking approval for icotrokinra for the treatment of adults and pediatric patients 12 years of age and older with moderate to severe plaque psoriasis (6).

Icotrokinra and other potential targets:

ANTHEM-UC Trial- A phase 2b multicenter, randomized, placebo-controlled, dose-ranging study evaluating the efficacy and safety of icotrokinra in patients with moderately to severely active Ulcerative Colitis who had an inadequate response or intolerance to conventional therapy, prior biologics, and/or ozanimod or approved JAK inhibitors. All doses met Primary endpoint with response rate of 63.5% at 12 weeks (7).

ICONIC-PsA 1- A Phase 3, multicentre, randomized, double-blind, placebo-controlled Study Evaluating the Efficacy and Safety of Icotrokinra in multiple dose-range for the Treatment of Biologic-naïve Participants with Active Psoriatic Arthritis (8).

ICONIC-PsA 2- A Phase 3, multicentre, randomized, double-blind, placebo-controlled study is to evaluate

the efficacy of icotrokinra compared to placebo in biologic-experienced participants with active psoriatic arthritis (9).

Conclusion:

Patients with moderate-to-severe psoriasis, psoriatic arthritis, and IBD are generally treated with injectable mAbs or small-molecule therapeutics. Although oral peptides are a focus of research, none are currently available for treating IMIDs. Icotrokinra stands as the first investigational orally administered peptide being assessed in patients with these conditions. It has shown encouraging potential for individuals suffering from moderate-to-severe plaque psoriasis, providing a combination of complete skin clearance and a favourable safety profile compared to existing options, all within the ease of a once-daily tablet.

References:

1. Stein Gold L, Eyerich K, Merola JF, Torres J, Coates LC, Allegretti JR. Oral Peptide Therapeutics as an Emerging Treatment Modality in Immune-Mediated Inflammatory Diseases: A Narrative Review. Adv Ther. 2025;42(7):3158–72.
2. Gold LS, Armstrong AW, Bissonnette R, Magnolo N, Vender RB, Sebastian M, et al. Once-daily oral icotrokinra versus placebo and once-daily oral deucravacitinib in participants with moderate-to-severe plaque psoriasis (ICONIC-ADVANCE 1 & 2): two phase 3, randomised, placebo-controlled and active-comparator-controlled trials. The Lancet. 2025 Sep 27;406(10510):1363–74.
3. Bissonnette R, Soung J, Hebert AA, Pink AE, Pinter A, Moore AY, et al. Oral Icotrokinra for Plaque Psoriasis in Adults and Adolescents. New England Journal of Medicine. 2025 Nov 5;393(18):1784–95.
4. Chiricozzi A, Saraceno R, Chimenti MS, Guttman-Yassky E, Krueger JG. Role of IL-23 in the pathogenesis of psoriasis: a novel potential therapeutic target? Expert Opin Ther Targets. 2014 May;18(5):513–25.
5. Nye J, PhD. Icotrokinra for Plaque Psoriasis Superior to Placebo for Skin Clearance, Safety [Internet]. Rheumatology Advisor. 2025 [cited 2025 Nov 11]. Available from: https://www.rheumatologyadvisor.com/news/icotrokinra-plaque-psoriasis-superior-to-placebo-for-skin-clearance-safety/
6. Drugs.com [Internet]. [cited 2025 Nov 11]. Icotrokinra: What is it and is it FDA approved? Available from: https://www.drugs.com/history/icotrokinra.html
7. MA AB. ANTHEM-UC: Icotrokinra (JNJ-2113) Shows Promise for Ulcerative Colitis | HCPLive [Internet]. 2025 [cited 2025 Nov 12]. Available from: https://www.hcplive.com/view/anthem-uc-icotrokinra-jnj-2113-shows-promise-ulcerative-colitis
8. Janssen Research & Development, LLC. A Phase 3, Multicenter, Randomized, Double-blind, Placebo-controlled Study Evaluating the Efficacy and Safety of JNJ-77242113 for the Treatment of Biologic-naïve Participants With Active Psoriatic Arthritis [Internet]. clinicaltrials.gov; 2025 Nov [cited 2025 Nov 13]. Report No.: NCT06878404. Available from: https://clinicaltrials.gov/study/NCT06878404
9. Janssen Research & Development, LLC. A Phase 3, Multicenter, Randomized, Double-blind, Placebo-controlled Study Evaluating the Efficacy and Safety of JNJ-77242113 for the Treatment of Biologic-experienced Participants With Active Psoriatic Arthritis [Internet]. clinicaltrials.gov; 2025 Nov [cited 2025 Nov 13]. Report No.: NCT06807424. Available from: https://clinicaltrials.gov/study/NCT06807424

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SECOND-GENERATION SENOLYTICS: The Future of Anti-Ageing Pharmacotherapy in Geriatrics

INTRODUCTION: Ageing is driven by the accumulation of **senescent cells** which are non-dividing, metabolically active "zombie cells" that secrete inflammatory mediators (SASP). These cells accelerate organ decline, frailty, metabolic dysfunction, and age-related diseases. **Second-generation senolytics** represent the next major leap in anti-ageing therapeutics, offering high specificity and safety compared with older agents.

Why Geriatrics Needs Them : Senescent cells accumulate in key systems:

- **Joints** → osteoarthritis
- **Cardiovascular system** → HFpEF, vascular stiffness
- **Kidney** → CKD progression
- **Brain** → neuroinflammation & cognitive decline
- **Muscle** → sarcopenia & frailty

Clearing senescent cells targets ageing **at its molecular source**

How Second-Generation Senolytics Work :

AGEING → DNA Damage → ↑p16 / ↑uPAR / FOXO4–p53 Stabilization

Senescent Cells

SASP → Chronic Inflammation

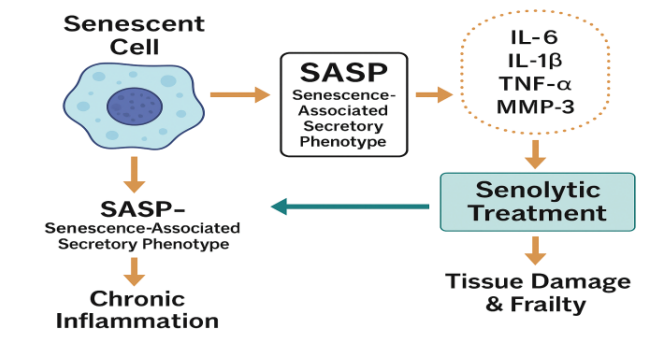
Tissue Damage, Frailty, Organ Decline

SECOND-GENERATION SENOLYTICS

FOXO4-DRI DT2216 uPAR-CAR-T p16-Nano

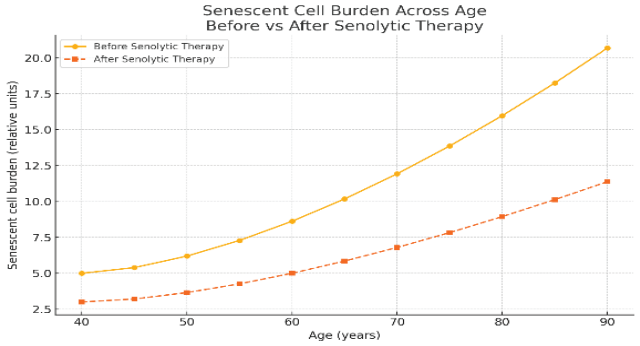
Selective Senescent Cell Removal → ↓SASP → ↑Regeneration

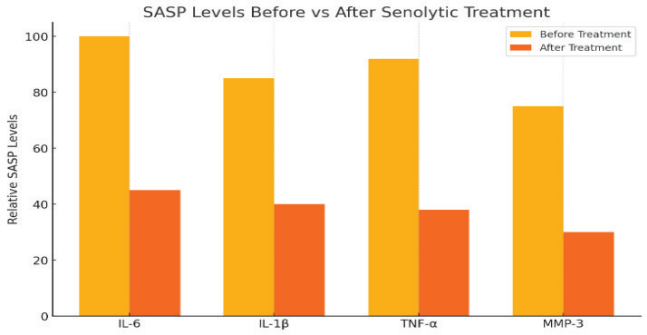
What Are Senolytics? Senolytics are drugs that **selectively eliminate senescent cells**, reducing SASP-driven inflammation and restoring organ resilience. Benefits include improvements in mobility, bone health, metabolic stability, vascular function, renal performance, and cognition.



First vs Second Generation Senolytics : At a Glance

First gen	Second gen
Broad action	High specificity
Affect normal cells	Targets p16, uPAR, FOXO4
Higher toxicity	Minimal adverse effects
Examples: D+Q, Fisetin	FOXO4-DRI, DT2216, uPAR-CAR-T
Limited clinical translation	Strong translational readiness





Leading Second-Generation Senolytic Agents

A) FOXO4-DRI Peptide-Breaks FOXO4–p53 interaction → induces apoptosis strictly in senescent cells → improves organ repair.

B) PROTAC Senolytics (DT2216)-Selective BCL- Xl degradation → avoids thrombocytopenia seen with older drugs → progressing through Phase 1–2 trials.

C) uPAR-CAR-T Senolytic Cells-Engineered CAR-T cells eliminate uPAR-high senescent cells → strong effects in fibrosis, metabolic ageing.

D) p16-Targeted Nanoparticle Senotherapeutics- Nanocarriers deliver drugs exclusively to p16-high senescent cells → reduced systemic toxicity.

Current Limitations

- Senescent markers vary across tissues
- Immune-related risks with CAR-T
- Need for long-term safety data
- Limited large-scale geriatric trials

Why Second-Generation Senolytics Stand Out

✓ Precision Targeting

Recognize senescence-specific markers → spare normal cells.

✓ Better Safety Profile

Reduced toxicity → suitable for older adults with multi-morbidity.

✓ Multi-Organ Benefits

Improved mobility, joint function, vascular elasticity, renal filtration, insulin sensitivity, and cognition.

✓ Intermittent Dosing

Short treatment cycles produce long-lasting benefits.

✓ Synergistic Potential

Enhances outcomes when combined with rehab, anti-inflammatory therapy, and metabolic drugs.

What the Next 5–7 Years May Bring:

- DT2216 advancing to Phase 2 efficacy testing
- Peptide senolytics closer to first-in-human studies
- Senolytic CAR-T therapies moving toward clinical translation
- Potential early clinical use in: frailty, sarcopenia, osteoarthritis, CKD, HFpEF, pulmonary fibrosis, neuroinflammatory ageing.

REFERENCES:

1. Kirkland JL, Tchkonia T. Advances in senolytics. *JCI Insight*. 2023.
2. Sougata Sarkar. Pharmacology Update 2024 – Senotherapeutics & Ageing Biology.
3. Mahadevan D et al. Clinical Development of PROTAC DT2216 for Senolysis. *Clinical Cancer Research*. 2024.
4. Goodman & Gilman's The Pharmacological Basis of Therapeutics, 14th Edition (2024 Update on Ageing Pharmacology).
5. Frontiers in Ageing Research. Senolytics & Senotherapeutics — Comprehensive Review 2024.
6. Cellular Senescence Review Committee. Senolytics & Gerotherapeutic Pathways. *Frontiers in Geroscience*. 2025.
7. Kirkland JL, Tchkonia T. Advances in Senolytic Pharmacology. *JCI Insight*. 2025.

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Direct patient reporting in pharmacovigilance

Background:

Direct patient (consumer) reporting — the submission of suspected adverse drug reactions (ADRs) by patients, caregivers or consumers — complements health professional reports in spontaneous reporting systems and has been progressively enabled by regulators and national programmes worldwide. It is now an accepted route for pharmacovigilance data collection by WHO-UMC, national centres (including PvPI/IPC in India), and regulatory agencies such as the US FDA. [1–3]

How patients report ADR:

Mechanisms vary by jurisdiction and include online portals, downloadable forms, telephone helplines, mobile apps and QR code links to web forms. Patient forms are usually simplified (consumer versions of professional ADR forms) and capture patient identifiers, suspected medicine(s), event description, onset dates, seriousness and reporter contact details. National programmes commonly forward validated reports to WHO-UMC / global databases for signal detection. [2,3]

Benefits:

1. **Complementary content**: Patient reports often provide richer narrative descriptions of symptom onset, severity and impact on daily life compared with HCP reports — yielding complementary data that may aid signal detection and benefit-risk assessment. Systematic reviews and empirical studies indicate patients report different reaction types and can highlight patient-centred outcomes. [4,5]
2. **Novel signals & under-recognized ADRs**: Patient reports have contributed to detection of new or previously under-reported ADRs, particularly those affecting quality of life (e.g., cognitive or sensory adverse effects) that patients notice earlier. [4,6]

Engagement & trust: Facilitating patient reporting may increase public engagement in medicine safety, improve health literacy and build trust in pharmacovigilance processes when patients receive feedback. [7]

Limitations and challenges:

1. **Under-reporting and awareness**: Despite positive trends, patient reporting rates remain low in many settings due to lack of awareness, uncertainty about what to report, and perceptions that reporting is a clinician's role. [4,8]
2. **Data quality and completeness**: Patient reports can lack clinical detail (e.g., lab results, concomitant conditions), making causality assessment harder; however, structured forms and follow-up can mitigate this. [3,4]
3. **Duplicate reports & signal noise**: With multiple reporting routes (HCPs, patients, manufacturers) duplication and variable terminology can complicate database handling; robust deduplication and coding are required. [9]

Digital divide & equity: Reliance on online tools may disadvantage populations with low digital access; alternatives (phone, paper, community outreach) remain important. [10]

Implementation:

- **User-friendly forms**: Provide clear, short consumer forms (plain language) and multiple modalities (web, phone, paper, QR code) to maximize accessibility. Examples: FDA Form 3500B (consumer), PvPI consumer helpline and forms. [2,3]
- **Public awareness campaigns**: Education campaigns targeting patients, pharmacists and the public increase reporting. National PvPI experience shows improved reporting after outreach and training. [11]
- **Feedback & follow-up**: Providing acknowledgement and (where possible) feedback encourages continued patient participation and trust. [7]

Standardised coding & causality methods: Use WHO-UMC causality assessment and standard terminologies (MedDRA) to harmonize patient reports for analysis. [9,12]

Conclusion:

Direct patient reporting is a valuable and increasingly indispensable component of modern pharmacovigilance. It enriches safety databases with patient-centred details, can reveal unique signals, and strengthens public engagement — provided systems address awareness, accessibility, data quality and analytic challenges. National programmes (e.g.,

PvPI/IPC) and regulators (WHO-UMC, FDA) provide practical models and tools that can be adapted to local contexts. Continued investment in outreach, user-centred reporting tools, and robust database practices will maximize the utility of patient reports for safer medicines.

References:

1. WHO Uppsala Monitoring Centre. *Guidelines for setting up and running a pharmacovigilance centre* (WHO-UMC). Uppsala: WHO-UMC. [Internet]. Available from: WHO-UMC guidance. (Uppsala Monitoring Centre)
2. US Food and Drug Administration. *MedWatch Forms for FDA Safety Reporting* (consumer and health professional forms; MedWatch). FDA. 2025 Oct 22 [cited 2025 Nov 12]. Available from: MedWatch forms page. (U.S. Food and Drug Administration)
3. Indian Pharmacopoeia Commission (National Coordination Centre — PvPI). *Suspected Adverse Drug Reaction Reporting Form & guidance for consumers/ professionals*. Ghaziabad: IPC/PvPI. [Internet]. Available from: ADR reporting form (PvPI).
4. Inácio P, Cavaco A, Airaksinen M. The value of patient reporting to the pharmacovigilance system: a systematic review. Br J Clin Pharmacol. 2017;83(2):227–46. Available from: PubMed/PMC summary. (PubMed)
5. Sienkiewicz K, et al. The importance of direct patient reporting of adverse drug reactions: analysis of EudraVigilance data. Int J Environ Res Public Health. 2021;19(1):413. (MDPI)
6. Kruger P, Gasperini C. The value of direct patient reporting in pharmacovigilance. Ther Adv Drug Saf. 2020;11:2042098620940164. doi:10.1177/2042098620940164. (Editorial). (PubMed)
7. Sienkiewicz K, Kieć-Kononowicz K. The importance of direct patient reporting of adverse effects and the feedback loop—benefits for patients and pharmacovigilance. Int J Environ Res Public Health. 2021. (MDPI)
8. Global Forum / DIA / commentary: "How valuable is patient direct reporting in pharmacovigilance?" (industry/advocacy perspective). DIA Global Forum. 2021. (DIA Global Forum)
9. WHO-UMC. *Pharmacovigilance indicators / guidance for monitoring PV systems*. WHO-UMC. [Internet]. (Uppsala Monitoring Centre)
10. Dedefo MG, Ellett LK, Roughead EE, et al. Consumers' knowledge and experiences of adverse drug reaction reporting in Australia: a national survey. Eur J Clin Pharmacol. 2024. (SpringerLink)
11. Kalaiselvan V, Thota P, Singh S. Pharmacovigilance Programme of India: recent developments and future perspectives. Indian J Pharmacol. 2016;48(6):447–52. (PMC)
12. WHO Collaborating Centre for International Drug Monitoring (UMC). *The use of the WHO-UMC system for standardised case causality assessment*. UMC; 2013. (World Health Organization

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SITAGLIPTIN INDUCED BULLOUS PEMPHIGOID IN A PATIENT OF TYPE 2 DIABETES MELLITUS: A CASE REPORT

INTRODUCTION:

Bullous pemphigoid (BP) is an autoimmune blistering disease resulting from autoantibodies directed against hemidesmosomal proteins within the dermal-epidermal junction[1]. Although the precise cause remains unclear, BP has been associated with several medications, including DPP-4 inhibitors[2]. BP largely affects elderly patients, particularly those above 70 years[6], and can lead to significant morbidity and mortality. It presents with intense pruritus and tense bullae that may arise on both normal and erythematous skin.

Gliptins are widely used in the management of type 2 diabetes mellitus. They enhance incretin activity by inhibiting the DPP-4 enzyme responsible for incretin degradation[3]. Increasing evidence links gliptins to a spectrum of cutaneous adverse effects, including BP[4]. DPP-4 is expressed in multiple tissues including skin[5], suggesting a probable biological basis for this association.

We report a case of Sitagliptin-induced BP in an elderly diabetic male, contributing to the growing Indian and global evidence connecting gliptin therapy with autoimmune blistering disorders.

CASE REPORT:

A 75-year-old male with longstanding type 2 diabetes mellitus, coronary artery disease and hypertension was initiated on Sitagliptin in addition to his existing antidiabetic regimen. Approximately three months later, he developed multiple large, tense, haemorrhagic bullae over the feet and legs, which progressively generalised. Lesions occurred on both normal and erythematous skin and were associated with significant pruritus.Initial management with oral doxycycline and topical corticosteroids resulted in minimal improvement. Subsequently, he was started on oral Prednisolone (30 mg daily), tapered gradually over three months. This produced partial improvement but failed to induce complete remission.

Owing to the persistent disease activity, the patient was further evaluated and initiated on subcutaneous Omalizumab 300 mg once monthly. Over the next several months, the number and size of bullae decreased markedly, and symptoms improved substantially. Omalizumab was subsequently tapered and discontinued. For long-term disease control, he was transitioned to Mycophenolate Mofetil 500 mg daily, which he tolerated well.

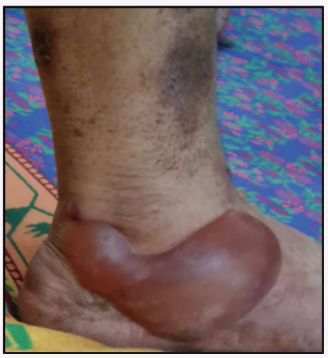


Figure 1: Showing large tense bulla with serous fluid on foot

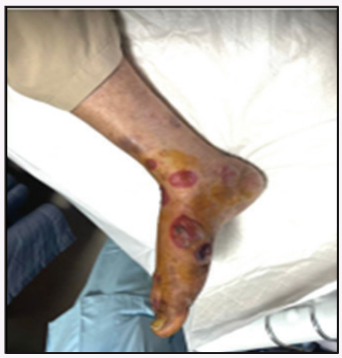
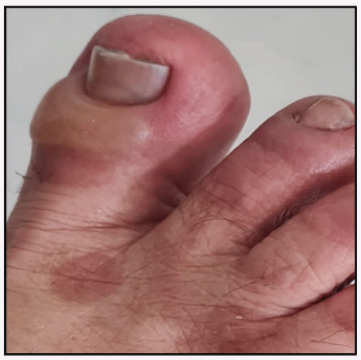


Figure 2: Showcasing multiple bullae on foot



Serological evaluation revealed:

BP180 antibody: POSITIVE (112.53 RU/mL)
BP230 antibody: NEGATIVE (10.37/ RU/mL)

Based on the temporal relationship between Sitagliptin initiation and symptom onset, the positive BP180 serology, and the favourable response following drug withdrawal, a diagnosis of Sitagliptin-induced bullous pemphigoid was made.

DISCUSSION:

This case illustrates a probable instance of Sitagliptin-associated bullous pemphigoid in an elderly diabetic patient. Numerous observational studies and meta-analyses have confirmed an association between DPP-4 inhibitors and BP[7]. The latency period between drug initiation and symptom onset varies widely, ranging from a few days to several years[8]. In our patient, onset at three months is consistent with previously reported timelines.

While all gliptins have been implicated, the risk varies among individual agents. Vildagliptin appears to carry the highest risk, but Sitagliptin and Linagliptin have also demonstrated significant associations[7].

The pathophysiology of gliptin-related BP remains incompletely understood. DPP-4 is involved in T-cell regulation and immune signalling[9]. Its inhibition may unsettle immune tolerance mechanisms and promote autoimmune responses against BP180 and BP230 antigens, leading to blister formation[10].

Clinically, gliptin-associated BP may resemble classical BP, as seen in our patient, though atypical presentations with non-inflammatory lesions have also been documented[8].

Diagnosis relies on clinical features, immunological testing and, crucially, recognition of temporal association with gliptin exposure. Management requires prompt discontinuation of the offending drug along with standard BP therapy. This patient responded well to a combination of systemic corticosteroids, Omalizumab and subsequent Mycophenolate, consistent with existing therapeutic strategies. Drug withdrawal played a central role in disease improvement, further supporting causality.

CONCLUSION:

This report underscores the association between Sitagliptin and bullous pemphigoid and highlights the importance of clinical vigilance when prescribing gliptins, especially in elderly patients. New-onset pruritus or bullous lesions in diabetic individuals should raise suspicion for drug-induced BP. Early recognition and immediate withdrawal of the triggering agent are essential for achieving disease control and reducing morbidity.

REFERENCES:

1. Kasperkiewicz M. Clin Rev Allergy Immunol. 2007;33:67-77.
2. Chouchane K. J Transl Med. 2021;19:520.
3. Herman GA. J Clin Pharmacol. 2006;46:876-86.
4. Desai S. Arch Intern Med. 2010;170:1169-71.
5. Thielitz A. Front Biosci. 2008;13:2364-75.
6. Parker S. Clin Dermatol. 2011;29:69-79.
7. Kridin K. J Am Acad Dermatol. 2018;79:659-67.
8. Prajesh K. Indian Dermatol Online J. 2024;15:836-40.
9. Attaway A. J Dermatol Case Rep. 2014;8:24-8.
10. Benzaquen M. J Am Acad Dermatol. 2018;78:1090-6.

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Contribution of Pharmacologists to Pharmacotherapeutics

Scan QR to Access:

What is Digital Drug Information Center?

A Digital Drug Information Center is an online or electronic platform designed to collect, store, manage, and disseminate reliable information about drugs and therapeutics.

Need for a Drug Information Center(DIC)

- Provides authentic & unbiased information (unlike MR, who highlight benefits and hide harms).
- Offers complete knowledge: benefits, risks, alternatives, and cost aspects.
- Especially supports residents & young doctors for safe, evidence-based prescribing.

Purpose and Scope – Why Was This Created?

Purpose	Scope
Promote rational drug use	Drug monographs (uses, doses, contraindications)
Ensure patient safety	Therapeutic guidelines & treatment protocols
Provide evidence-based information	Drug interaction checking
Support healthcare professionals	ADR reporting & monitoring
Encourage pharmacovigilance	Hospital formulary management
Aid education & training	Public health programs, research & academia

Who Can Use This? – Real-World Scenarios - **Doctors**, **Pharmacists**, **Nurses**, **Students & Academics**. **Hospitals & Institutions**, **Public Health Programs**, **Researchers**.

Key Features that Set DIC Apart

- Evidence-based – relies on authentic, unbiased sources.
- Updated – regularly revised with the latest drug guidelines & safety alerts.
- Problem-oriented – addresses specific queries from healthcare professionals.
- Comprehensive – covers drug data, interactions, ADRs, and guidelines.
- Accessible – available via phone, email, online, or direct contact.
- Patient safety focused – promotes rational drug use & reduces errors.
- Educational – supports teaching, training, and academic growth.
- Pharmacovigilance linked – encourages ADR reporting & monitoring

References

1. Behera SK, Xavier AS, Gunaseelan V, Ravindra BK, Selvarajan S, Chandrasekaran A, Dkhar SA. Drug information center as referral service in a South Indian tertiary care hospital. Int J Pharm Investig. 2017;7(4):182-7. doi:10.4103/jphi.JPHI_90_17. PMID:29692977; PMCID:PMC5903022.
2. Gabay MP. The evolution of drug information centers and specialists. Hosp Pharm. 2017;52(7):452-3. doi:10.1177/0018578717724235.
3. de Sousa IC, de Lima David JP, Noblat Lde A. A drug information center module to train pharmacy students in evidence-based practice. Am J Pharm Educ. 2013;77(4):80. doi:10.5688/ajpe77480. PMID:23716748; PMCID:PMC3663634.
4. Amerson AB. Drug information centers: an overview. Drug Inf J. 1986;20(2):173-8. doi:10.1177/009286158602000209. PMID:10277071.

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OVERVIEW OF CONTEMPORARY CLINICAL TRIAL DESIGNS

TRIAL DESIGN	PRIMARY OBJECTIVE	KEY FEATURES	ADVANTAGES	LIMITATIONS	EXAMPLE
Superiority Trial	To show the new treatment is better than standard/ control	Requires statistically significant difference in outcome	Strong clinical evidence;widely accepted	Needs large sample size	DAPA-HF trial showing dapagliflozin superiority
Bioequivalence Trial	To show two products have similar bioavailability	Compares AUC, Cmax; 90% CI within 80–125%	Approves generics; cost-effective	Limited to PK evaluation	Generic vs. innovator metformin bioequivalence
Non-inferiority Trial	To show new treatment is not worse than standard by a set margin	Uses predefined NI margin	Useful when new therapy is safer/ cheaper	Margin selection subjective	Apixaban non-inferior to Warfarin in AF
Sequential Trial	To allow interim analysis and early stopping	Allows early stop for benefit/futility	Saves time/resources	Statistically complex	RECOVERY dexamethasone arm early stop
Basket Trial	To test one therapy across multiple diseases with same biomarker	Tumor-agnostic; biomarker-driven	Useful for rare mutations	Heterogeneity across diseases	NCI-MATCH trial
Umbrella Trial	To test multiple therapies in one disease based on biomarkers	Multi-arm within one disease	Personalized therapy	Logistically complex	Lung-MAP trial in lung cancer
Challenge Trial (Human Infection Model)	To assess intervention by deliberately exposing participants to a controlled pathogen	Participants are infected under monitored conditions	Requires smaller sample size; rapid evaluation	Ethical concerns; limited to low-risk pathogens	Controlled Human Malaria Infection (CHMI) studies

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Paracetamol in the Womb: Decoding the Autism Puzzle

Introduction

Paracetamol (acetaminophen) is the most commonly used antipyretic during pregnancy and is used as the first-line option since alternatives like non-steroidal anti-inflammatory drugs (NSAIDs) are not considered safe throughout the period of gestation. However, recently there has been concern regarding the possible association between exposure to paracetamol prenatally and risk of neurodevelopmental disorders such as autism spectrum disorder (ASD) and attention-deficit/hyperactivity disorder (ADHD).This has come in light of the press statement by the U.S. Food and Drug Administration (FDA) in September 2025, in which it announced that that it would review the product labelling of Acetaminophen, mentioning the observational evidence that "maternal use of acetaminophen during pregnancy may be associated with an increased risk of neurological conditions such as ASD and ADHD" in children. However, FDA also has stated that a causal relationship has not been established yet, and that Acetaminophen remains the only over-the-counter drug approved for fever during pregnancy. FDA has urged clinicians to prescribe Paracetamol only when necessary medically and to prescribe the lowest effective dose for the shortest duration possible.

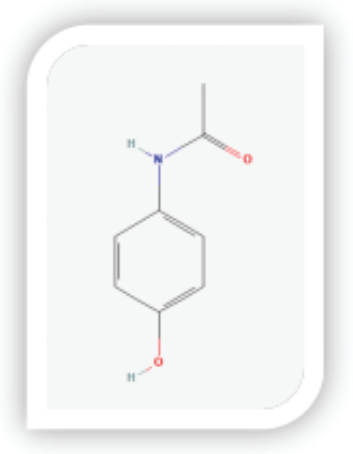

Fig1. Structure of Paracetamol (Source: PubChem)

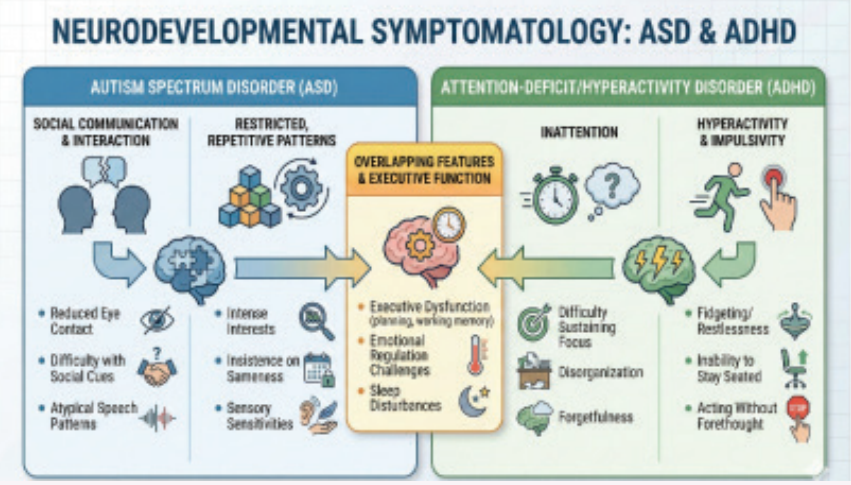

Fig2. Clinical Manifestations of ASD & ADHD (Source: Generated by Author)

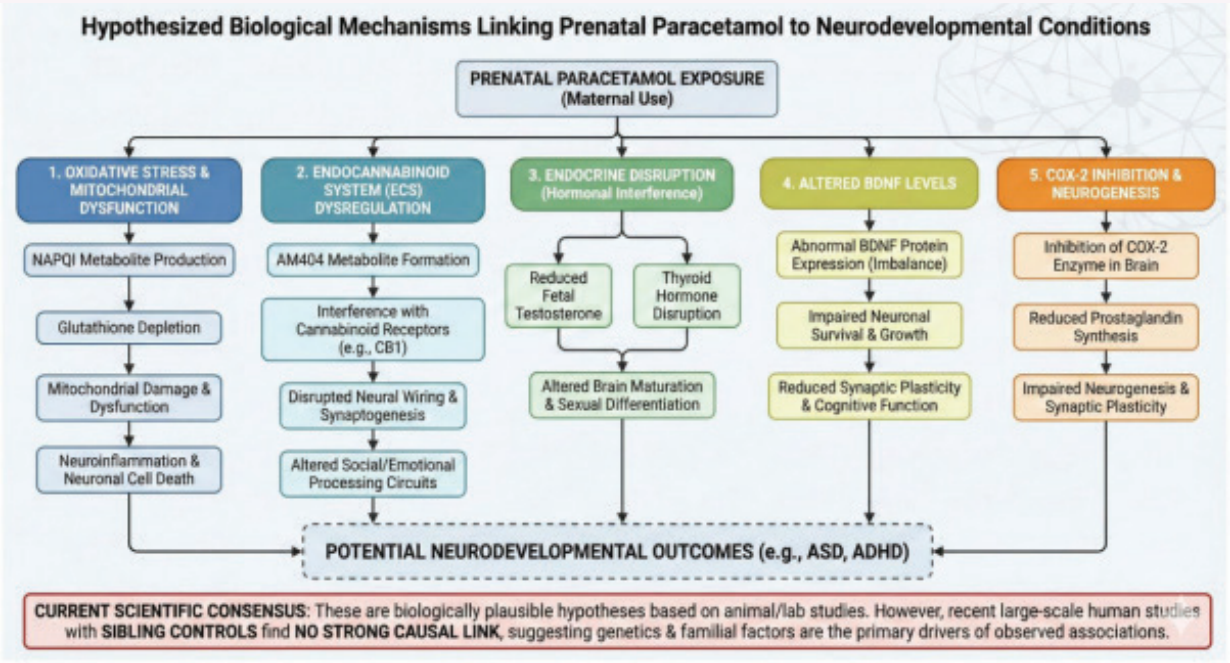

Fig1. Proposed Hypotheses linking Paracetamol to Neurodevelopmental conditions (Image: Generated by Author)

Proposed Hypotheses: Linking Paracetamol to Neurodevelopmental Disorder: Several pre-clinical models & human observational data have proposed the following hypotheses linking Paracetamol to ASD/ADHD:

Human studies - Evidence So Far

While observational studies of the past had suggested correlation between Paracetamol and autism/ADHD risk – recent large, well-designed human studies have so far shown only small associations which weaken or disappear after adjustment for confounding factors such as family history or the maternal fever itself.

Large Cohort Data

A recent nationwide study in Sweden by Ahlqvist VH et al. ,which included around 2.48 million births from 1995 to 2019 , demonstrated that prenatal consumption of Paracetamol was associated with only marginally higher risk of ASD (hazard ratio [HR] ~1.05 with an absolute risk difference ~0.09%). However, in the matched sibling analysis which controls for shared genes and family history, there was no association (HR 0.98, 95% CI 0.93–1.04). ADHD and intellectual disability showed the same pattern..

Meta-Analyses and Systematic Reviews

A 2025 systematic review and meta analysis by Ricci et al which pooled observational studies - revealed no statistically significant association between prenatal use of paracetamol and ASD (pooled odds ratio 1.10, 95% CI 0.98–1.24). A small association with ADHD was reported but the studies included were acknowledged to be heterogenous. A nationwide study by Yusuke Okubo et al. indicated that although there was small increase in risk , further sensitivity analyses suggested partial explanation by confounding factors. An umbrella review study by Sheikh J et al. which included multiple meta-analyses demonstrated the fact that while many published studies reported positive correlations with ASD or ADHD, the certainty of evidence was low to critically low – mainly due to presence of confounding factors, misclassification of exposure, and an overlap of primary datasets.

Confounding Factors which cloud judgement

Several strong alternative confounding factors have been documented including:

Confounding by the indication: Indications such as maternal fever, infection, migraines, or chronic pain are conditions that themselves may affect the foetal neurodevelopment.

Genetic factors: Neurodevelopmental disorders can occur hereditarily.

Non-specificity to Paracetamol : Similar small ASD/ADHD signals are seen with other analgesics (NSAIDs, opioids) and not exclusive to Paracetamol.

Current Guidelines

No regulatory agency has currently advised against the paracetamol use in pregnancy when medically indicated.

The FDA itself stated that acetaminophen still remains appropriate in pregnancy and that it should be used judiciously but also warns clinicians and the public to be aware of the evidence even if no definite causal proof has ben demonstrated.

The American College of Obstetricians and Gynecologists (ACOG) stated in 2025 that "acetaminophen plays an important and safe role in the well-being of pregnant women" when used correctly.

The Society for Maternal–Fetal Medicine (SMFM) similarly reinstated that current studies do not still demonstrate a causal relationship of Paracetamol with ASD.

Conclusion

The possible link between prenatal exposure to Paracetamol exposure and Autism/ADHD remains clouded and unresolved with the balance of high-quality evidence leaning towards no meaningful causal association. Observed associations may be plausibly explained by the underlying maternal illness itself or genetics, and other confounding factors. Paracetamol remains the first choice drug for maternal fever- however, as stated by FDA - clinicians should recommend paracetamol only when it is indicated and should do so at minimal effective doses and patients need to consult the physician before consumption of Paracetamol. Further rigorously controlled research like sibling controlled studies are the need of the hour to further study this association and uncover data without confounding factors.

References

1. Sheikh J, Allotey J, Sobhy S, Plana MN, Martinez-Barros H, Naidu H, Junaid F, Sofat R, Mol BW, Kenny LC, Gladstone M. Maternal paracetamol (acetaminophen) use during pregnancy and risk of autism spectrum disorder and attention deficit/hyperactivity disorder in offspring: umbrella review of systematic reviews. bmj. 2025 Nov 10;391.
2. Ahlqvist VH, Sjöqvist H, Dalman C, Karlsson H, Stephansson O, Johansson S, Magnusson C, Gardner RM, Lee BK. Acetaminophen use during pregnancy and children's risk of autism, ADHD, and intellectual disability. Jama. 2024 Apr 9;331(14):1205-14.
3. https://www.fda.gov/news-events/press-announcements/fda-responds-evidence-possible-association-between-autism-and-acetaminophen-use-during-pregnancy
4. Okubo Y, Hayakawa I, Sugitate R, Nariai H. Maternal acetaminophen use and offspring's neurodevelopmental outcome: a nationwide birth cohort study. Paediatric and perinatal epidemiology. 2025 Sep 2.
5. Parker W, Hornik CD, Bilbo S, Holzknecht ZE, Gentry L, Rao R, Lin SS, Herbert MR, Nevison CD. The role of oxidative stress, inflammation and acetaminophen exposure from birth to early childhood in the induction of autism. Journal of International Medical Research. 2017 Apr;45(2):407-38.
6. Viberg H, Eriksson P, Gordh T, Fredriksson A. Paracetamol (acetaminophen) administration during neonatal brain development affects cognitive function and alters its analgesic and anxiolytic response in adult male mice. Toxicological sciences. 2014 Mar 1;138(1):139-47.
7. van den Driesche S, Macdonald J, Anderson RA, Johnston ZC, Chetty T, Smith LB, Mckinnell C, Dean A, Homer NZ, Jorgensen A, Camacho-Moll ME. Prolonged exposure to acetaminophen reduces testosterone production by the human fetal testis in a xenograft model. Sci Transl Med 7 (288): 288ra80 [Internet]. 2015
8. Hinz B, Cheremina O, Brune K. Acetaminophen (paracetamol) is a selective cyclooxygenase-2 inhibitor in man. The FASEB journal. 2008 Feb;22(2):383-90.

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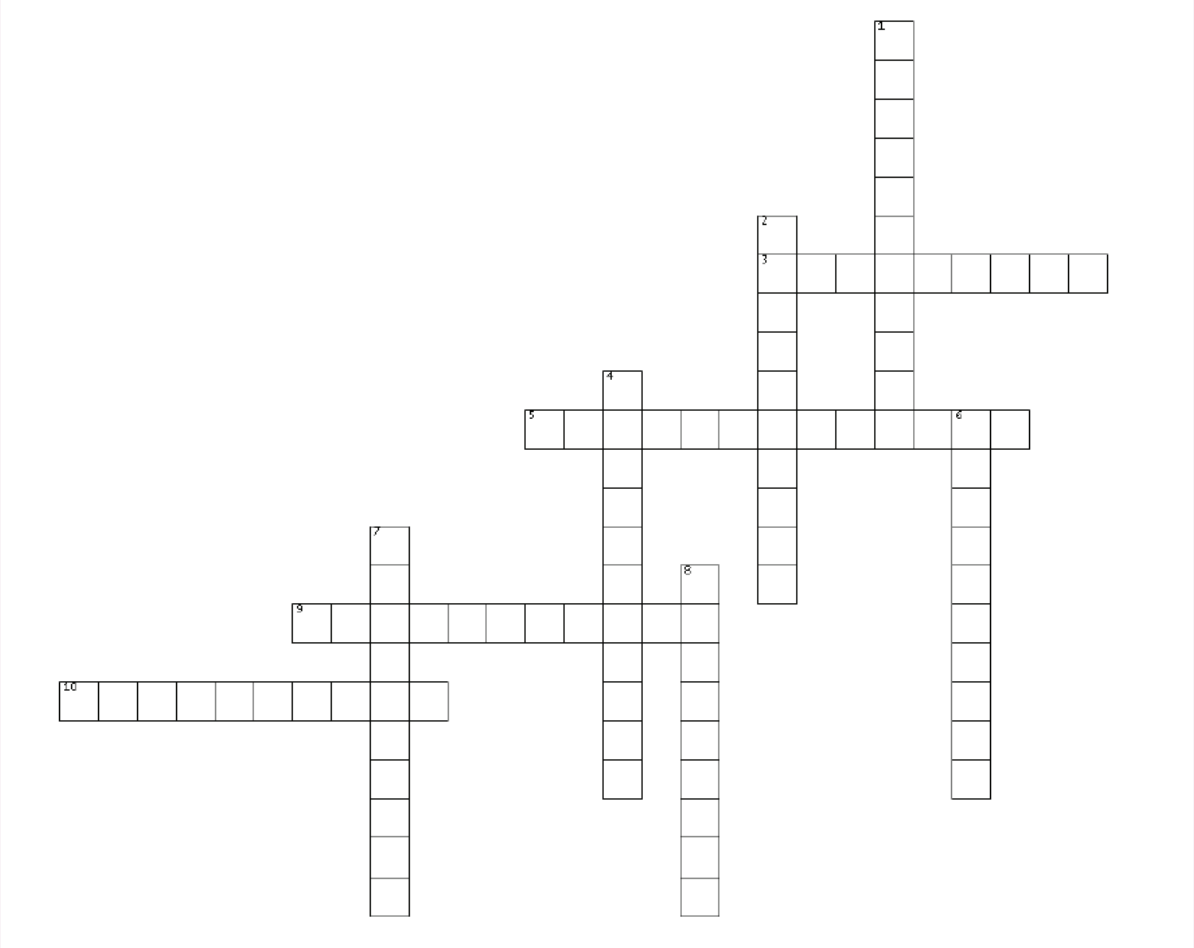
Cross word puzzle



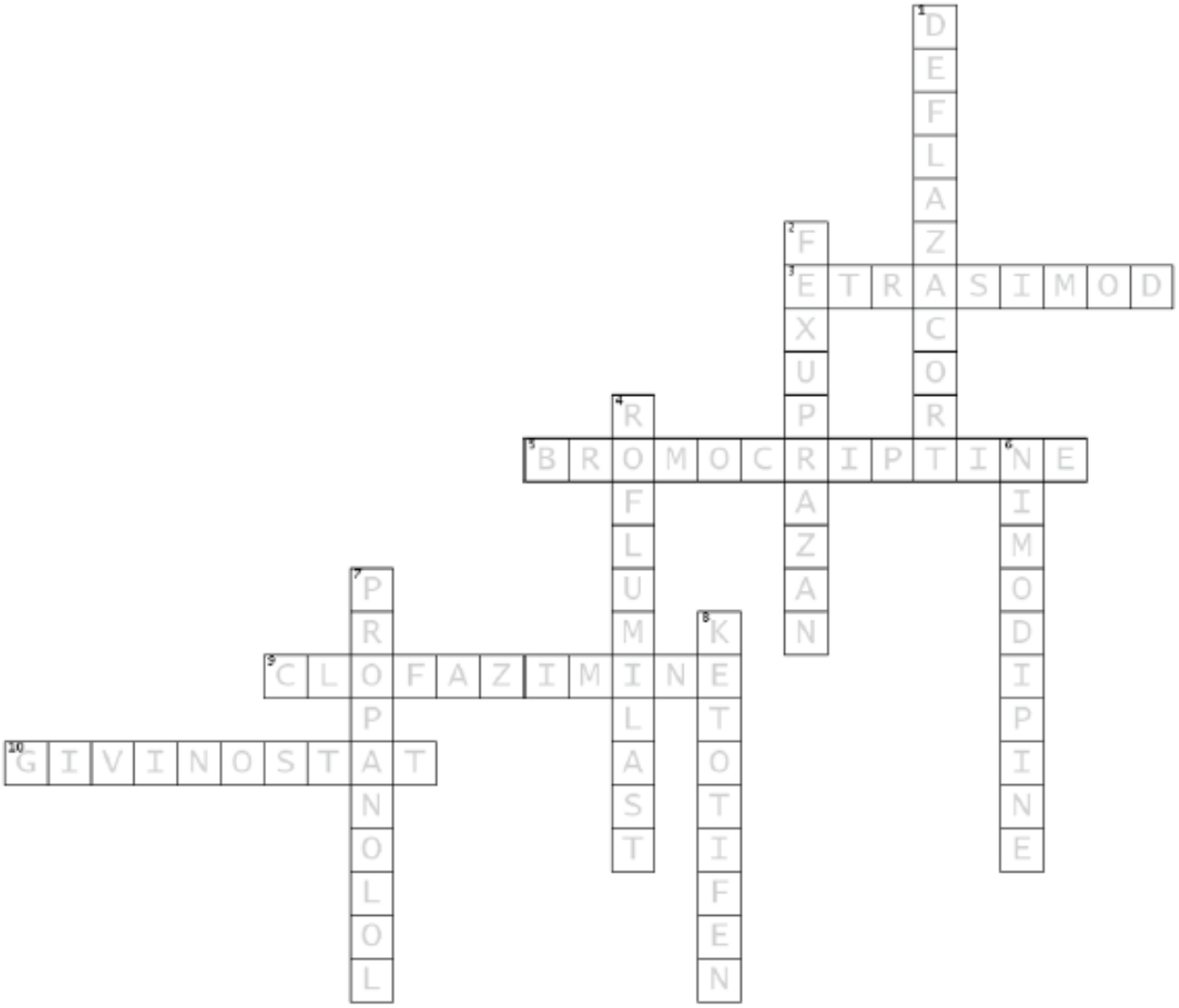
ACROSS

3. S1P receptor modulator for ulcerative colitis & New oral drug for ulcerative colitis(9)
5. Ergot derivative used in Diabetes treatment(13)
9. Anti-inflammatory, Immunomodulatory, Antibiotic drug for Chronic disease causing Skin disclouration(11)
10. First oral drug for Duchenne Muscular Dystrophy(10)

DOWN

1. Glucocorticoid without mineralocorticoid activity used for long term use(11)
2. New P-CAB drug approved in india for GERD, known for its rapid action(10)
4. Phosphodiesterase inhibitor used in COPD and Psoriasis(10)
6. Dihydrpyridine CCB used for cerebral Vasodilatation(10)
7. First Non-Selective Betablocker(10)
8. Oral drug used for prophylaxis of Bronchial Asthma(9)

Answers:



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Extract:

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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.

#As per NMC MSR 2023:6.4. Department of Pharmacology- Animal holding area:F or teaching Pharmacology in undergraduate curriculum, the required knowledge and skills should be imparted by using computer assisted module. However, if research and postgraduate training is envisaged, only an animal hold area, as per CPCSEA Guidelines is required.

#Waiver for clinical efficacy studies for monoclonal antibody biosimilars: In a groundbreaking milestone for global healthcare and pharmaceutical innovation, Professor Sarfaraz K. Niazi, Adjunct Professor at the University of Illinois at Chicago and founder of multiple biopharmaceutical enterprises, has secured the first-ever FDA acceptance to waive clinical efficacy studies (CESs) for monoclonal antibody biosimilars. This decision fundamentally redefines how biological drugs will be developed, approved, and made affordable for patients worldwide.

 # Suggestion for P.G. e-logbook by a senior pharmacologist : Prepare a PG logbook format in Microsoft office word. Blank copy is given to PG student to write in it and update the information every month. They are instructed to upload blank format (word document file) on their google drive and share the link with PG guide and HOD. Updated information in their logbook can be accessed any time and from any place (need only internet connectivity)

#Issue of contaminated cough syrups: According to WHO, in the last 3 years, over 300 children have died from taking cough syrups - Gambia, Uzbekistan, Cameroon, India are some of the worst affected places. Glycerin and propylene glycol are used to sweeten syrups. They are sourced from industrial suppliers. If the supplier is not careful, they may get contaminated with ethylene glycol or diethylene glycol (DEG). Pharmaceutical grade ingredients are costlier than industrial grade ingredients. In the recent case, Coldrif syrup from Sresun pharmaceuticals in Kanchipuram, TN got contaminated with DEG. The syrup had 48.6% DEG. The permissible upper limit is 0.1 % . To tackle with these issue suggestion was that we should have separate division for each therapeutic category, all staffed by pharmacologists and analytical pharmacists in labs who report to the division head.

#Hyderabad paediatrician's eight-year battle leads to FSSAI prohibiting usage of ORS on food products: The Food Safety and Standards Authority of India (FSSAI) has issued an order stating that no food brand may use the term 'Oral Rehydration Salts' or 'ORS' on its products unless the formulation adheres strictly to the standards recommended by the World Health Organisation (WHO).

#WHO Recommends GLP-1s for Obesity Management in New Guidance: The new guideline "recognizes that obesity is a chronic disease that should be treated with comprehensive and lifelong care.The guideline contains two key conditional recommendations based on evaluations of two GLP-1 receptor agonists (liraglutide and semaglutide) and one dual glucagon insulinotropic polypeptide (GIP)/GLP-1 agonist (tirzepatide).

GLIMPSES OF NAPTICON-2025: EVENT IN A NUTSHELL

The Fourth Annual Conference of the National Association of Pharmacology and Therapeutics (NPT) was held at PSG Institute of Medical Sciences & Research, Coimbatore, from **28–29 November 2025**, with pre-conference workshops on **27 November**. The meeting was a grand success, bringing together **700+ delegates** from across India.

Three well-received pre-conference workshops were conducted on:

A. PK–PD Modelling & Simulation in Phase I Trials

B. Simulation-based Learning in Pharmacotherapeutics

C. LC-MS and AAS in Drug Analysis Each was led by experts from academia and industry.

The conference opened with an impressive inaugural ceremony graced by **Dr. A. Senthil Vadivu** as Chief Guest, along with addresses by Dr. K. Bhuvaneswari and Dr. T.M. Subbarao. Highlights included a keynote by **Dr. C.M. Kamaal**, release of the souvenir and books, NPT progress report, and annual awards.

Across two days, the event featured **12 plenary** and **9 short sessions**, along with **310+ e-posters and oral presentations** across multiple venues. Competitive sessions included the **Young Pharmacologists Speak (YPS)** and the prestigious **Dr. S. Ramalingam Memorial Award** for postgraduate oration in Pharmacogenetics/ Bioethics. The conference also hosted major NPT events including Life-time Achievement Award (to **Dr. Urmila Thatte**), National Spotlight Awards, and NPT Research Awards.

The much-anticipated **NAPTIQUIZ-2025**, led by Dr. C.M. Kamaal and Dr. Jeetendra Singh, shortlisted finalists from ~50 teams, culminating in an exciting finale on day two.

The conference also hosted major NPT events including Life-time Achievement Award (to **Dr. Urmila Thatte**), National Spotlight Awards, and NPT Research Awards.

The much-anticipated **NAPTIQUIZ-2025**, led by Dr. C.M. Kamaal and Dr. Jeetendra Singh, shortlisted finalists from ~50 teams, culminating in an exciting finale on day two. The evening of Day one of Conference was fully lighted by a Conclave meeting of Senior Experts in Pharmacology all over the Country, by invitee only at Sarvaa Ball Room, Hotel Grand Regend, Coimbatore. The Day two evening comprised of Gala Dinner and Cultural festival . Day two concluded with the **pink cap ceremony**, handing over NAPTICON-2026 to **CMC Ludhiana** under Dr. Dinesh K. Badyal, followed by the General Body Meeting and valedictory. The event was supported by trade stalls and covered by two press meets.

With the theme **"Crafting Clinical Excellence with Pharmacology and Therapeutics,"** NAPTICON-2025 successfully delivered high-quality scientific learning, meaningful discussions, and widespread engagement through the joint efforts of NPT and PSG IMSR.


NAPTICON
Organized by
Department of Pharmacology
PSG Institute of Medical Sciences & Research
Coimbatore


PSG
INSTITUTE OF MEDICAL SCIENCES & RESEARCH
Since 1985
NATIONAL ASSOCIATION OF PHARMACOLOGY AND THERAPEUTICS

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

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