Friday, 14th November 2025

IRISH ASSOCIATION OF PHARMACOLOGISTS

ANNUAL MEETING

(IAP 2025)

Who We Are

Welcome to The Irish Association of Pharmacology (IAP), a distinguished organisation dedicated to representing Pharmacology and Therapeutics on the island of Ireland. Founded in the mid-1990s by the esteemed late Professor John Feely, a visionary researcher and clinician, the IAP remains committed to fostering the advancement of pharmacology locally and globally.

In 2016, the IAP gained formal recognition as an unincorporated association, solidifying its status as a reputable and influential entity within the field. Building upon the remarkable legacy of Professor Feely, our association strives to honour his ambitions and unwavering dedication to the development of pharmacology.

Our Future Aims

- **Advancing Pharmacology Research**: We wholeheartedly promote both basic and clinical pharmacology research throughout the island of Ireland, fostering scientific discoveries that drive medical advancements and improve patient outcomes.
- **Facilitating Exchange and Collaboration**: We provide a dynamic platform for scientists in Pharmacology & Therapeutics, as well as allied disciplines in research and industry, to exchange knowledge, ideas, and collaborate on innovative projects.
- **Forging International Connections**: As a member of esteemed international associations, we open doors to global forums and contacts, facilitating research, development, and fruitful international collaborations for our esteemed members.
- **Advocacy for Positive Change**: By becoming an effective advocate for the field, we champion the interests of pharmacologists and promote policies that drive progress, improve healthcare, and contribute to the betterment of society.

Irish Association of Pharmacologists

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Our Committee

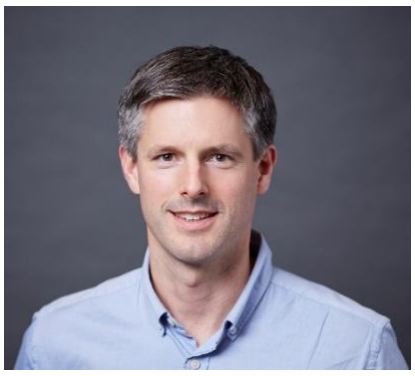
President: Dr Monica de Gaetano

Monica de Gaetano is an Assistant Professor in Pharmacology and a Principal Investigator of the Diabetes Complications Research Centre (DCRC) at University College Dublin. After obtaining her BSc in the School of Pharmacy in Italy, she moved to Ireland to pursue a PhD in Molecular Medicine and a PD training in Medicinal Chemistry. Her teaching consists in basic and advanced pharmacology, with a focus on cardiovascular and renal pharmacology, and on drug discovery and development.

She is an international leader in Resolution Pharmacology, with a long-standing interest in the monocyte-macrophage-foam cell axis role in progression and regression of atherosclerosis, where she has given an important contribution to characterise the plasticity of such pivotal cell axis. She coordinated a translational screening programme, through which she set up an *in vitro* platform for drug candidate discovery, as well as a robust *ex vivo* model of atherosclerosis. She has patented two novel molecules with pro-resolving properties in the context of vascular inflammation. Her research group at the UCD Conway Institute is currently focused on tackling the 'residual inflammatory risk' in diabetes-associated atherosclerotic patients to better tailoring therapeutic interventions.

More recently, she gained interest in the delivery of resolving molecules to the site of inflammation, *via* lipid nano particles-based systems, with the idea to design a site-specific drug delivery to macrophages in athero-prone regions of the vessel walls.

Vice President: Dr Cormac Kennedy



Dr Cormac Kennedy is currently a Consultant Clinical Pharmacologist and Physician at St James Hospital as well as a Clinical Senior Lecturer at Trinity College Dublin. His experience gives him a perspective of the journey of medicines from the bench to the bedside including the pharmaceutical, regulatory, economic, policy and clinical contexts. Cormac is a graduate of UCD Medical School and the School of Pharmacy Trinity College. He completed a PhD at the Royal College of Surgeons in Ireland, the product of which was subsequently patented. He received a HSE-NDTP Management and Leadership Scholarship to undertake a Master's in Health Economics, Outcomes and Management at the London School of Economics and was awarded a Distinction. Recently, he completed a Master's in Clinical Trials at Oxford University. His clinical interests include the specialist treatment of hypertension and lipid disorders, and he has presented internationally and published in these areas. His other research interests are across a spectrum of areas related to medicines including pharmacoepidemiology, pharmaco-economics and appropriate drug usage.

Secretary: Dr Aisling Heeran

Dr Aisling Heeran obtained a BSc in Pharmacy and M. Pharm from RCSI. Following this she completed an MSc in Translational Oncology from Trinity College Dublin (TCD). Aisling was awarded an Irish Research Council Government of Ireland Postgraduate Scholarship to complete a Ph.D. on radiation bystander events in gastrointestinal cancers in TCD. After completing her Ph.D., Aisling continued her research in gastrointestinal cancers, specifically examining the role of FKBPL across the Barrett's oesophagus to oesophageal adenocarcinoma disease progression. Aisling is now a Lecturer in Pharmacy in the School of Pharmacy and Biomolecular Sciences in RCSI and the International College for Pharmaceutical Innovation, SUDA, Suzhou, China.

Treasurer: Ms Natalie O'Regan

Natalie O'Regan serves as the Treasurer for the Irish Association of Pharmacologists, bringing a wealth of experience and dedication to the role. As the Executive Assistant in the Department of Pharmacology and Therapeutics at University College Cork, Natalie plays a vital role in supporting the department's operations. With a keen eye for detail and a passion for fostering collaboration within the field of pharmacology, Natalie ensures the financial well-being of the association. Her role as Treasurer involves managing the financial aspects of the organization, contributing to strategic decision-making, and supporting initiatives that advance the goals of the Irish Association of Pharmacologists. Natalie's commitment to excellence and her integral role in the academic community make her a valuable asset to the association. Her collaborative spirit and administrative expertise contribute to the smooth functioning of the organization and the success of its endeavours.

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Our Executive Committee

Professor David Williams ***(Past President, Advisor)***
Royal College of Surgeons, Ireland

Professor of Stroke Medicine and National Specialty Director for Clinical Pharmacology and Therapeutics. His research focuses on stroke medicine, hypertension, and patient safety. He co-directs the Irish Clinical Academic Training (ICAT) Programme and the Irish Stroke Clinical Trials Network (SCTNI).

Professor Steven Kerrigan ***(Advisor)***
Royal College of Surgeons, Ireland

Professor of Precision Therapeutics and Deputy Head of the School of Pharmacy and Biomolecular Sciences at RCSI. His translational pharmacology research has identified novel therapeutic and diagnostic targets in sepsis, and he is Founder/CEO of Inthelia Therapeutics.

Professor Christian Waeber ***(Past President, Advisor)***
University College Cork, Ireland

Professor Waeber's research explores sphingosine-1-phosphate receptor signalling and its neuroprotective potential in stroke and brain injury. His work advances understanding of preconditioning, neuroprotection, and immune regulation in stroke outcomes.

Professor Martina Hennessy ***(Advisor)***
Trinity College Dublin / St James's Hospital, Ireland

Clinical Pharmacologist and Consultant Physician with expertise in hypertension and cardiovascular risk. She is Director of the Welcome-HRB Clinical Research Facility and Chief Academic Officer for the Dublin Midlands Hospital Group, leading research and education integration.

Ms Orla Murphy ***(IAP Administrative Officer)***
University College Dublin, Ireland

Finance manager at UCD Conway Institute.

Mr Adam Kelly ***(Student Representative)***

PhD Graduate and IAP Student Representative, providing a voice for early-career pharmacologists and supporting engagement across the student and postgraduate community.

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ANNUAL MEETING

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Programme

Time	Activity
9:00 am (30 mins)	**Registration** at reception desk Location: Foyer
9:30 am (10 mins)	**Opening Remarks** by Dr Monica de Gaetano, IAP President (UCD SBBS, Dublin) Location: Red Room
9:40 am (100 mins)	**Session 1 (EXPERIMENTAL PHARMACOLOGY):** *"Bridging eras: Integrating classical pharmacology with cutting-edge therapies"* Location: Red Room Chair: Prof. Stephen Kerrigan (RCSI, Dublin) -- **9:40 – 10:25 am (Invited Keynote Speaker 1): "**Current concepts and innovative targets in antiplatelet therapy for disease prevention, treatment, and diagnostics: Advancing precision medicine" – **Prof. Paola Patrignani ("G. d'Annunzio" University, Chieti, Italy)** **10:25 – 10:40 am (Selected Speaker 1):** "Investigating the SRF signalling network as a druggable target in advanced prostate cancer" – **Dr Kim Zitzmann (UCD SBBS, Dublin)** **10:40 – 10:55 am (Selected Speaker 2):** "Investigating the atherosclerotic immune-modulatory potential of the multi-mineral supplement Aquamin" – **Dr Sashki Hans (TCD Medicine, Dublin)** **10:55 – 11:00 am (Elevator Pitch A)**: "Accelerating pharmacology with Thermo Fisher Scientific" – **Dr John Synnott (Thermo Fisher Scientific)**
11:00 am (25 mins)	**Coffee Break & Moderated Poster Session 1** (Even numbers) Location: Foyer

Time	Activity
11:25 am (80 mins)	**Session 2 (CLINICAL PHARMACOLOGY):** *"From bench to bedside: Challenges, unmet needs, and new perspectives"* Location: Red Room Chair: Prof. Catherine Godson (UCD Medicine, Dublin) -- **11:25 – 12:10 pm (Invited Keynote Speaker 2):** "Obesity and GLP-1 – Lessons from patients" – **Prof. Donal O'Shea (SVUH/UCD Medicine, Dublin)** **12:10 – 12:25 pm (Selected Speaker 3):** "Responses to medication and lifestyle questionnaires in a weight loss clinical trial for patients with resistant hypertension: An initial report" – **Mr Jayce O'Shields (TCD Medicine, Dublin)** **12:25 – 12:40pm (Selected Speaker 4):** "Stroke alert, but not a stroke: Unmasking mexiletine neurotoxicity" – **Dr Mary Enright (University Hospital Limerick)** **12:40 – 12:45 pm (Elevator Pitch B)**: "Validation or just viability – Are you letting your workflow flipping the coin often?" – **Dr Madhurima Mitra (Brennan & Co.)**
12:45 pm (45 mins)	**12:45 – 1:30 pm Lunch & Poster Viewing** Location: Foyer
1:30 pm (45 mins)	**John Feely Lecture & Award** Location: Red Room Chair: Prof. Mark Evans (BPS VP, Univ. of Edinburgh – UK) Awardee: **Prof. Christian Waeber**, Head of Pharmacology & Therapeutics (UCC, Cork): *"From serendipity to translation: A meandering journey through pharmacology and drug discovery"*
2:15 pm (80 mins)	**Session 3 (EDUCATIONAL PHARMACOLOGY):** *"Learning-by-doing: Active engagement strategies in pharmacology education"* Location: Red Room Chair: Prof. Helen Gallagher (UCD Medicine, Dublin) --

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Time	Activity
	2:15 – 3:00 pm (Invited Keynote Speaker 3): "From bench to brain: Enhancing pharmacology education through active engagement – **Assoc. Prof. Carmel Hensey,** Head of Pharmacology **(UCD SBBS, Dublin)** **3:00 – 3:15 pm (Selected Speaker 5):** "Game-based learning as a scalable solution for modern pharmacy education" – **Ms Ashley Ajie (RCSI, Dublin)** **3:15 – 3:30 pm (Selected Speaker 6):** "A pedagogical approach: Online interactive learning in the core principles of pharmacology" – **Dr Gerardine Meade-Murphy (UCC, Cork)** **3:30 – 3:35 pm (Elevator Pitch C): Mr Cameron Cruickshank (Avantor Science)**
3:35 pm (25 mins)	**Coffee Break & Moderated Poster Session 2** (Odd numbers) Location: Foyer
4:00 pm (45 mins)	**4:00 – 4:15 pm** **Annual General Meeting (AGM)** Location: Red Room Chair: Dr Cormac Kennedy, IAP VP (TCD Medicine, Dublin) **4:15 – 4:45 pm** **Roundtable Discussion** *"Being a pharmacologist: Duties & responsibilities"* Location: Red Room Chair: Prof. David Williams, IAP Executive Advisor (RCSI, Dublin)
4:45 pm (30 mins)	**Prof. John Crean Memorial Lecture** Location: Red Room Chair: Prof. Keith Murphy (UCD SBBS, Dublin) "Prof. John Crean. Pioneering the Future of Tissue Engineering and Regenerative Medicine: A Lasting Legacy" – **Dr Jessica Davis (UCD SBBS, Dublin)**
5:15 pm (15 mins)	**Prizes & Closure Remarks** Location: Red Room **5:15 – 5:25 pm:** BPS-sponsored Awards: **Prof. Mark Evans (BPS VP, UK)** **5:25 – 5:30 pm:** Closing remarks by **Dr Monica de Gaetano, IAP President (UCD SBBS, Dublin)**
From 5:30 pm	**Networking Evening** Location: UCD Clubhouse Student Bar

Attendees

Keynote Speakers

Prof. Paola Patrignani (Gabriele d'Annunzio University, Chieti, Italy)

Title: Current concepts and innovative targets in anti-platelet therapy for disease prevention, treatment, and diagnostics: Advancing precision medicine

Abstract:

The essential roles of platelets in haemostasis and atherothrombosis are well known, highlighting he importance of drug-based control of platelet activation to prevent thromboembolic events in atherosclerotic conditions. While current antiplatelet treatments significantly improve outcomes for coronary artery disease patients, they also increase the risk of bleeding. In addition to their functions in haemostasis and atherothrombosis, platelets also play a role in inflammatory processes, known as thrombo-inflammation, as well as in microcirculatory pathways. Managing platelet hyperreactivity could therefore affect tissue inflammation, such as myocardial ischemia, and vascular inflammation, including the development of vulnerable plaques, atherosclerosis, and cardiac fibrosis.

Evidence indicates that aspirin reduces cancer risk at doses used for cardiovascular prevention, although its exact mechanism remains partially understood. Low-dose aspirin (75-100 mg daily) irreversibly inhibits the cyclooxygenase (COX)-1 enzyme in platelets, preventing thromboxane (TXA2)-dependent platelet activation. Recent clinical trials and biomarker analyses have explored the associations among platelet activation, inflammation, and cancer development and progression. It is proposed that persistent platelet activation exerts two tumorigenic effects via TXA2 release: first, by triggering local inflammation at gastrointestinal mucosal lesions through COX-2 induction and increased prostaglandin (PG)E2 production, which contributes to early carcinogenesis; second, by impeding T-cell-mediated anti-cancer immunity through activation of TXA2 receptors on lymphocytes, thus facilitating cancer progression and metastasis.

Emerging therapies targeting innovative antiplatelet mechanisms—some with safer bleeding profiles—are in development, potentially opening new avenues for treating both cardiovascular disease and cancer and developing primary prevention strategies. Furthermore, advancements in platelet-based diagnostic tools exploit platelets' ability to acquire DNA from nucleated cells via the internalization of extracellular vesicles (EVs) and the uptake of cell-free DNA (cfDNA). When combined with platelet and EV proteomics and transcriptomics, and sophisticated bioinformatics, these developments have the potential to transform disease diagnosis and preventive

strategies for cardiovascular disease and cancer. These technologies could help identify individual patient platelet phenotypes, paving the way for personalized targeted therapies-promoting an era of precision antiplatelet treatment in cardiovascular disease and cancer.

Biography:

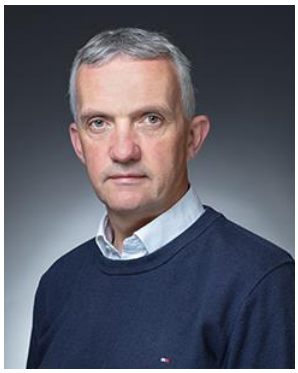
Professor Paola Patrignani, born in Rome, Italy, is an internationally renowned pharmacologist and one of Europe's leading authorities on eicosanoids, platelet biology, and inflammation in cardiovascular disease and cancer. She graduated with honours in Biological Sciences from the University of Rome *La Sapienza* in 1979 and carried out postgraduate research in pharmacology under the mentorship of Professor Carlo Patrono at the Catholic University, Rome.

Since 2002, she has served as Full Professor of Pharmacology at the *"G. d'Annunzio" University of Chieti-Pescara*, where she leads a multidisciplinary research group integrating pharmacology, molecular biology, lipidomics, and translational medicine. Her research has made seminal contributions to understanding the mechanisms of action of aspirin and other cyclooxygenase inhibitors, elucidating how these drugs modulate platelet activation, prostanoid biosynthesis, and tumour biology. Professor Patrignani's pioneering studies have provided key insights into aspirin's role in cardiovascular prevention and cancer chemoprevention, as well as the identification of biomarkers of drug response and toxicity relevant to precision medicine. She has authored over 220 peer-reviewed scientific papers, numerous book chapters, and holds six international patents. Her work has been cited extensively worldwide, reflected in an H-index of 60 (Scopus).

She is a Fellow of the International Union of Basic and Clinical Pharmacology (IUPHAR) and an active member of several international scientific societies. Among her many honours, she received the International Aspirin Foundation Senior Science Award (2018) in recognition of her outstanding contributions to the field.

Professor Patrignani continues to collaborate internationally to develop novel pharmacological strategies targeting eicosanoid pathways for the treatment and prevention of cardiovascular and neoplastic diseases.

Prof. Donal O'Shea (St. Columcille's Hospital and SVUH/UCD Medicine)
Consultant Endocrinologist



Title: **Obesity and GLP-1 – Lessons from patients**

Abstract:
I will focus on how the environment drives obesity and how the body regulates weight. We now know that for 90% of people weight gain is 90% irreversible. Adipose tissue thermogenesis regulates this process and is influenced by medication – contributing to medication induced weight gain. Understanding this interaction between medication and weight helps with empathy in raising the issue of weight and allows a positive start to address weight reduction with realistic goal setting and less self-stigmatisation. From a health or economic perspective, we can't afford to continue as we are. What we eat and drink is now a bigger driver of disease than smoking. I will also discuss how bold public policy is essential in shaping a food environment that protects our patient's health.

Persistent metabolic adaptation is a barrier to sustained weight loss. Glucagon-like peptide-1 (GLP-1) - based medicines are the class of pharmaceuticals underlying the newer obesity treatments. They are designed to mimic the action of a naturally occurring hormone in the body, which plays an important role in the regulation of satiety and metabolism. These medications are delivering significantly better weight-loss outcomes than other pharmacotherapies, with recent studies having shown body weight reduction of up to 30%. In humans, the primary mechanisms of action of GLP-1 analogs are considered to be the promotion of early satiety and delay of gastric emptying. We have previously demonstrated increased visceral adipose tissue (VAT) thermogenesis and subsequent energy expenditure in mice in response to GLP-1 analogs, but this potential mechanism remains largely unexplored in humans.

I will present data derived from a randomized controlled trial in individuals with obesity and obstructive sleep apnea (OSA) that supports a role for increased VAT metabolic activity in mediating the weight effects of GLP-1. GLP-1 analog treatment over 24 weeks increased VAT metabolic activity, and there was a correlation with the degree of weight loss. This supports the hypothesis that increased energy expenditure contributes to the weight-loss effects of GLP-1 analog therapy. Finally, the emerging role for GLP-1 in neurobiology will be discussed based on some patient responses I have seen and some retrospective review of US databases.

Biography

Qualified from University College Dublin in 1989 and then moved to Hammersmith Hospital in London. Completed research with Professor Sir Stephen Bloom on how the brain controls appetite. Moved to current position in 1999 establishing two hospital-

based multidisciplinary treatment teams for the management of adult obesity and gender dysphoria. Is a member of the Department of Health Policy group on obesity and chaired a group carrying out a health impact assessment on the potential benefits and harms of a tax on sugar sweetened drinks. Has presented the EU Ministers for Health and the Director General of the WHO on the importance of prevention of childhood obesity. Has specific research interests in obesity and gender and has published over 250 research papers. He is currently the national lead for management of obesity with the Health Service Executive.

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UCD DUBLIN

Assoc. Prof. Carmel Hensey (UCD SBBS, Head of Pharmacology)

Title: **From bench to brain: Enhancing pharmacology education through active engagement**

Abstract:

This presentation will showcase the development of innovative teaching practice in the Pharmacology degree at University College Dublin. With a focus on actively engaging students in both the classroom and laboratory settings, we aim to deliver elements of the pharmacology curriculum in a dynamic and interactive way. Strategies to promote active learning including infographic design, poster presentations, the design of preclinical testing programmes and an array of research-based activities will be presented. In addition, we will address the challenges encountered when integrating new content and assessment methods.

Biography:

Associate Professor Carmel Hensey BSc, PhD. joined UCD as a lecturer in Pharmacology in 1999 and is the current Head of Subject for Pharmacology (Sept. 2022- to date). One primary area of interest is pharmacology education, contributing to multiple degree programmes in Science, Medicine and Nursing.

As former Vice-Principal for Teaching and Learning in the College of Science and Fellow in Teaching and Academic Development she is passionate about teaching and has an interest in curriculum design, student engagement, assessment strategies and developing metacognitive skills in the virtual learning environment. Her scientific research investigates aspects of reproduction and early development using frog oocytes and embryos as a model system, with a particular focus on cell cycle, apoptosis and the DNA damage response.

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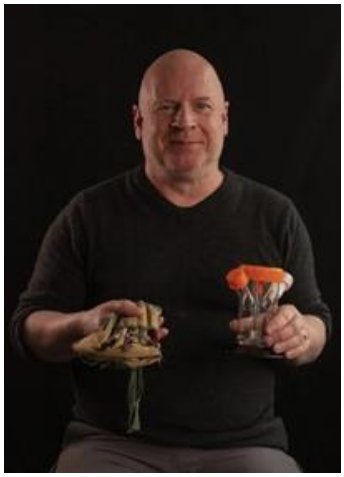
In Loving Memory of the Late Professor John Crean



Professor John Crean was a distinguished pharmacologist, researcher, and mentor whose career at University College Dublin left a lasting legacy in the fields of diabetic kidney disease, tissue engineering, and regenerative medicine. Originally from Dublin, John spent his early years in Cootehill, Co. Cavan, before returning to the capital to pursue his academic career. At UCD, he served as Associate Professor of Pharmacology in the School of Biomolecular and Biomedical Science and led a research team within the Diabetic Complications Research Centre, at the Conway Institute.

An international expert in pathogenic signalling in diabetic kidney disease, Associate Professor Crean made major contributions to understanding the interplay between pathogenic growth factors and the altered chromatin landscape of diabetes. His pioneering work used iPSC-derived kidney organoid models to study cell fate transitions and explore new strategies for disease repair. Deeply interested in stem-cell technology, his group successfully developed “mini-kidneys” in the lab, shedding light on how diabetes leads to kidney damage and paving the way for potential regenerative therapies.

Throughout his career, Professor Crean secured over €3.5 million in research funding, supervised 18 PhD students, and published more than 100 papers in leading international journals. His innovative work in tissue engineering led to the establishment of NGENKID Ltd., a company developing workflows to generate autologous stem cells and kidney organoids for patients with Autosomal Dominant Polycystic Kidney Disease.

Professor Crean will be remembered not only for his scientific excellence and innovation but also for his fascinating mentorship, smart curiosity, magnetic extravagance, and profound commitment to improving the lives of patients through research. His contributions continue to inspire colleagues and students alike, leaving a profound legacy in biomedical science and pharmacology.

Dr Jessica Davis, former postdoc of the Crean group and current programme manager at NGENKID Ltd., will kindly deliver the memorial lecture entitled: “Prof. John Crean. Pioneering the Future of Tissue Engineering and Regenerative Medicine: A Lasting Legacy”. The lecture will be introduced by Prof. Keith Murphy, SBBS Pharmacology.

Abstracts

Oral Presentations

A. Dr Kim Zitzmann (University College Dublin)
"Investigating the SRF signalling network as a druggable target in advanced prostate cancer"

B. Dr Sakshi Hans (Trinity College Dublin)
"Investigating the atherosclerotic immune-modulatory potential of the multi-mineral supplement Aquamin"

C. Dr Jayce O'Shields (Trinity College Dublin)
"Responses to medication and lifestyle questionnaires in a weight loss clinical trial for patients with resistant hypertension: an initial report"

D. Dr Mary Enright (University Hospital Limerick)
"Stroke alert, but not a stroke: Unmasking mexiletine neurotoxicity"

E. Ms Ashley Ajie (Royal College of Surgeons, Ireland)
"Game-based learning as a scalable solution for modern pharmacy education"

F. Dr Gerardene Meade-Murphy (University College Cork)
"A pedagogical approach: Online interactive learning in the core principles of pharmacology"

G. Dr Rachel Lamerton (Royal College of Surgeons, Ireland) – *Unable to attend*
"The infectious endocarditis-causing bacterium Streptococcus oralis triggers platelet aggregation via the sialic acid-binding adhesin Fap1, human antibodies, and the platelet receptor GPIIb/IIa"

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Poster Presentations

Poster presenters Rules:

- **Even Numbers** to present during first session (11:00 – 11:25 am) *
- **Odd Numbers** to hang posters during second session (15:35 – 16:00 pm)

Please remove your posters after your moderated session to allow people to hang posters for the next session

Everyone must remove their posters by the end of the day!

Poster Numbering List

IAP01 Tala Abdullatif (University Hospital Limerick)

IAP02 Rasha Alshaikh (University College Cork)

IAP03 Muhammad Arsalan (University Hospital Limerick)

IAP04 Szilárd-Krisztián Belényesi (Trinity College Dublin)

IAP05 Anchalin Bussayajirapong (University Hospital Limerick)

IAP06 Jay Campbell (University College Dublin)

IAP07 Niamh Clarke (University College Dublin)

IAP08 Luke Conroy (University College Dublin)

IAP09 Jessica Davis (University College Dublin)

IAP10 Stephen Fitzsimons (University College Dublin)

IAP11 Darren Freyne (Dublin City University)

IAP12 Donatas Galickas (University Hospital Limerick)

IAP13 Sauranil Guha (University College Cork)

IAP14 Helen Ye Rim Huang (RCSI/University Hospital Limerick)

IAP15 Eva Sophia Kalus (University College Cork)

IAP16 Liam Kelly (University Hospital Limerick)

IAP17 Roisin Kelly-Laubscher (University College Cork)

IAP18 Ciarán Kennedy (University College Dublin)

IAP19 Andrea Kwakowsky (University of Galway)

IAP20 Benjamin Leahy (University Hospital Limerick)

IAP21 Jiuang li (University of Galway)

IAP22 Orlaith Magnier (University Hospital Limerick)

IAP23 Shalen Maharaj (University Hospital Limerick)

IAP24 Konstantinos Matheoudakis (University College Dublin)

IAP25 Oran McNamara (University of Galway)

IAP26 Braden Millar (University College Dublin)

IAP27 Marwa Mustaf (University Hospital Limerick)

IAP28 Seema Nathwani (University College Dublin)

IAP29 Nouman Niaz (University Hospital Limerick)

IAP30 Eilidh O'Connor (University College Dublin)

IAP31 Francis N. Odinukaeze (University of Galway)

IAP32 Anisha Pattanayak (Trinity College Dublin)

IAP33 Louise Rabbitt (University of Galway)

IAP34 Domhnall Roe (Trinity College Dublin)

IAP35 Domhnall Roe (Trinity College Dublin)

IAP36 Anastasia Saleh (Trinity College Dublin)

IAP37 Aditi Sooknarine-Rajpatty (University Hospital Limerick)

IAP38 Aditi Sooknarine-Rajpatty (University Hospital Limerick)

IAP39 Huiyuan Yang (University College Cork)

IAP40 Wuyun Zhu (Royal College of Surgeons Ireland)

Irish Association of Pharmacologists

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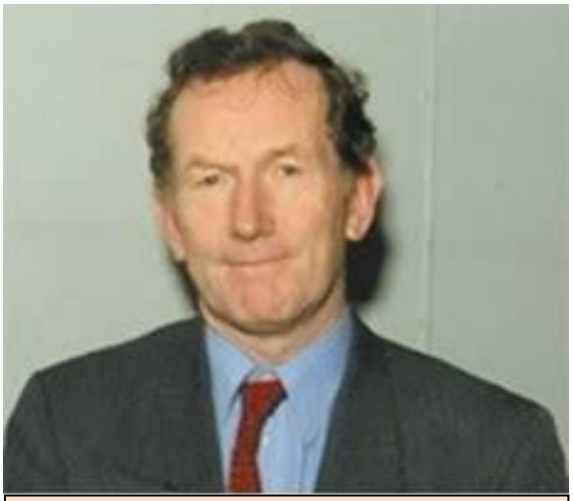
John Feely Medal

History

Professor John Feely was a leading Irish pharmacologist and international authority on hypertension and clinical therapeutics. A graduate of University College Dublin, he became Professor of Pharmacology and Therapeutics at Trinity College Dublin in 1984, where he built a department of global reputation. He published over 300 research papers, edited New Drugs (BMJ Publishing, 1991), and helped establish the National Medicines Information Centre and National Pharmacoeconomics Unit. As Chairman of the Irish Blood Pressure Council and Registrar of the RCPI, he advanced education, research, and patient care-leaving a lasting legacy in Irish medicine.



Prof. John Feely (1947 – 2009)
MB, BCh, NUI, BSc (1971), MRCPI (1974), MD (1979), FRCPI (1984), FRCP Edin (1992), FRCP (2000)

Significance

This medal is awarded annually at the IAP annual meeting to a single individual chosen by a panel of their peers. It is awarded in memory of the contribution made by Professor Feely to the discipline of pharmacology. This medal will be awarded in recognition of the role of the successful candidate in the delivery of the service to the integrated disciplines of clinical therapeutics or basic pharmacology through their teaching, research, innovation, mentorship and or delivery of exemplary clinical care to patients. Candidates will normally be active on the island or Ireland, however, in exceptional circumstances, international candidates with strong links to the island of Ireland may be considered. The successful candidate will deliver a keynote lecture titled: “The John Feely Lecture”.

We are thankful to Prof. Martina Hennessy (Trinity College Dublin), who proudly issued and carved the John Feely medal also this year.

Irish Association of Pharmacologists

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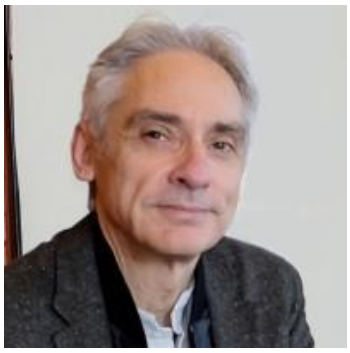
UCD DUBLIN

2025 Awardee



Professor Christian Waeber (UCC, Head of Pharmacology & Therapeutics)

Lecture Title: *From serendipity to translation: A meandering journey through pharmacology and drug discovery*

Abstract:

Drug discovery often meanders between intention and coincidence. An early ambition to study migraine collapsed for lack of a PhD position, only for my PhD work to eventually illuminate the pharmacology of triptans, and much later, the ditans as better antimigraine drugs. Each detour reinforced a preference for research that impact positively on patients: making antimigraine drugs safer through receptor selectivity, identifying new therapeutic targets, designing novel ocular drug-delivery systems, and improving how research itself is done. Later work on fingolimod in stroke, and more recently the creation of the Munster Integrated Stroke Research and Education Catalyst, reflect a continuing effort to bridge experimental pharmacology research and clinical outcomes. My answer to the reproducibility crisis in experimental pharmacology and the failed translation in many therapeutic areas has been on ensuring that animal experiments produce reliable, translatable data with real potential for patient benefit.

Taken together, these experiences suggest that while a scientific career rarely follows a straight path, allowing curiosity and rigour to guide the course can still lead to meaningful discoveries.

Short bio:

Professor Christian Waeber joined University College Cork in 2013, where he is Professor and Head of Pharmacology & Therapeutics with a joint appointment in the School of Pharmacy. His career spans industry and academia, beginning at Sandoz (now Novartis), followed by research at CNRS-INSERM in Montpellier and two decades at Harvard Medical School. His work focuses on translational pharmacology, from characterising serotonin receptors that led to the triptan and ditan classes of antimigraine drugs, to studying sphingosine-1-phosphate (S1P) receptor biology in stroke and developing ocular and bone-regenerative drug-delivery systems. He founded the Munster Integrated Stroke Research and Education Catalyst (MISREC) and chairs UCC's Animal Experimentation Ethics Committee (AEEC), serving also as a member of Ireland's National Committee for the Protection of Animals used for Scientific Purposes (NCPA).

Partners

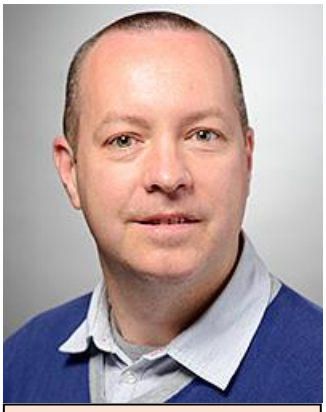
British pharmacological Society (BPS)

Prof. Mark Evans (BPS Vice President)

Professor Evans, VP of Committee Chairs at BPS, developed an interest in Pharmacology when working as a research technician at Beecham Pharmaceuticals. He studied Pharmacology at the University of Sunderland and graduated with a BSc Honours 1st Class. He then studied for a Ph.D. under the supervision of Professor B. L. Ginsborg at Edinburgh's Department of Pharmacology, which was awarded in 1990. Postdoctoral work followed, notably with Professor R. J. Martin at Edinburgh, which preceded a move to London. There, Professor Evans developed his interest in the regulation by oxygen of blood flow within the pulmonary vasculature. This led to a Wellcome Trust Research Career Development Fellowship at the Department of Pharmacology, University of Oxford. In 2001 he was appointed to a Lectureship at the University of St Andrews and was promoted to Reader in 2005. Professor Evans was then appointed Chair of Cellular Pharmacology at the University of Edinburgh in 2009, following a brief sabbatical at Norwich. His current research focuses on the role of AMPK in the regulation of breathing and oxygen supply, and the coordination of intracellular signalling by nanojunctions of the sarcoplasmic reticulum.

We are thankful to BPS, who proudly sponsored the event by issuing the communication awards (Best Oral and Poster Presentations) and four Travel Bursaries

UCD School of Biomolecular and Biomedical Science (UCD SBBS)



Prof. Cormac Murphy (SBBS Head of School)

"SBBS – Guiding the next generation of scientists: Our mission is to deliver teaching and research excellence that fosters discovery and learning through collaboration within UCD, nationally and internationally."

We are thankful to the UCD School of Biomolecular and Biomedical Science, who proudly sponsored the event by arranging the catering and the venue for the event.

Book of Abstracts

Selected Oral Presentations

Ashley Ajie (RCSI)

"Game-based Learning as a Scalable Solution for Modern Pharmacy Education"

Introduction

Changing classroom dynamics, expanding class sizes, increasingly diverse student populations and exposure to technology are reshaping how students learn. To meet these challenges, educators must adapt teaching approaches to better promote active engagement, foster inclusivity and enhance long-term retention. Game-based learning (GBL) has demonstrated promising results for increased student motivation and engagement. However, institutions in countries such as China, which prioritise traditional didactic learning have been slower to incorporate these new teaching practices. This study explored student perceptions of GBL within Chinese pharmacy education.

Methodology

Following ethical approval, first year pharmacy students (n=80) in a Chinese university were invited to participate in this study. A game-based activity designed as an escape room explored how students engaged with this format of teaching. This activity required teamwork and application of pharmaceutical knowledge to solve clues. Student engagement and enjoyment of this activity was assessed via a pseudonymised survey.

Results

Forty-eight percent of the class (n=38) participated in the post-activity survey. The activity was rated as 4.78/5. Students surveyed reported high levels of engagement and motivation to participate in the activity. All students indicated a preference for GBL when compared to traditional teaching methods.

Conclusion

This activity was designed to address communication and teamwork skills as well as pharmacological and pharmaceutical skills. Students highlighted a sense of autonomy which contributed to their enjoyment of the activity. Overall, the findings highlight the significant potential of GBL to increase student engagement and motivation, key elements in learning and retention.

Mary Enright (University Hospital Limerick)

"Stroke Alert, But Not a Stroke: Unmasking Mexiletine Neurotoxicity"

Up to 30% of stroke presentations may represent stroke mimics (1). Drug-induced neurological deficits are rare, but important to recognise. Mexiletine, a class 1B antiarrhythmic, is used in the management of ventricular arrhythmias. Mexiletine causes neurological symptoms by excessive neuronal sodium channel blockade.

We describe the case of a 77-year-old-man who had a background history of ventricular tachycardia with ICD in situ and multiple previous shocks, previous MI, and autoimmune thyroid disease. His arrhythmia had been unsuccessfully treated with beta-blockers, and amiodarone had not been trialled due to his thyroid disease. He presented to the emergency department with symptoms of dizziness, blurred vision, paraesthesia, and ataxia, which occurred while lying in bed and sitting in a chair. He underwent extensive investigation including MRI brain, tilt-table test, and vestibular testing by specialist physiotherapist. He had no further evidence of cerebellar signs or peripheral vestibular hypofunction. His ICD was interrogated with no cardiac cause found for his symptoms. Further evaluation revealed that mexiletine had recently been started by his cardiologist for VT. Mexiletine dose was reduced from 200mg TDS to 150mg TDS, but unfortunately symptoms recurred. He underwent ablation for his arrythmia, and mexiletine was discontinued.

This case shows the challenges of prescribing in complex patients with multiple comorbidities and highlights the importance of thorough drug history in evaluating neurological deficits. In instances where drug therapy is ineffective or poorly tolerated, procedures such as ablation should be considered as a valuable therapeutic alternative.

Sakshi Hans (Trinity College Dublin)

"Investigating the atherosclerotic immune-modulatory potential of the multi-mineral supplement Aquamin"

Aquamin (Aq) is a multi-mineral supplement derived from *Lithothamnion glaciale* with reported immune modulating properties [1,2]. Immune dysregulation and chronic inflammation underlie the pathology of cardiovascular disease states including atherosclerosis [3]. This study investigates the immune-modulating effects of Aquamin and its impact on cellular behaviours in atherogenesis.

Inflammatory markers (TNF-α and IL-6) were assessed in human peripheral blood mononuclear cells (hPBMCs) and murine bone marrow-derived macrophages (mBMDMs) using ELISA and RT-PCR following toll-like receptor stimulation and Aq treatment. Primary Human Aortic Endothelial Cells (HAECs) were tested for changes in adhesion (ICAM-1, VCAM-1, E-selectin) and chemotaxis (CXCL10) markers. Migration was evaluated in Human Aortic Smooth Muscle Cells (HASMCs) using a scratch assay and in THP-1 monocytes using platelet-derived growth factor (PDGF-BB)- driven transwell assay. THP-1 proliferation was measured by CFSE and BrdU staining. Oxidized LDL (oxLDL) uptake was assessed in THP-1–derived macrophages. Aq (0.5-2 mg/mL) dose-dependently reduced LPS-induced production of inflammatory mediators including TNF-α and IL-6 in hPBMCs and mBMDMs. In LPS-activated HAECs, CXCL10 chemotaxis marker, ICAM-1 and VCAM-1 adhesion markers were significantly reduced with Aq treatment.

Aq also reduced migration in HASMCs and monocytes without affecting proliferation. Furthermore, Aq decreased oxLDL uptake in macrophages. Aquamin thus attenuates inflammatory responses in macrophage immune cells, reduces endothelial cell adhesion and chemotaxis markers, inhibits smooth muscle cell and monocyte migration, and limits macrophage oxLDL uptake- all key processes underlying atherogenesis. These findings suggest Aq may exert protective effects against atherosclerotic disease progression. Further studies are warranted to fully characterize its therapeutic potential.

Rachel Lamerton (RCSI) – (unable to attend)

"The infectious endocarditis-causing bacterium Streptococcus oralis triggers platelet aggregation via the sialic acid-binding adhesin Fap1, human antibodies, and the platelet receptor GPIIb/IIa"

Background: Infective endocarditis is a devastating infection of the heart's endocardial surface, carrying a one-year mortality rate nearing 30% (1). Oral streptococci, particularly *Streptococcus oralis,* are frequent culprits, entering the bloodstream after dental procedures, trauma, or poor oral hygiene. Once in circulation, *S. oralis* adheres to damaged or prosthetic heart valves triggering formation of vegetations - dense aggregates of platelets, fibrin, and bacteria - that shield the pathogen from immune clearance and antibiotics. These protective niches enable persistent infection and complicate treatment, highlighting the need to understand early platelet-bacterial interactions.

Methods: Platelet aggregation was assessed using light transmission aggregometry in platelet-rich plasma (PRP) and washed platelets from healthy donors in response to *S. oralis* ATCC 10557.

Results: *S. oralis* triggered platelet aggregation in PRP after a 2.9±1.0-minute lag phase. Deletion or point mutation of the *S. oralis* sialic acid binding protein Fap1 (2) abolished aggregation. Blocking the highly sialylated platelet receptor GP1ba eliminated aggregation, suggesting critical Fap1–GPIba interaction. However, this interaction alone was insufficient to trigger aggregation in washed platelets, suggesting an additional plasma-derived cofactor. Antibody involvement was implicated by inhibitor studies of FcγRIIA in PRP, and reconstitution of washed platelets with pooled human IgG confirmed this by fully restoring aggregation. Importantly, IgG failed to rescue aggregation in both Fap1 mutant strains.

Conclusions: We propose that *S. oralis*-induced platelet aggregation requires Fap1–GPIb binding, IgG–FcγRIIa engagement, and GPIIb/IIIa signalling, with disruption of any step blocking aggregation. These findings reveal key mechanisms in platelet–bacteria interactions and suggest targets for early intervention in infective endocarditis.

Gerardene Meade-Murphy (University College Cork)

"A Pedagogical Approach: Online Interactive Learning in the Core Principles of Pharmacology"

Our online interactive learning in pharmacology was developed to address specific learning needs of professional and undergraduate paramedic students (BSc. Paramedic Studies), while facing challenges of balancing academic study with clinical duties.

Our pedagogical strategy was to develop accessible, engaging, online-interactive learning on core pharmacology principles, while ensuring academic standards in learning, support, and assessment.[1]

We applied the "ADDIE" model to analyse, design, develop, implement, and evaluate course material.[2,3] Due to lack of suitable pharmacology resources for student learning needs, all content was created in-house. Continuous refinement to course material has produced a 20-unit online resource, each learning-unit featuring interactive chapter and graded end-of-chapter assessment. Additional assessment includes peer-discussions, individual written-assignments. Interactive learning: Using Articulate-Rise360 software, each learning-unit was organised with hierarchy and interactivity, structuring content to enhance student learning. For each chapter, course material is packaged into bite-sized learning blocks, accessible through interactive scrolling and navigation. Supporting components include interactive graphics, learning activities, knowledge-checks (all in-house). Modes of interactivity include accordion or tab-navigation, click-and-reveal, graphic "hotspot labels", storyline-figures.

To support self-directed learning, course material is delivered via adaptive release, requiring sequential progression through chapter-and-assessment before advancing to the next learning-unit, with all units rolled-out chronologically. With its advancement, the School of Pharmacy has adapted the online-learning material to support its MSc. in Pharmaceutical Technology-and-Quality systems (MSCPTQ). Furthermore, our students typically provide very positive evaluation-and-feedback. We believe our innovative learning material facilitates and enhances student understanding of pharmacology, fosters student engagement, peer communication, reflective learning, writing skills and academic integrity.

Jayce O'Shields (Trinity College Dublin)

"Responses To Medication and Lifestyle Questionnaires In A Weight Loss Clinical Trial For Patients With Resistant Hypertension: An Initial Report"

Semaglutide has been shown to improve blood pressure (1). In preparation for a randomized trial assessing semaglutide in patients with obesity and resistant hypertension (SUPPORT), we analysed pre-randomization survey data to characterize baseline cohort characteristics.

Consented participants completed four instruments: EQ-5D-3L, Beliefs about Medicines Questionnaire (BMQ), Hill-Bone Compliance Scale (HB-HBP), and the Best Health Program Questionnaire (BHQ), assessing quality-of-life, health beliefs, medication adherence, and lifestyle behaviours. Likert scales were numerically coded; some domain scores were summated.

Seventeen participants returned surveys (65% male, median age 63 [48 – 65], 88% White Irish). Most reported no issues in health-related-quality-of-life. BMQ responses indicated moderate necessity and concern regarding hypertension medications without strong beliefs in general harm or overuse. HB-HBP results suggested generally high medication adherence. BHQ findings showed most participants consumed moderate fruits and vegetables and avoided sugary drinks, though nearly half ate convenience foods weekly and over half used screens while eating. Sedentary behaviours were prevalent; walking and housework were the mostly reported physical activities. Most participants reported positive mood and adequate sleep. Interpretation of some HB-HBP items could vary, highlighting potential limitations.

In this pre-intervention cohort, quality-of-life, adherence, and health beliefs were favourable, while lifestyle behaviours varied. These findings contextualize post-intervention interpretations, including the influences of supplemental lifestyle interventions on secondary endpoints alongside semaglutide's primary effects on blood pressure, weight, and appetite.

Kim Zitzmann (University College Dublin)

"Investigating the SRF signalling network as a druggable target in advanced prostate cancer"

i) The problem being addressed in the study

Prostate cancer (PCa) is the most common invasive cancer in men worldwide. Current treatments target the Androgen Receptor (AR) through androgen deprivation therapies (ADT), such as enzalutamide. While initially effective, resistance to these therapies inevitably develops. This highlights the need for alternative strategies that disrupt AR signalling without directly targeting AR, such as inhibiting AR co-regulators. One such candidate is the Serum Response Factor (SRF), previously implicated in PCa progression. Previous studies in our laboratory identified shared interactors between AR and SRF, including HSP70, HSP90, and members of the PI3K/Akt pathway (1). This study investigates AR-SRF signalling crosstalk and explores whether targeting common co-regulators offers novel therapeutic strategies in advanced PCa.

ii) How the study was performed

Proliferation and viability were assessed via MTT assays and IncuCyte analysis using inhibitors targeting SRF (CCG1423, Lestaurtinib), AR (Enzalutamide, EPI7170), HSP70/90 (VER-15508, JG-98, Ganetespib), and the PI3K/Akt pathway (Ipatasertib, Alpelesib).

iii) The salient results

Inhibiting AR, SRF, and shared co-factors, both individually and in combination, reduced PCa cell viability and proliferation. Notably, several drug combinations demonstrated synergy at IC 30 and IC 10 concentrations, including Lestaurtinib + Ipatasertib, CCG1423 + EPI7170, EPI7170 + Ipatasertib, and EPI7170 + Lestaurtinib. Proteomics and phospho-proteomics analysis is currently ongoing.

iv) What the authors conclude from the results

These findings suggest that targeting the AR-SRF signalling network is a promising strategy for overcoming ADT resistance in PCa. Ongoing proteomic analyses aim to uncover deeper insights into resistance mechanisms and identify additional therapeutic targets.

Selected Poster Presentations

IAP01 Tala Abdullatif (University Hospital Limerick)

"Prevalence of Potentially Inappropriate Prescriptions in Older Adults with Cardiovascular Comorbidities Attending the Emergency Department in a University Teaching Hospital: A Secondary Analysis"

Potentially inappropriate prescribing (PIP) contributes to adverse health outcomes in older adults, particularly those with cardiovascular comorbidities, who often experience polypharmacy and frailty (1). This study aimed to determine the prevalence of PIP and associated risk factors in older adults with cardiovascular conditions attending the emergency department (ED) of a university teaching hospital in Ireland. A secondary analysis was conducted using merged datasets from two cohort studies (SOAED and SOLAR), involving patients aged ≥65 years (2,3). The STOPP version 3 criteria were applied to identify PIP among 251 patients with cardiovascular comorbidities using the Charleson Comorbidity Index. Descriptive and inferential statistical analyses, including multivariable logistic regression, were performed using SPSS version 29. The prevalence of PIP was 32.6%. The most common PIPs were dual antithrombotic therapy (10.2%), lack of laxatives with regular opioids (6.9%), and anticholinergic use in dementia (4.9%). Frailty, assessed using the ISAR score, was significantly associated with PIP (OR 2.12, 95% CI 1.00–4.50, p=0.049). Other factors, such as age ≥75 years, male gender, and polypharmacy, were associated with increased odds of PIP but did not reach statistical significance. Approximately, one-third of older ED patients with cardiovascular comorbidities had at least one PIP. Frailty was significant predictor of PIP, underscoring the importance of routine frailty screening and medication review in acute care settings. These findings highlight the need for targeted interventions to optimise prescribing, reducing adverse outcomes in this high-risk population.

IAP02 Rasha Alshaikh (University College Cork)

"Siponimod as a Novel Therapy for Ocular Neovascular Diseases: Mechanistic Characterisation and Development of a Sustained Ocular Delivery System"

Neovascular ocular diseases remain a leading cause of preventable blindness, yet current therapies are limited by their biological nature, high cost, short half-life, treatment resistance, and poor patient compliance due to frequent intravitreal injections.

This study investigated siponimod, an FDA-approved sphingosine-1-phosphate receptor (S1PR) modulator, as a potential inhibitor of ocular angiogenesis, and developed an injectable sustained delivery system to address delivery challenges. Siponimod effects on human retinal microvascular endothelial cell (HRMEC) monolayer integrity and barrier function were assessed using transendothelial electrical resistance (TEER) and fluorescein isothiocyanate-dextran (FITC-DEX) permeability assays, while in vivo efficacy was evaluated in a suture-induced corneal neovascularisation model in albino rabbits. Finally, Electrospinning was used to develop an injectable poly (lactic-co-glycolic acid) (PLGA) to provide sustained drug release and minimise injection frequency.

In vitro, siponimod enhanced HRMEC barrier integrity and protected against stress-induced junctional disruption. These actions are mainly mediated by S1PR1 modulation. In vivo, siponimod prevented the progression of suture-induced corneal neovascularisation in albino rabbits, supporting its antiangiogenic potential. Building on this therapeutic promise, a novel electrospun PLGA implant was engineered to provide sustained, intravitreal siponimod delivery. Optimised low-porosity fibres stabilised siponimod in an amorphous solid dispersion, enabling controlled Higuchi-type release over 90 days. Released siponimod retained biological activity, inhibiting retinal endothelial migration without cytotoxicity, while the implant demonstrated stability under accelerated conditions.

In conclusion, our studies support the therapeutic potential of siponimod in ocular neovascular diseases and, when combined with sustained-release intravitreal delivery, represent a promising small-molecule alternative to biologics in the management of these diseases.

IAP03 Muhammad Arsalan (University Hospital Limerick)

"The critical care management of patients with severe behavioural disorders"

Problem:

The management of critically ill adults with severe behavioural disorders poses significant challenges. These patients are at an increased risk of aggressive behaviour and unintentional self-harm through forceful removal of indwelling catheters, arterial lines and central venous catheters, especially during sedation breaks and extubation. These behaviours complicate airway management and recovery after prolonged ventilation in intensive care unit (ICU).

Methods:

We report the management of a critically ill adult with a background of severe autism spectrum disorder, intellectual disabilities and aggressive behaviours who was admitted to ICU with seizures secondary to hyponatraemia. He was intubated for airway protection and control of agitation. His stay was complicated by *Pseudomonas* ventilator-associated pneumonia. Our primary concern was his safe extubation and management of aggressive behaviours complicated by his sensory intolerance of anything touching his skin – central venous catheter, arterial line, nasogastric tube and urinary catheter had to removed prior to extubation. Sedation was maintained with low dose propofol infusion (50 mg/hr), dexmedetomidine 1.4 mg/kg/hr as well as oral diazepam 10 mg TDS and olanzapine 10 mg BD. Given concerns for QTc prolongation and neuroleptic malignant syndrome, the patient was loaded with a single dose of chloral hydrate 1g and promethazine 50mg. All indwelling devices were removed, and the patient extubated successfully with the assistance of his family and residential carers.

Results:

The patient was successfully extubated without significant agitation or complications. Involvement of familiar carers provided emotional and behavioural support, facilitated a calm environment and prevented previously experienced aggressive episodes such as pulling intravenous lines and catheters. Sedation was carefully weaned and switched to all per oral medication and neurological status improves progressively. A weaning plan for all medication on discharge was implemented.

IAP04 Szilárd-Krisztián Belényesi (Trinity College Dublin)

"Extracellular vesicles in plasma of patients with epithelial ovarian cancer are larger EVs diagnostic, and linked to advanced disease"

Introduction: Epithelial ovarian cancer (EOC) is the most common type of ovarian cancer. As its symptoms can be non-specific, the disease often goes undiagnosed until it is too late. Biomarkers for diagnosing EOC are predicting disease progression urgently needed. Thus, we aimed to investigate if blood-based extracellular vesicles (EVs) have relevance as minimally invasive biomarkers for this purpose.

Methods: Plasma samples obtained prior to medical intervention from patients with benign (*n=20*) and malignant (*n=42*) ovarian tumours were processed by size-exclusion chromatography to collect EVs, then characterised by immunoblotting, TEM, NTA, and flow cytometry.

Results: EVs from women with benign ovarian lesions, compared to malignant tumours, were significantly smaller ($p<0.0001$), but more abundant ($p<0.0001$). When the proportion of EVs in two size ranges was assessed, samples from patients with benign lesions showed a predominance of small EVs (<120 nm; 77.7%); those from malignant tumours were enriched in larger EVs (120–550 nm; 71.5%). Interestingly, when comparing early-stage (stage I+II, *n=20*) with advanced-stage (stage III+IV, *n=22*) disease, total particle amounts did not differ. However, early-stage patients (stage I+II, *n=20*) had proportionally more smaller particles (*p=0.026*), whereas advanced-stage patients (stage III+IV, *n=22*) had significantly higher proportions (*p=0.045*) and absolute amounts (*p=0.048*) of larger EVs.

Conclusion: Our research indicates differences in the sizes of the most abundant EVs produced by malignant tumours compared to benign lesions and suggests that the amounts of blood-based larger particles correlate with the presence of cancer and more advanced disease.

Acknowledgements: HEA's North-South funding of the All-Ireland Cancer Liquid Biopsies (CLuB).

IAP05 Anchalin Bussayajirapong (University Hospital Limerick)

"A Prospective Audit Against the 4M Quality Improvement Tool in Medical Patients Age 65 and Over Admitted in Model 4 Hospital in Mid-West Ireland"

Background:

The percentage of patients aged 65 and over requiring admission in acute hospital have grown over the years. Age-Friendly Health Systems use four evidence-based components in older adults to improve care quality and outcomes, termed "4Ms": What Matters, Medication, Mentation, and Mobility. Polypharmacy is associated with significant increase in mortality (1).

Aim:

This audit aims to investigate compliance of 4M by medical teams in the management of patients age 65 and over in a model 4 hospital in Mid-West of Ireland.

Methods:

Data was collected by advanced nurse practitioner and medical doctor involved in managing older patients. A checklist of points relevant to each criteria of 4M's were created according to the Age-Friendly Health Systems. This tool was used to collect data from patient's medical files, medication Kardex and nursing notes in 50 medical patients age 65 and over.

Results:

Analysis of data found that 64% (32/50) of patients had all 4 criteria met by medical teams. Across all 4 criteria that were not met, 'Medication' and 'What Matters' were the two most common criteria. 16% (8/50) did not meet the 'Medication' criteria, specifically polypharmacy, dose adjustment and documentation of high-risk medication use.

Discussion:

Results reveal that the majority of medical teams managing patients aged 65 and over were compliant with the 4Ms. However, a significant number did not have evidence of review of medications, which contributes to prolonged hospitalisation, complications and increased mortality. To improve the quality of care and outcome amongst these patients, further education with regards to the evidence-based 4Ms is required.

IAP06 Jay Campbell (University College Dublin)

"Serum Response Factor in Triple-Negative Breast Cancer: A Proteomic and Phosphoproteomic Study"

Breast cancer is the leading cancer diagnosis in women. Triple-negative breast cancer (TNBC) accounts for 15-25% of cases and is associated with poor prognosis. Studies in prostate cancer have shown a correlation between increased expression of Serum Response Factor (SRF), a transcription factor involved in many cancer types, and poor prognosis. Preliminary data by our research group show that SRF expression is upregulated in TNBC and is associated with increased disease recurrence. The aim of this research was to determine whether SRF is a viable therapeutic target in TNBC.

To elucidate the molecular mechanisms of SRF in TNBC, we conducted proteomic analysis in MDA-MB-231 treated with lestaurtinib for 48 hours, using mass spectrometry. Out of approximately 5700 hits, 710 proteins were differentially expressed with at least a 15%-fold change using the permutation-based false discovery rate statistical parameter. Ingenuity Pathway Analysis showed the pathways most significantly affected are those involved in the cell cycle, mitosis, and RHO GTPase signalling.

Additionally, we analysed phosphoproteomic changes by mass spectrometry following lestaurtinib treatment to further study the signalling pathways under SRF control. Phosphorylation data was normalised to the whole proteome, and 99 phosphosites across 88 proteins were found to be differentially phosphorylated at 48 hours post treatment. Ingenuity Pathway Analysis found that the pathways most significantly differentially phosphorylated were those involved in mitosis and RHO GTPase signalling.

In conclusion, we have demonstrated that SRF inhibition with lestaurtinib results in significant differential expression in both the whole- and phosphoproteome, and significantly effects signalling pathways involved in the cell cycle and mitosis.

IAP07 Niamh Clarke (University College Dublin)

"Exploring the neurotherapeutic potential of novel Schiff base compounds in vitro models of TLE"

The authors have opted out of publishing their content online

IAP08 Luke Conroy (University College Dublin)

"The Effect of Lipoxin-A4 & Liraglutide in a Glucose-Challenged Macrophage Model of Early Diabetes-Associated Atherosclerosis"

Diabetes affects >800 million people globally, and greatly enhances the risk of cardiovascular disease, including atherosclerosis: the current leading cause of death worldwide[1]. Diabetes promotes atherogenesis through several distinct mechanisms, including sustained hyperglycaemia and chronic inflammation[2,3].

Monocyte and macrophage cells are crucial players in diabetes-associated atherosclerosis (DAA). Circulating monocytes are transformed into macrophages by various inflammatory stimuli. Mature macrophages are responsible for scavenging pathogens and cell-debris. They can also engulf excess lipids, facilitating the development of fat-laden foam cells, which progress to form the fatty streak, a definitive hallmark of atherosclerosis. Using an *in vitro* diabetic macrophage-based model, mimicking early DAA, this study aimed to assess the therapeutic role of specialised pro-resolving mediators (SPMs) and GLP-1 receptor agonists. Specifically, we try to elucidate the therapeutic potential of *Lipoxin-A4* (*LXA4*) and *Liraglutide*, in the context of DAA. Hence, THP-1 monocytes are challenged with persistent high-glucose [25mM] to induce an inflammatory diabetic-macrophage. Subsequently, the inflammatory macrophage generated is treated with *LXA4* and/or *Liraglutide* before evaluating their effect on the macrophage inflammatory response using qPCR, ELISA, and migration assays. *LXA4* treatment alters expression of inflammatory markers, specifically upregulating *IL-1β* and *TRAIL*, and enhancing IL-1β and IL-18 secretion in macrophages. Moreover, Lipoxin attenuates monocyte and macrophage migration towards human plaque-conditioned-media in low-glucose.

Liraglutide also displays potent anti-inflammatory effects, reducing *MCP-1* and *TNF* expression, whilst mitigating IL-1β secretion. High-glucose alters macrophage phenotype and enhances inflammation in a diabetic-macrophage model. Combined treatment with *Lipoxin* and *Liraglutide* may help attenuate the inflammatory response in an *in-vitro* model of DAA.

IAP09 Jessica Davis (University College Dublin)

"Characterising the role of the master transcription factor, PRRX1, during the specification of neural cell fate"

Maternal addictive substance abuse has been estimated at around 5% with epidemiological studies suggesting as high as 750,000 cocaine exposed pregnancies every year. Foetal cocaine exposure causes abnormalities in brain development, which as later linked to development of anxiety, depression and cognitive dysfunction. Previous studies in rodent models have indicated that prenatal cocaine exposure effects the proliferation, differentiation and connectivity of neural cell types. Master transcription factors control cellular identity and act as master switches that determine cell type and function during development. PRRX1 is a transcription factor known for its regulatory functions in specifying cell fate during development and epithelial-to-mesenchymal transition (EMT). Genetic mutations in PRRX1 disrupts developmental patterning and impairs the maintenance of progenitor cell proliferation, profoundly affecting cranial suture biology. Recent studies have shown, addictive substances such as nicotine, inhibit PRRX1 in progenitor cells resulting in delayed development, yet the role of PRRX1 in neural cell fate specification is still not well understood. This study aimed to elucidate the role of PRRX1 in neural specification, utilising NTERA2 cells and iPSC-derived neuroectoderm as complementary models. The temporal expression patterns of PRRX1 expression was increased in the early stages of neuron differentiation in both NTERA2 and iPSC-derived neuroectoderm indicating its role in neural specification. PRRX1 expression was transcriptionally modulated during NTERA2 differentiation using a targeted CRISPR interference approach. Finally, treatment of organoids and NTERA2 cells with cocaine affected neurogenesis.

IAP10 Stephen Fitzsimons (University College Dublin)

"Extracellular vesicles derived from induced pluripotent stem cells mediate anti-inflammatory effects in primary human macrophages"

Extracellular vesicles derived from induced pluripotent stem cells (iPSC EVs) have immunoregulatory potential with the ability to alter the macrophage phenotype. Modulating the macrophage phenotype towards an anti-inflammatory, pro-resolving state may be beneficial in the treatment of chronic inflammatory diseases. The contents of iPSC EVs and their effects on macrophages are poorly understood. Here iPSC EVs were characterized and analysed by mass-spectrometry based proteomics and a targeted microRNA (miR) panel and their immunomodulatory effects on primary human macrophages were assessed.

Podocalyxin-like protein 1 (PODXL1) and Insulin (INS) were the most abundant proteins unique to the iPSC EVs while miR-302d-3p was the most abundant miR. Notably, thioredoxin- and peroxiredoxin-related proteins were detected. iPSC EVs increased the anti-inflammatory associated Mannose Receptor C-Type 1 (*MRC1*) and miR-21, while monocyte chemoattractant protein 1 (MCP-1) and IL-8 were decreased. Proteomics revealed that treated macrophages had decreased levels of chemoattractant proteins, Azurocidin 1 (AZU1), Growth Differentiation Factor 15 (GDF15), and Ribosomal Protein S19 (RPS19). Conditioned media from macrophages treated with iPSC EVs inhibited monocyte transmigration, a key component in the propagation of inflammation. This study provides insights into the protein and miR cargo of iPSC EVs and highlights their capacity to inhibit chemotactic proteins in macrophages.

IAP11 Darren Fayne (Dublin City University)

"Using tiered computational screening to discover small molecule inhibitors of the SARS-CoV-2 NSP3 protein Mac1 domain"

As new medications are used to treat COVID-19, many studies have reported that proteins such as spike, polymerase and proteases are prone to high levels of mutation that can create resistance to therapy over time. Thus, it becomes necessary to, not only target other viral proteins such as the non-structural proteins (NSP's), but to also target the most conserved residues of these proteins. A synergistic combination of bioinformatics, computer-aided drug-design and in-vitro studies can feed into better understanding of SARS-CoV-2 (SC-2) and therefore help in the development of small molecule inhibitors against the NSP's [1]. As part of our initial anti-viral work, a pharmacophore study on NSP15 found a hit molecule (INS316) that made interactions with Ser293, Lys344 and Leu345 residues [2] which are highly conserved across SC-2.

Our group was selected to enter an international challenge organized by CACHE to find inhibitors for the Mac1 domain of SC-2 NSP3. Our MSA alignment results of ~1 million NSP3 sequences indicated that the Mac1 domain is a highly conserved pocket that can be targeted for developing promising SC-2 inhibitors. We used a tiered screening workflow which included the use of volume/shape information of the binding pockets (fastROCS), use of in-house pharmacophore generation software (MoPBS [3]/MOE) and performed docking in the binding pocket (FRED) to rank compounds for subsequent clustering and to identify hits that bind to these conserved pockets. The primary experimental validation results provided by CACHE found that two of our predicted hits show activity in HTRF and SPR assays.

IAP12 Donatas Galickas (University Hospital Limerick)

"Management of Plasmodium vivax malaria in Pregnancy and Lactation"

We present a case of a 22-year-old female at 15 weeks' gestation who attended a maternity hospital with a one-week history of malaise and vomiting. Her pregnancy had been uncomplicated, with no past medical history or regular medications of note. She was from Afghanistan and had moved to Ireland 9 months earlier.

Investigations revealed a microcytic anaemia and an incidental finding of malarial parasites on blood film, later confirmed as *P. vivax*.

She had no features of severe malaria and was tolerating oral intake. Treatment was started with Artemether-Lumefantrine (Riamet) 4 tablets at 0, 8, 24, 34, 48 and 60 hours. As primaquine was required for hypnozoite eradication but contraindicated in pregnancy, she was switched to Chloroquine base 310mg weekly. Weekly clinic follow up was uneventful apart from mild pruritus.

The patient delivered healthy male infant at 40+2 weeks and planned to breastfeed, remaining on chloroquine suppression postnatally.

As our patient resided in direct provision, the risks of primaquine treatment during breastfeeding were weighed against potential loss to follow-up and relapse without hypnozoite eradication.

Qualitative Maternal G6PD testing was negative with normal levels reported. Without quantitative testing, heterozygote carrier status could not be excluded in the patient and therefore G6PD in the male infant was not ruled out. Subsequent Infant G6PD testing was negative. It was decided to commence primaquine once they are older than 6 months, as safety data in neonates is lacking.

This case highlights the complexities of managing *P. vivax* malaria in pregnancy and breastfeeding, the importance of maternal and infant G6PD testing and the utility of chloroquine for relapse prevention.

IAP13 Sauranil Guha (University College Cork)

"Investigating the role of early life microbiome in stress-social neurodevelopment and behaviour in zebrafish"

The microbiota communicates with the host through the gut–brain axis, shaping neurodevelopment from the earliest stages of life. Perturbations in this communication can result in long-lasting behavioural impairments, underscoring the need to identify critical windows during pre- and post-natal neurogenesis when microbial influence is most impactful (1). Zebrafish provide a powerful model to address these questions due to their transparent embryos, rapid external development, and amenability to microbial manipulation (2). By depleting microbiota from early embryogenesis and simultaneously tracking neurodevelopmental trajectories, we have uncovered critical neurodevelopmental deficits arising from microbial absence.

Our findings reveal that microbial depletion disrupts conserved stress–social circuits, including oxytocinergic pathways and the hypothalamic–pituitary–adrenal (HPA) axis, at the molecular level. These perturbations impair expression of key embryogenesis-related genes, alter hypothalamic development, compromise neurogenesis pathways, and delay hatching. Taken together, our work highlights the essential role of the microbiota in orchestrating normal brain development and the maturation of circuits underpinning stress responsivity and social behaviour.

These insights position the microbiota as a pharmacologically relevant target, with the potential to guide the development of novel therapeutic strategies aimed at mitigating neurodevelopmental and neuropsychiatric disorders linked to early-life microbial disruption (3).

IAP14 Helen Ye Rim Huang (RCSI/University Hospital Limerick)

"The potential role of carbapenems as an immunomodulator in severe refractory Hidradenitis Suppurativa: a case report"

Hidradenitis Suppurativa (HS) is a chronic, relapsing inflammatory disease characterised by recurrent abscesses and scarring. Severe Hurley stage III HS is often refractory to conventional therapy and complicated by secondary infections, significantly impairing quality of life (1). We report a case highlighting the potential immunomodulatory role of carbapenems in this context.

A 60-year-old male with Hurley stage III HS, psoriasis and monoclonal gammopathy of undetermined significance presented with bilateral buttock and groin abscesses, fever, and six-stone weight loss over one year. Despite extensive prior medical therapy - including doxycycline, infliximab, secukinumab and previous courses of clindamycin/rifampicin, dapsone, adalimumab, and minocycline – his condition was worsening. Examination revealed multiple discharging lesions with surrounding tenderness. Swab cultures of sacral ulcers, abscesses and buttocks grew staphylococcus aureus sensitive to flucloxacillin and piperacillin-tazobactam. With a working diagnosis of infective HS, we commenced these antibiotics with topical fusidic acid and metronidazole but failed to improve his condition. Surgical drainage was deemed high risk due to epidermal thinning and excoriation.

Following consultation with infectious disease and dermatology, meropenem was initiated. Over two weeks, the patient demonstrated marked clinical improvement, with reduced pain and discharge on follow-up. Post-admission, the patient was switched to ertapenem. He was referred abroad for reconstructive skin flap surgery.

This case illustrates the potential dual role of carbapenems as both antimicrobial agents and an immunomodulator in refractory HS. Few studies have described its use in this setting (2,3). Further investigations are warranted to explore the role of carbapenems in modulating inflammation, reducing infectious complications, and enhancing wound healing in HS.

IAP15 Eva Sophia Kalus (University College Cork)

"In vitro exploration of the therapeutic potential of siponimod in dry Age-Related Macular Degeneration"

Dry age-related macular degeneration (dAMD) is a leading cause of vision loss in the elderly, with no approved treatments for its advanced form. Chronic inflammation and dysregulated angiogenesis drive disease progression. S1P receptor modulators like siponimod may therefore offer therapeutic benefit by dampening immune cell trafficking and protecting retinal cells, addressing key pathogenic mechanisms.

We first established a model of dAMD by exposing Human Retinal Pigmented epithelium cells (RPE) to different concentration of oxidative stress inducers (H_2O_2, $KBrO_3$) and measuring Transepithelial Electrical Resistance (TEER), FITC-dextran permeability, and cell metabolic activity (MTT) under different cell culture conditions (serum levels) and different duration. Changes in TEER and FITC-DEX permeability suggested that H_2O_2 (800 µM) or $KBrO_3$ (150 µM) were the optimal test conditions to test siponimod. Assays were performed in X-Y independent experiments.

While siponimod had an effect on various parameters, none was statistically significant. H_2O_2 exposure increased apparent FITC-DEX permeability ($P_{app;}$ 1.45), which was modestly reduced by siponimod (1.38). $KBrO_3$ caused moderate barrier disruption (1.20), and siponimod restored permeability close to baseline levels (1.05). Furthermore, at 72 and 78 hours post treatment, siponimod increased the TEER of RPE monolayer compared to $KBrO_3$ or H_2O_2 treated controls. It is possible that the overall lack of statistical significance was related to the relatively weak disease phenotype induced under the conditions we tested, leaving siponimod too small a window for improvement. Future studies will therefore attempt to induce a more severe phenotype in primary cells to provide a more robust assessment of the therapeutic potential of siponimod or other candidate agents.

IAP16 Liam Kelly (University Hospital Limerick)

"Venous Thromboembolism Prophylaxis Prescribing in a Model 4 Hospital"

Background:

Venous thromboembolism (VTE) affects millions of individuals yearly. Annual incidence is estimated to be 1-2 per 1000 individuals (1). VTE is a leading preventable cause of hospital death. 50-60% of all VTE cases occur during or after hospitalization. Risk assessing patients and prescribing thromboprophylaxis early in their hospital admission reduces their risk (2). NICE guidelines recommend risk assessments be performed on all patients on admission. Our audit aimed to assess the compliance with VTE prophylaxis prescribing guidelines and utilisation of the drug kardex risk assessment tool.

Methods:

A review of the drug kardex of consecutive medical inpatients was undertaken in a university teaching hospital. Completion of the VTE risk assessment tool, prescription of VTE prophylaxis, and assessment of prescribing appropriateness was recorded.

Results:

In total, 152 medical patients were included. In 71 cases, VTE prophylaxis was appropriately prescribed. VTE prophylaxis was inappropriately prescribed in one patient. VTE prophylaxis was appropriately not prescribed in 72 patients, in 8 patients VTE prophylaxis was inappropriately omitted.

A VTE/bleeding risk assessment was documented in 3.3% of drug kardexes.

Discussion:

This audit demonstrated 94.1% compliance with current NICE VTE prophylaxis guidelines. Despite high compliance, adverse VTE complications can have life-threatening implications for patients therefore improved utilisation of the VTE risk/bleeding risk assessment tool has potential for benefit. Audit outcomes informed updates to the drug kardex, via the Drugs and Therapeutics and VTE Committees to facilitate higher compliance with the VTE/bleeding risk assessment tool and behavioural change.

IAP17 Roisin Kelly-Laubscher (University College Cork)

"Effects of ethanolamine on in vitro and in vivo doxorubicin-induced cardiotoxicity"

Introduction. Doxorubicin, an anthracycline chemotherapeutic drug, is associated with significant cardiotoxicity. Ethanolamine protects the isolated heart against ischaemia-reperfusion injury; however, whether it can protect against doxorubicin-induced cardiotoxicity (DIC) is unknown [1].

Methods The effects of ethanolamine (3 µM-300 mM) on doxorubicin (5 µM or 10 µM)-induced cytotoxicity were determined in H9c2 cardiomyoblasts, NIH3T3 fibroblasts and isolated murine cardiomyocytes using MTT, trypan blue exclusion and LDH assays. Male and female C57/Bl6 mice (n=12/group), pretreated with ethanolamine (1.6, 16, or 160 mg/kg), were exposed to acute (20 mg/kg i.p.) or chronic (6 mg/kg/week i.p. for 3 weeks) doxorubicin treatment. After 1 week (acute) or 6 weeks (chronic), cardiac biomarkers and histology were assessed. Data was analysed using one-way or two-way ANOVA followed by Dunnett's *post hoc* test.

Results. Ethanolamine pretreatment of NIH3T3 cells significantly increased metabolic activity (30 µM; 94.80±18.68%, 300 µM; 89.45±29.56%, 3 mM; 100.40±16.52%) compared to doxorubicin (14.96±14.37%, $p<0.05$). Doxorubicin alone did not cause significant changes BNP and troponin levels, in the acute or chronic models. However, pretreatment with 16 mg/kg ethanolamine increased serum levels of BNP (161.2±31.52 pg/mL) compared to doxorubicin (86.42±13.02 pg/mL; $p=0.0099$). This increase was only seen in female animals.

Conclusion Ethanolamine increased cardiac damage in females in the chronic model of DIC. While the nonsignificant effect of doxorubicin alone suggests subthreshold exposure in this model, it is possible that the observed cardiac damage in females results from a combined effect of doxorubicin and ethanolamine. Considering ethanolamine is found in the diet, these findings warrant further investigation.

IAP18 Ciarán Kennedy (University College Dublin)

"Tipping the balance: Investigating the potential of the specialised pro-resolving mediator Lipoxin-A4 to modulate the inflammatory landscape in atherosclerosis"

Atherosclerosis, the leading cause of cardiovascular disease (CVD)-related mortality, is a chronic inflammatory condition characterized by complex and multifactorial progression. Diagnosis typically occurs only after significant disease advancement, limiting opportunities for early therapeutic intervention. While *in vivo* models have yielded key insights, their translational relevance is often limited. To address this, we employed a human *ex vivo* model using carotid endarterectomy tissue, which remains metabolically active and responsive to *stimuli*. Tissue sections (3×3×3 mm) were cultured in the presence or absence of the pro-inflammatory cytokine Tumour Necrosis Factor (TNF; 5 ng/mL) and treated with the specialized pro-resolving mediator (SPM) Lipoxin A4 (LXA4; 1-100 nM). We demonstrate that this model reliably responds to inflammatory *stimuli* and allows for effective evaluation of therapeutic candidates. Importantly, we established a method for extracting both RNA and protein from the same tissue sections, maximizing tissue use and reducing inter-patient variability. LXA4 treatment attenuated TNF-induced pro-inflammatory cytokine release (IL-6, IFNγ, IL-1β, IL-10) and promoted a pro-resolving tissue environment, as evidenced altered markers of macrophage phenotype. These findings show that LXA4 can actively reshape the inflammatory profile of human atherosclerotic plaque tissue, promoting resolution pathways. This work underscores the utility of *ex vivo* human plaque models for translational research and highlights SPMs as promising therapeutic candidates for targeting chronic inflammation in atherosclerosis.

IAP19 Andrea Kwakowsky (University of Galway)

"Treatment of β-amyloid-induced increased tonic conductance and cognitive deficits by an α5 inverse agonist"

Alzheimer's disease (AD) is a chronic progressive neurodegenerative disorder characterized by cognitive impairment, which may arise from disruptions in the excitatory/inhibitory balance within the brain. Gamma-aminobutyric acid (GABA), the principal inhibitory neurotransmitter in the central nervous system, plays a crucial role in maintaining the excitatory/inhibitory balance and regulating neuronal activity involved in memory. In AD, changes in α5 GABA A receptor (α5GABAAR) expression and activity increase tonic inhibition, disturbing the neuronal excitatory/inhibitory balance and ultimately impairing cognitive processes. Therefore, targeting α5GABAAR offers a promising therapeutic strategy. This study examined the potential of an α5GABAAR-selective inverse agonist, α5IA, in treating β-amyloid-induced cognitive deficits and the mechanism underlying this using ex vivo microelectrode array and patch clamp electrophysiology. α5IA significantly reduced β-amyloid-induced long-term potentiation and long-term spatial memory deficits. α5IA reversed Aβ-induced increase in neuronal excitability, as indicated by input-output curves, and mitigated elevated tonic conductance. These findings highlight α5IA's ability to restore excitatory/inhibitory balance and cognitive function. The selective targeting of α5-GABAARs and enhancing the efficacy of α5-GABAAR inverse agonists, such as α5IA represents a promising direction in developing novel AD therapies.

IAP20 Benjamin Leahy (University Hospital Limerick)

"Sometimes it takes steel, not just syringes"

Venous thromboembolism (VTE) is a leading cause of maternal morbidity and mortality and poses significant challenges in the peri-partum period.

A 36-year-old woman (G8P2), with obesity, multiple miscarriages and gastric bypass, developed an iliofemoral deep vein thrombosis (DVT) day-5 post Caesarean-section, despite being on therapeutic enoxaparin 100mg BD (1mg/kg–102kg) for a tibiofemoral DVT. This developed despite prophylactic tinzaparin, and aspirin given for the duration of her pregnancy. Of note, her therapeutic enoxaparin was paused for C-section for 36 hours (12 hours longer than recommended).

With progression of her DVT, anti-Xa-levels were measured at 0.54 IU/ml (target 0.6-1). Enoxaparin was increased to 1.1mg/kg BD (110mg), achieving therapeutic anti-Xa levels. A medication strategy only was considered inadequate, in view of the progression of thrombosis despite enoxaparin and clinical appearances suggestive of an evolving phlegmasia cerulea dolens. Therefore, a thrombectomy was performed with pre-op red-cell transfusion (Hb7). Thrombolysis was not appropriate with recent surgery.

This case underscores the potential risks of underdosing of enoxaparin in the pregnancy/postpartum setting. Physiological changes in pregnancy /early post-partum alter pharmacokinetics of enoxaparin due to 1) increased renal clearance, [1] 2) increased volume of distribution [1] 3) lower anti-thrombin levels (lower heparin co-factor associated with reduced LMWH efficacy) [2] and 4) higher heparin binding proteins (reduce heparin bioavailability). This demonstrates that weight-based dosing may not always be therapeutic.

Enoxaparin was continued on discharge to avoid DOAC absorption concerns relating to gastric bypass and fluconazole interaction (severe perineal candidiasis). With multiple miscarriages, testing for anti-phospholipid syndrome was negative.

IAP21 Jiuang Li (University of Galway)

"Examination of the Hippocampal Transcriptome in Alzheimer's Disease: A Systematic Review of the Excitatory and Inhibitory Imbalance Based on Transcriptomics Data"

Excitatory/inhibitory (E/I) imbalance in the hippocampus is increasingly recognized as a key mechanism in Alzheimer's disease (AD) progression yet the transcriptomic architecture linking E/I synaptic signalling to disease pathology remains incompletely understood. To investigate the molecular basis of this dysregulation, four hippocampal transcriptomic datasets from the GEO database were selected, and differentially expressed genes (DEGs) were identified using GEO2R (adj. $p<0.05$, $|\log_2 FC|>0.58$). After merging and deduplication, 986 DEGs were obtained and subjected to Kyoto Encyclopedia of Genes and Genomes (KEGG) and Gene Ontology (GO) enrichment via the SangerBox platform. Six KEGG pathways directly related to E/I balance were identified based on pathway names and gene function, from which key genes were extracted. A high-confidence protein–protein interaction (PPI) network was constructed in STRING, followed by topological analysis in Cytoscape. Genes within the top 10% of degree and betweenness centrality were further analysed. Reactome enrichment was performed — for the full DEG set and also for the topological core genes — with pathway ranks compared to identify consistent functional signatures. The highest-scoring MCODE module (37 genes) was extracted from the PPI network and analysed for protein complex enrichment using the CORUM database. Fisher's exact test revealed significant overrepresentation of mitochondrial respiratory chain complexes. These findings suggest that mitochondrial dysfunction may underlie hippocampal E/I imbalance in AD. This integrative approach highlights biologically coherent gene modules and protein complexes linking synaptic signalling disruption with impaired cellular metabolism in the AD hippocampus.

IAP22 Orlaith Magnier (University Hospital Limerick)

"A Case Report showing Successful Use of Ranolazine for Symptomatic Bradycardia as an Alternative to Beta Blocker"

An 87-year-old female with uncontrolled hypertension presented with symptomatic bradycardia and elevated blood pressure, shortly after initiating diltiazem therapy. Holter monitoring demonstrated a high burden of supraventricular ectopy (SVE), with frequent bradycardic episodes. Transthoracic echocardiography, 24-hour ambulatory blood pressure monitoring (whilst on valsartan), and head-up tilt testing were unremarkable. Beta-blocker therapy with bisoprolol was commenced but resulted in exacerbation of symptomatic bradycardia. Subsequently, the patient was transitioned to ranolazine, which led to normalization of heart rate (mean 70–80 bpm) and resolution of symptoms.

High-burden SVEs increase the risk of atrial arrhythmias, left ventricular dysfunction, and potential progression to cardiomyopathy. Standard management often includes beta blockers or calcium channel blockers to maintain sinus rhythm and suppress ectopy. However, beta blockers may aggravate bradycardia, limiting their use in certain patients. Ranolazine, an inhibitor of late sodium currents that reduces intracellular calcium overload, exhibits electrophysiological stabilization without significant negative chronotropic effects. While licensed for chronic angina, ranolazine's off-label use in arrhythmia management – particularly supraventricular arrhythmias – remains tentative.

This case illustrates successful symptomatic control of SVEs with ranolazine in a patient contraindicated for beta-blocker therapy due to bradycardia. Further clinical studies are needed to elucidate the role of ranolazine in arrhythmia management.

IAP23 Shalen Maharaj (University Hospital Limerick)

"Ischaemic Stroke Following First Cycle of Cisplatin/Etoposide for Non-Seminomatous Germ Cell Tumour: Incidental, Cisplatin related?"

Abstract:

Background: Cisplatin-based chemotherapy remains the backbone of treatment for non-seminomatous germ cell tumours (NSGCT). While venous thromboembolism (VTE) risk is well recognised, systematic reviews and meta-analyses also highlight cisplatin's prothrombotic potential, and emerging data implicate early arterial thromboembolic events (ATE) including stroke.

Case Presentation: A 29-year-old smoker with stage pT2 mixed NSGCT underwent right radical orchidectomy followed two months later by adjuvant cisplatin–etoposide. Seven days after cycle 1 (EP C1D8), he developed in-hospital acute left hemiplegia, hemianopia, dysarthria, and neglect (NIHSS 16). Imaging confirmed right internal carotid and middle cerebral artery thrombus with partial vertebral occlusion. He received intravenous thrombolysis and mechanical thrombectomy (Figure 1) with carotid stenting, achieving full neurological recovery. No alternative cause was identified.

Management and Outcome: Antiplatelet therapy, high-dose statin, and smoking cessation were initiated. Multidisciplinary discussion highlighted the curative benefit of cisplatin compared with the inferior efficacy of carboplatin. After weighing survival benefit against thrombotic risk, the decision was made to rechallenge with cisplatin, adding prophylactic LMWH. Temporal proximity, negative work-up, early ATE signal, and explicit label warnings indicated a plausible cisplatin-related arterial event. The Naranjo algorithm supported this probability.

Conclusion / Learning Points:

A meta-analysis of RCTs confirmed increased VTE risk with cisplatin (1), and large cohorts demonstrate a significantly increased short-term risk of ATE in testicular cancer (2,3). Regulatory authorities explicitly list arterial thrombotic events as cisplatin toxicities (4–6). Pathophysiological mechanisms include endothelial injury with ↑vWF, pro-coagulant shifts, hypomagnesemia-induced vasospasm, and thrombotic microangiopathy. Clinicians should recognise cisplatin as both lifesaving and thrombogenic, integrating cardiovascular risk reduction and vigilance during early cycles.

IAP24 Konstantinos Matheoudakis (University College Dublin)

"Using oxygen glucose deprivation to investigate the neuroprotective and modulatory effects of antioxidant compounds on synaptic transmission and plasticity"

The authors have chosen not to make the contents of this abstract available online

IAP25 Oran McNamara (University of Galway)

"Bumetanide as a Candidate Therapy for Neurological Disorders: Insights from Meta-Analysis and Preclinical Models"

Neurological disorders represent a major global health burden, yet effective treatments remain limited. Drug repurposing offers a cost-effective strategy, with bumetanide, a loop diuretic antagonising the NKCC1 transporter, showing promise in correcting pathological GABAergic signalling (1). To evaluate the therapeutic potential of bumetanide, we conducted a systematic literature review and meta-analysis alongside *in vitro,* and *ex vivo* experiments.

Meta-analysis revealed that bumetanide significantly reduced childhood autism spectrum disorder symptom severity compared with placebo, with additional evidence of benefit in Alzheimer's disease. Preclinical data also suggests possible efficacy across several neurodegenerative and neurodevelopmental disorders. However, bumetanide displays limited blood-brain barrier penetration, raising concerns about central bioavailability (2), with peripheral mechanisms potentially contributing towards clinical efficacy (3). To assess safety and efficacy, we examined the effect of bumetanide on primary hippocampal neurons and mouse hippocampal slices. *In vitro*, bumetanide exposure reduced neuronal viability at higher concentrations, but not at the lowest concentration tested (1 nM). Co-treatment with amyloid-β exacerbated neurotoxicity. *Ex vivo*, multi-electrode array analysis further demonstrated that bumetanide did not restore long-term potentiation in the hippocampal CA1 subregion following amyloid-β challenge.

Collectively, these findings highlight both the therapeutic promise and safety concerns associated with bumetanide. While clinical and preclinical data support potential use, our results suggest that bumetanide is neurotoxic at doses commonly used experimentally and fails to restore synaptic plasticity in amyloid-compromised tissue. Careful dose consideration and mechanistic clarification are therefore essential for the progression of bumetanide as a candidate therapy for neurodegenerative disease.

IAP26 Braden Millar (University College Dublin)

"Exploitation of Macrophage Plasticity using Specialised Pro-resolving Mediators (SPMs) in an in vitro cell model of Early Atherogenesis"

Macrophages play a pivotal role in atherosclerosis disease pathogenesis, as they are directly involved in plaque formation. Classification of macrophages into two sub-classes, inflammatory M1 and anti-inflammatory M2, is widely considered as outdated, due to high plasticity of these cells, and the multitude of functions associated with different macrophage phenotypes. This plasticity plays a major role in disease pathogenesis and its exploitation, using SPMs, may provide a new route to returning a chronic inflammatory disease to homeostasis [1][2].

Our *in vitro* model of early atherogenesis aimed to characterise the different macrophage phenotypes using a combination of differentiating and polarising agents. These phenotypes were then subjected to various functional assays such as migration and cholesterol trafficking to examine their functionality in atherosclerosis.

Analysis of cholesterol trafficking in M1 macrophages from our *in vitro* model, mimic the *in vivo* generation of foam cells *vs* naïve macrophages (by 2.81 ± 0.18, $p<0.0001$), and also the persistence of an inflammatory environment *via* increased oxidised-LDL exposure. Subsequent RT-PCR analysis shows this is mediated through an inflammatory modulation of cholesterol influx/efflux proteins *OLR1* (by 3.39 ± 1.7, $p<0.05$, increase) and *ABCA1* (by 0.6 ± 0.24, $p<0.01$, decrease), respectively.

SPM treatment yielded a significant alteration of monocytes, M1 and M2 macrophage migration towards monocyte chemoattractant protein-1 or conditioned-medium from human carotid plaque tissue, presenting a promising view into monocyte and macrophage migration/emigration, a crucial aspect of disease pathogenesis/regression.

These findings highlight a robust *in vitro* model of disease pathogenesis, unearthing a potential role for SPMs as a therapeutic *via* exploitation of macrophage plasticity.

IAP27 Marwa Mustaf and **Jethen Maharaj** (University Hospital Limerick)

"Re-Audit of Polypharmacy in Inpatients"

Polypharmacy, defined by the World Health Organization as the concurrent use of five or more medications, is increasingly prevalent in older adults due to multimorbidity and ageing populations. It is associated with adverse drug reactions, drug–drug interactions, falls, and hospital readmissions (1). An initial audit in 2024 across medical wards revealed high rates of polypharmacy, limited documentation of medication reconciliation, and frequent potentially inappropriate prescribing. Educational interventions were subsequently implemented to promote safer prescribing practices.

This re-audit, conducted in July 2025 over two days period, assessed 35 consecutive inpatients aged ≥65 years admitted to medical wards. Data collected included age, sex, number of medications at admission and discharge, evidence of medication reconciliation, deprescribing activity, and potential drug interactions. Standards were based on the HSE Medication Safety Programme, STOPP/START criteria, and NICE Medicines Optimisation guidance (2,3).

The mean age was 81 years (range 67–95); 15 patients were female. Documentation of medication reconciliation improved from 22% (8/35) in 2024 to 46% (16/35) in 2025, while patients without reconciliation fell from 78% (27/35) to 54% (19/35). Potential drug interactions decreased slightly from 17% (16/35) to 14% (12/35). Polypharmacy, however, remained widespread.

These findings suggest modest improvement in reconciliation and a small reduction in inappropriate prescribing but highlight the persistence of polypharmacy and incomplete documentation. The results underscore the need for sustained multidisciplinary interventions, including pharmacist-led reviews and electronic prescribing supports. A repeat audit is planned within 12 months to evaluate further progress.

IAP28 Seema Nathwani (University College Dublin)

"The Nature-Action-Consequence (NAC) Tool: A Structured Framework to Support Pharmacology Learning"

Pharmacology can be a challenging subject due to its content breadth, the continual emergence of new drugs and the need to link molecular mechanisms with therapeutic outcomes [1]. Various pedagogical innovations -aimed at improving engagement- can be employed [2,3], however, many of these techniques (such as Q&A flashcards) reinforce fragmented recall, rather than conceptual integration. To fill this gap, we have developed a structured framework, the *Nature-Action-Consequence (NAC)* tool, by scaffolding knowledge in a causal sequence: "nature" (what the drug/class is); "action" (molecular/physiological mechanism) and "consequence" (therapeutic benefits, side effects, contraindications). By requiring learners to capture this triad, NAC helps to condense content into a meaningful structure. Our aim was to explore students' perceptions of NAC as a complement to existing teaching.

The NAC tool was implemented in undergraduate pharmacology modules. Flashcards for core drug classes were created using NAC and shared as study aids. Lectures and tutorials also signposted students to NAC. We analysed routine module evaluations and extracted comments in which NAC was mentioned unprompted. Feedback suggested that the NAC tool was positively perceived. Students described NAC as "very helpful," "well structured," and "a great learning tool". These preliminary findings indicate NAC provides a useful framework to support pharmacology learning. Unlike traditional flashcards – that present isolated Q&A pairs – NAC integrates molecular and clinical perspectives, reducing overload and prioritising core knowledge. To support our initial feedback, a formal survey is planned. If validated, NAC could be adapted into analogue or digital formats, offering a scalable strategy for pharmacology education.

IAP29 Nouman Niaz (University Hospital Limerick)

"Unmasking Impulse control disorders: Ropinirole and Hypersexuality"

Background:

Dopamine agonists, particularly those with high affinity for dopamine D3 receptors such as ropinirole, are widely used in the management of Parkinson's disease and restless legs syndrome. Impulse control disorders (ICDs) are increasingly recognised as significant behavioural complications of dopamine agonist therapy in Parkinson's disease. The mesolimbic dopaminergic pathway, modulated by dopamine D3 receptor activity, appears central to the development of these behaviours. Hyper-sexuality, although less commonly reported than other ICDs, can severely affect patient quality of life. Recognition of these adverse behavioural outcomes remains a clinical challenge.

Methods:

We report a 77-year-old man with Parkinson's disease who developed new-onset hyper-sexuality within 3 weeks of ropinirole dose escalation to 12mg/day. The behaviour included compulsive sexual activities and socially inappropriate advances. There was no prior psychiatric history, and no use of other medications implicated in behavioural dysregulation. As ropinirole was gradually tapered to 2mg/day, parkinsonian symptoms worsened, prompting the introduction of opicapone, a COMT inhibitor, to optimise motor control. This adjustment resulted in complete resolution of hyper-sexuality over several weeks without exacerbation of parkinsonian symptoms.

Results:

This case illustrates a dose-dependent association between ropinirole and impulse control disorders, specifically hyper-sexuality, highlighting the mesolimbic dopaminergic pathway's role in mediating reward and compulsive behaviours. Incorporation of screening tools such as QUIP and regular behavioural assessments, especially during dose adjustments, is warranted.

Conclusion:

Hyper-sexuality is an under-recognised but reversible adverse effect of ropinirole therapy. Education of patient and caregivers, regular behavioural screening, and timely therapeutic adjustments are essential to minimise these potentially disabling outcomes.

IAP30 Eilidh O'Connor (University College Dublin)

"Targeting metabolic and inflammatory drivers of Barrett's oesophagus progression with 1,4-Dihydroxy Quininib"

Barrett's Oesophagus (BO) is a pre-malignant condition of the oesophagus and a major risk factor for oesophageal adenocarcinoma (OAC), an aggressive cancer with poor prognosis and limited treatment options. Progression from metaplasia to dysplasia and eventually carcinoma is driven by chronic inflammation and metabolic dysregulation. However, the molecular mechanisms underpinning this progression remain poorly defined. Crucially, no therapeutic options are currently available to slow or prevent disease progression, and no biomarkers exist to stratify patients based on progression risk.

This study investigated the effects of 1,4-dihydroxy quininib (Q7), a novel cysteinyl leukotriene receptor 1 ($CysLT_1$) antagonist with anti-inflammatory and anti-cancer properties, on metabolic, inflammatory, and proliferative pathways in a three-stage in vitro BO-to-OAC progression model: QH (metaplasia), GO (dysplasia), and OE33 (OAC). Real-time metabolic profiling revealed a progressive increase in oxidative phosphorylation (OXPHOS), indicating a metabolic shift toward mitochondrial respiration. Q7 significantly reduced OXPHOS in all three cell lines without affecting glycolysis, suggesting a selective inhibition of mitochondrial metabolism. Multiplex cytokine analysis showed that Q7 enhanced IL-12p70 and IFN-γ, while reducing IL-4 and IL-8, consistent with an anti-tumour immune response. Proliferation assays demonstrated a significant reduction in long-term cell survival following Q7 treatment in all 3 cell lines. Gene expression analysis identified PROM1, STMN1, and CYSLTR1 as markers of progression modulated by Q7. Together, these findings suggest that Q7 alters critical oncogenic processes across the BO-OAC axis. Q7 therefore emerges as a promising candidate for chemoprevention and targeted therapy in patients at high risk of BO progression.

IAP31 Francis N. Odinukaeze (University of Galway)

"Differential Expression of Inflammasome-Related Genes in the Alzheimer's Disease Hippocampus: A Bioinformatics Perspective"

Inflammasomes, multiprotein complexes that modulate inflammatory responses, have emerged as critical players in neurodegeneration, particularly Alzheimer's disease (AD). Among them, the NOD-like receptor family, pyrin domain containing 3 (NLRP3) has been most extensively studied (Maran *et al.*,2023), with dysregulated activation linked to amyloid pathology, tau hyperphosphorylation, and neuroinflammation (Holbrook *et al.*, 2021; McLarnon *et al.*, 2006). This study investigated differentially expressed inflammasome-associated genes (DEGs) in the human hippocampus of AD patients using publicly available transcriptomic datasets. Four eligible RNA-Seq and microarray studies from the GEO database were analysed for DEGs in hippocampal tissues from AD versus control cases. Inflammasome-related genes were retrieved from GeneCards and mapped to identified DEGs. KEGG and Reactome pathway enrichment, as well as protein–protein interaction (PPI) network analyses, were performed to uncover key regulatory nodes. Core inflammasome genes (NLRP3, NLRP1, AIM2, CASP1, IL1B, IL18, PYCARD, P2RX7, and TXNIP) showed inconsistent expression patterns across datasets. However, network analyses revealed central involvement of TNF/NF-κB signalling, apoptosis, NOD-like receptor signalling, and TRP channels in the regulation of the inflammasome. Hub and bottleneck genes such as MAPKs, ITPRs, and PLCB4 emerged as critical mediators linking inflammasome activity to hippocampal dysfunction in AD. Although bulk hippocampal transcriptomics revealed limited consistency in direct inflammasome gene dysregulation, integrative network analyses highlighted pathways that converge on inflammasome activation. These results suggest that inflammasome-related pathology in AD may be driven by cell-type-specific or post-transcriptional mechanisms not captured by bulk tissue analysis. Targeting upstream regulators of inflammasome signalling may hold therapeutic potential in AD.

IAP32 Anisha Pattanayak and Daniel Partridge (Trinity College Dublin)

"Applying clinical trials to clinical practice: Generalisability of novel lipid lowering therapies to a specialist lipid clinic cohort"

Numerous novel lipid-lowering agents are progressing through late-phase trials, yet restrictive eligibility criteria may limit their applicability to real-world patients. The aim of this project is to map the eligibility criteria of clinical trials of novel lipid lowering therapies to a cohort of patients attending a specialist lipid clinic, to determine whether emerging therapies can benefit patients typically seen by clinicians.

A study was conducted at a specialist clinic in St James's hospital, Dublin, over 6 months enrolling 370 patients. A search of clinicaltrials.gov and euclinicaltrials.eu identified phase 3 trials registered since 2018. Cohort characteristic data were mapped against trial eligibility criteria to estimate the proportion of clinic patients eligible for each study. Patients were additionally subclassified into clinically relevant groups to assess the generalisability of trial populations to real-world subgroups.

The search identified 38 trials which included 16 different novel lipid-lowering drugs. The percentage of the cohort (n=370) that would have been eligible for these lipid-lowering therapy trials ranged from 0% up to 62.4%, with an average patient eligibility of 19% across the 38 trials.

Eligibility varied considerably across trials, reflecting heterogeneity in target populations, therapeutic classes and their mechanisms of action. The included novel lipid-lowering therapy trials are reasonably applicable to a specialist lipid clinic cohort, suggesting generalisability to a wider group of patients seen regularly in clinics. The study is therefore indicative of the likely potential for use of these novel therapies in clinic practice.

IAP33 Louise Rabbitt (University of Galway)

"Biochemical Detection and Psychosocial Correlates of Antihypertensive Adherence in Hypertension: Initial Observations from a Cross-Sectional Study in Two Irish Tertiary Care Centres"

The authors have chosen not to make the contents of this abstract available online

IAP34 Domhnall Roe (Trinity College Dublin)

"Factors Affecting Clinical Trial Startup Speeds at an Irish Clinical Research Facility"

Successful navigation of the startup phase is crucial to initiating clinical trials efficiently [1]. This quantitative study aimed to evaluate startup times and influencing factors at an Irish clinical research facility.

156 clinical trials with available data were identified from the facility database. Data were extracted into Excel and analysed using descriptive statistics.

The mean and median startup speeds (i.e. number of days from receipt of clinical trial application to green light status) were 188 days and 207 days, respectively. 42% of trials had startup times of less than 150 days.

Studies with higher intensity scores showed longer mean startup times (247.5 days high-intensity versus 113.4 days low-intensity).

Analysis by funding status showed longer mean startup durations for commercial trials versus non-commercial trials (193.5 days versus 118.0 days, respectively).

Analysis showed a visual trend of increasing startup durations from 2015 until 2021 (excluding 2019), followed by a downward trend from 2021 onwards.

Less than half of trials met the UKCRD 150-day startup target, indicating a need for improvement [2]. The observed decrease in yearly startup speeds since 2021 suggests progress. Longer startup times in commercial and high-intensity studies points towards potential barriers affecting these trials. We aim to carry out further research into factors affecting startup times which incorporates statistical analysis and involves other clinical trials units to provide a larger dataset for interrogation.

IAP35 Domhnall Roe (Trinity College Dublin)

"Staff Perspectives on Clinical Trial Progression: Barriers, Enablers, and Solutions"

Various factors influence the progression of clinical trials. Understanding these factors is essential to inform trial design and improve the impact of clinical research [1]. This study aimed to explore staff experiences of factors affecting trial progression at a large Irish clinical research facility.

Semi-structured one-to-one interviews with staff members (n=7) were conducted, including interviewees from the professions of nursing, pharmacy, research, and quality/regulatory affairs. An interview schedule consisting of open-ended questions was used to guide interviews. Interview transcripts were analysed using thematic analysis to identify overarching themes and subthemes [2]. An ethics approval waiver was granted, and the study was registered by the Quality and Safety Improvement Directorate of St James' Hospital.

Four themes were identified from the thematic analysis. Good communication between stakeholders, adequate staff training and specialist input for trials were found to enable trial progression. Lack of trial understanding and accessibility for participants were major barriers identified. Stringent eligibility criteria and complex trial design lacking participant focus was another key theme. Operational challenges and their impact on staff morale, such as approval delays, sponsor dropout, and unpredictability at start-up stages, were emphasized.

Clinical trials staff experience a variety of enablers and barriers affecting trial progression, including staff-related, participant-related, design-related, and operational factors. This prompts consideration of important issues such as complex eligibility criteria, trial design challenges, and staff time constraints. Further research involving more clinical trial stakeholders and other clinical trials units is required to better understand these factors and identify solutions to barriers identified for future implementation.

IAP36 Anastasia Saleh (Trinity College Dublin)

"Evaluating the Environmental Impact of Fixed Dose Combinations for Chronic Disease Management"

Introduction

Polypharmacy contributes to environmental waste, drug regimen complexity, and healthcare costs. Fixed Dose Drug Combinations (FDCs) combine two or more active drugs in a single dosage form. FDCs may reduce the carbon footprint of healthcare compared to free equivalent combinations while simplifying regimens, lowering costs, and supporting adherence. This study aimed to assess evidence of the environmental benefit of FDCs.

Method

A systematic review followed by a scoping review was conducted in accordance with the JBI Manual for Evidence Synthesis and reported using PRISMA Extension for Scoping Reviews. Reviews were registered in PROSPERO (CRD42024612556) and the Open Science Framework. Databases searched included EMBASE, Medline OVID, CINAHL, Web of Science, and Google Scholar. Studies were screened by two independent reviewers with conflicts resolved by a third.

Results

A total of 1,367 studies were identified. After removing 114 duplicates, 1,253 abstracts were screened; 1,239 were irrelevant. Fourteen studies required third party adjudication, but none met inclusion criteria. The systematic review yielded no relevant studies. The repeated scoping review also did not yield results.

Conclusion

There is a dearth of evidence regarding environmental benefits of FDCs. Hypothetically, reduced packaging, transport, manufacturing emissions, and fewer hospital bed days from improved adherence suggest potential benefit. Given climate change concerns and healthcare's contribution to carbon emissions, further study into the environmental impact of FDCs represents a promising direction in sustainable healthcare delivery.

IAP37 Aditi Sooknarine-Rajpatty (University Hospital Limerick)

"A Case of Serotonin Syndrome secondary to Drug Interactions in a 78-Year-Old Gentleman: A Case Report"

Background

Serotonin syndrome (SS) is a potentially life-threatening disorder caused by serotonin excess, in the setting of higher therapeutic doses, drug-drug interactions, or recreational drug use[1]. It is an uncommon condition and is often difficult to diagnose particularly when presenting with vague or mild symptoms.

Case

A 78-year-old gentleman, DM, presented with ongoing nausea on a background of depression treated with sertraline. He was recently prescribed a prolonged course of linezolid for treatment of pyelonephritis. His nausea was initially treated with cyclizine, however due to poor response was switched to ondansetron. Following these medication changes, DM was confused, exhibiting both visual and auditory hallucinations, and delusions. There was evidence of tremors and myoclonic jerks in bilateral lower limbs and facial muscle. He was also noted to have ongoing postural instability. Following multidisciplinary discussion between psychiatry, geriatric medicine, and clinical pharmacology their impression was possible serotonin syndrome secondary to the combination of sertraline, linezolid, and ondansetron, which all increase serotonin concentration at the neuronal synapse. CK level was normal. Advice was to reduce sertraline, discontinue linezolid and ondansetron.

Discussion

DM had been intermittently confused for the preceding week, but the impression had been delirium due to resolving pyelonephritis. The SS became apparent following the emergence of tremors and myoclonic jerks; however, we had not suspected this in the setting of mild delirium. This case stresses the importance of considering drug interactions and potential SS as a differential for delirium particularly in older adults.

IAP38 Aditi Sooknarine-Rajpatty (University Hospital Limerick)

"Mapping the evidence on epistaxis and systemic thrombolysis: A Scoping Review"

Background:

Systemic thrombolysis using agents like alteplase and tenecteplase is central to treating acute ischaemic stroke. While intracranial haemorrhage is a known complication, extracranial bleeding events such as epistaxis have received less attention[1]. Clinical observations and case reports suggest a link between thrombolysis and epistaxis, especially in individuals with a history of nasal bleeding. However, the incidence, risk factors, and clinical relevance of these events remain unclear.

Objectives:

This review aims to systematically map existing literature on epistaxis as an adverse event following systemic thrombolysis.

Methods:

A scoping review, using PRISMA-ScR methodology, was conducted to synthesise evidence from case reports, cohort studies, RCTs, and pharmacovigilance data. Included studies involved adult patients who received thrombolysis, with particular focus on those with predisposing conditions like HHT. Extracted data included thrombolytic agent and dose, indication for thrombolysis, patient comorbidities, timing and severity of epistaxis, and management approaches.

Results & Impact:

The review identified 547 potentially relevant articles, with 71 assessed in full. Most included studies were case reports from the U.S. focusing on stroke patients. Epistaxis severity varied from mild to severe, with management ranging from conservative approaches to cessation of thrombolysis and blood transfusion. Findings will be presented in narrative and tabular formats, highlighting data gaps and reporting trends.

Conclusion:
We aim to inform clinical protocols, guideline development, and evidence-based decision-making in acute stroke care.

IAP39 Huiyuan Yang (University College Cork)

"Recombinant Pregnancy-Specific glycoprotein-1 is protective in an in vitro model of intracerebral haemorrhage"

Intracerebral hemorrhage (ICH) carries a high mortality (≈40–50% at 30 days) and currently has no specific pharmacotherapy [1]. Targeting the neuroinflammatory cascade, which drives secondary brain injury mediated by microglia activation, might be effective. Recombinant Pregnancy-Specific Glycoprotein-1 fused to Fc (rPSG1-Fc) is a candidate immunomodulator effective in ischemic stroke [2] but its potential in ICH has not been explored.

BV2 microglial cells were treated with hemin (50 μM) to mimic ICH in vitro, with or without rPSG1-Fc for 24 hours. Cell viability was measured (MTT assay); IL-10, TGF-β1, and TNF-α levels were quantified by ELISA; TGF-β1, Smad3, and CD32 expression was assessed by Western blot and qPCR; immunofluorescence was used to visualize M1 (CD86) and M2 (TGF-β1) markers. Statistical analysis used one-way ANOVA with Tukey's multiple comparisons.

rPSG1-Fc improved cell viability dose-dependently compared to the hemin-only group ($p<0.05$). rPSG1-Fc reduced pro-inflammatory cytokine TNF-α ($p=0.002$ vs hemin), while increasing anti-inflammatory cytokines IL-10 and TGF-β1 ($p<0.0001$ vs hemin). Both protein and mRNA levels of TGF-β1/Smad3 were elevated with rPSG1-Fc treatment. The pro-inflammatory marker CD32 was suppressed ($p<0.0001$ vs hemin), and the M2 marker TGF-β1 increased ($p<0.01$ vs hemin). Immunofluorescence confirmed a shift in microglia from $CD86^{+}/TGF\text{-}\beta1^{-}$ (M1) to $CD86^{-}/TGF\text{-}\beta1^{+}$ (M2).

rPSG1-Fc's anti-inflammatory effects may be via the TGFβ1/Smad 3 pathway. These findings confirm its therapeutic potential as an immunomodulatory agent for ICH-related neuroinflammation. Future studies will use an ICH model in rats (intrastriatal collagenase) to assess rPSG1-Fc's neuroprotective effects (neurological outcomes, brain edema reduction, lesion volume) and explore the mechanism.

IAP40 Wuyun Zhu (RCSI)

"Loss of FKBPL in Glucose Intolerance Links Early Islet Dysregulation to Type 2 Diabetes Pathogenesis"

The authors have chosen not to make the contents of this abstract available online

Friday, 14th November 2025

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