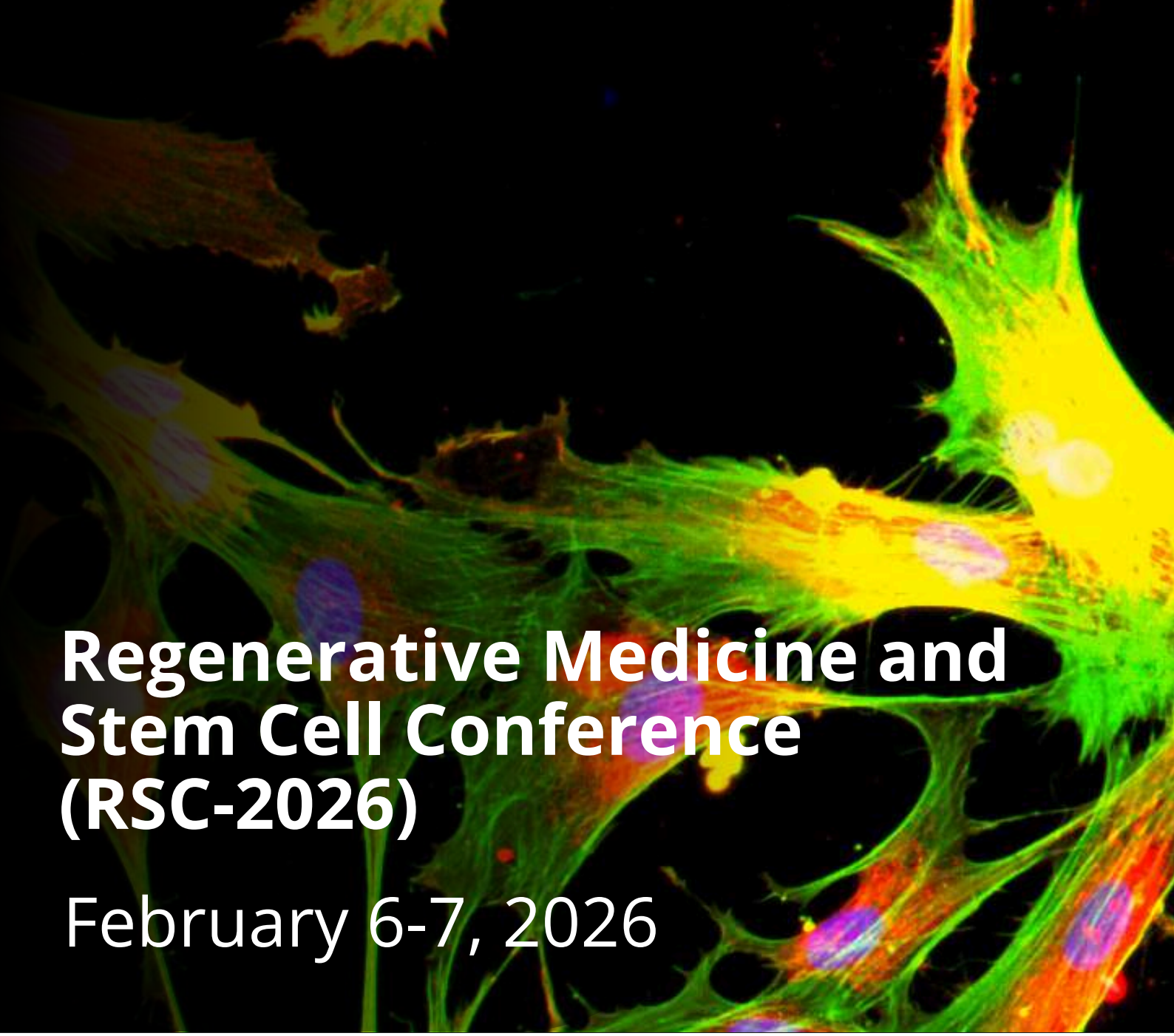

Regenerative Medicine and Stem Cell Conference (RSC-2026)

February 6-7, 2026

CONFERENCE GUIDE

THE AGA KHAN UNIVERSITY
CENTRE FOR REGENERATIVE MEDICINE
AND STEM CELL RESEARCH

ABOUT RSC-2026

The Regenerative Medicine and Stem Cell Conference 2026 (RSC-2026) aims to bring together scientists, clinicians, and policymakers to discuss recent advances in stem cells, regenerative medicine, and tissue engineering. This conference will focus on innovations and opportunities specifically tailored to the needs of low- and middle-income countries (LMICs), with a particular emphasis on Pakistan.

What to expect at RSC-2026

- Cutting-edge sessions showcasing research and translational advances relevant to LMICs.
- Engaging in dialogues on ethical, regulatory, and clinical translation challenges in resource-limited settings.
- Collaborative opportunities for formation of interdisciplinary and multicentre partnerships amongst academia, clinicians, industry, and policymakers.
- Forums to showcase research and engage in knowledge exchange with a diverse audience.
- Hands-on workshops providing exposure to advanced scientific techniques.

Who should attend

Researchers, Clinicians, Physician-Scientists, Biomedical Engineers, Postdoctoral Fellows, MPhil/PhD Students, Undergraduate Students, Early-Career Investigators, Industry Representatives, and Members of Regulatory Bodies.

For more information, please visit the event website: www.aku.edu/events/rsc-conf/
or contact:

Centre for Regenerative Medicine and Stem Cell Research,
Aga Khan University, Karachi, Pakistan
Tel:+92 21 3486 5557/4414
Email: crm.events@aku.edu

WELCOME MESSAGE CONFERENCE CHAIR

“

It is my distinct privilege to welcome you to RSC 2026, the first national conference of its kind in Pakistan dedicated exclusively to Regenerative Medicine and Stem Cell Research, taking place on 6–7 February 2026, preceded by hands-on workshops from 2–5 February 2026.

Regenerative medicine is among the most rapidly advancing frontiers of modern biomedical science. Across the world, it is reshaping how we think about disease, repair, and recovery, offering realistic hope for therapies that restore function rather than merely manage symptoms. For countries like Pakistan—where the disease burden is high and healthcare systems face persistent constraints—these advances carry particular significance. Thoughtfully adapted, they have the potential to create meaningful, scalable impact for patients who need it most.

RSC 2026 has been conceived as a national platform to reflect on where we stand, to learn from global experience, and to collectively chart a realistic and responsible path forward. The conference brings together an outstanding group of scientists and clinicians, both from Pakistan and abroad, whose work spans fundamental discovery, translational research, and early clinical application. Their commitment, preparation, and hard work have been central to making this meeting possible.

I see this conference as more than a series of lectures. It is an opportunity for cross-fertilization of ideas, for honest discussion of challenges, and for nurturing collaborations that bridge the laboratory and the clinic. Our aim is to strengthen the bench-to-bedside pathway and to ensure that scientific progress translates into tangible benefits for patients and society.

I warmly welcome all participants who share our commitment to advancing science, improving healthcare, and transforming lives through innovation, collaboration, and purpose.

Professor Syed Ather Enam, SI
Conference Chair

WELCOME MESSAGE CONFERENCE CO-CHAIR

Welcome to RSC-2026 entitled “Innovating today for the medicine of tomorrow” that is being held from 6-7 February 2026 at Aga Khan University. This conference is being hosted by the AKU-CRM and promises enlightening and thought-provoking keynote addresses, lectures and oral presentations from experts of stem cell research across the globe.

The progress of regenerative medicine and stem cell research relies on meaningful collaboration across disciplines and institutions, both nationally and internationally. RSC-2026 is envisioned as a platform where scientists, clinicians, educators, and policy leaders can come together to exchange ideas and critically examine the direction of the field in Pakistan. Therefore, this conference is focused in nurturing these collaborations as well as focusing on diseases that are more prevalent in our region.

This conference emphasizes dialogue, between basic science and clinical practice, innovation and ethics as well as discovery and delivery. It also seeks to cultivate a culture of shared learning and partnership, particularly for early-career researchers and trainees. We look forward to insightful discussions that will strengthen the scientific community and contribute to sustainable progress in regenerative medicine.

Finally, we hope that this conference benefits scientists, researchers, and students in not only understanding the impact of stem cell research in the future of managing many non-communicable diseases but also develops long lasting and meaningful collaborations between institutions, researchers and scientists leading to many success stories for the future.

Regards,

Dr Azhar Hussain,
Conference Co-Chair

WELCOME MESSAGE CHAIR ORGANIZING COMMITTEE

“

It is my great pleasure to welcome you to RSC-2026 being organized by AKU-CRM.
Regenerative medicine and stem cell science are undergoing rapid conceptual and technological evolution. Advances in stem cell biology, genome editing, single-cell genomics, and cell-based therapeutics are redefining our understanding of development, disease mechanisms, and tissue repair. These developments are raising both unprecedented opportunities and important scientific, ethical, and regulatory questions.

RSC 2026 has been conceived as a scientific forum to examine these advances across the continuum of discovery, translation, and clinical application. The program brings together fundamental and translational research spanning stem cell biology, genetics and genomics, gene and cell therapy, regenerative strategies, and therapeutic development.

A core objective of this meeting is to promote collaboration, mentorship, and knowledge exchange. By convening international and national experts alongside clinician-scientists, early-career investigators, and trainees, we seek to strengthen research capacity, encourage methodological rigor, and foster partnerships that are particularly relevant to health challenges in low- and middle-income country settings.

I am sincerely grateful to our distinguished speakers, session chairs and co chairs, organizing committee members, and institutional leadership for their commitment to this conference. I also thank all participants for contributing their scholarship and engagement to what we hope will be a stimulating and impactful scientific exchange. I would like to express my sincerest appreciation to the members of the CRM team, for their meticulous planning, scientific insight, and sustained efforts in bringing this conference together.

I warmly welcome you to RSC-2026 and look forward to productive discussions and meaningful collaborations.

Dr Sheerien Rajput
Chair Organizing Committee

MEET THE SPEAKERS

Afsar Mian, PhD
Associate Professor,
AKU-CRM

Dr Mian is a leading researcher in drug and peptide therapies for treatment-resistant leukemia. He began his career at the University of Frankfurt, Germany, contributing to the development of PF-114 (Vamotinib), now in Phase III clinical trials. At AKU-CRM, Dr Mian's team is developing CAR T Cell and peptide-based therapies for leukemia and lymphoma. His group has received major funding, including a World Bank/HEC Grand Challenge Fund and Wellcome Leap grants, to advance CRISPR/Cas9-based treatments for beta-thalassemia and sickle cell anemia.

Asmat Salim, PhD
Professor, Dr Panjwani Center for Molecular Medicine and Drug Research (PCMD), International Center for Chemical and Biological Sciences (ICCBS), University of Karachi

Dr Salim has been working as a basic scientist in the field of stem cell research since 2004. She did her graduation in Biochemistry and a PhD in Protein Chemistry from the University of Karachi. She completed her post-doctoral training at the University of Cincinnati, Ohio, USA, and established the Stem Cell Research Laboratory at PCMD, the first in the province. Her research is focused on the use of stem cells for degenerative disorders, with main emphasis on cardiomyogenesis and wound healing following ischemic injury and burn wounds, and exploring signaling pathways governing the role of stem cells in repair and regeneration. She has completed a number of research projects and published many papers in peer-reviewed journals. She has presented her research at various national and international scientific meetings and won many awards.

MEET THE SPEAKERS

Ambrin Fatima, PhD
Assistant Professor, BBS, AKU

Dr Fatima holds a PhD in Human Molecular Genetics from a joint program between NIBGE, Pakistan, and the University of Copenhagen, Denmark. At AKU, her research centers on the genetic architecture of neurodevelopmental and neuropsychiatric conditions, particularly in consanguineous populations where recessive disorders are highly prevalent. Dr. Fatima places strong emphasis on the functional validation of identified genes and variants using patient-derived cellular models, including iPSC-derived neurons and brain organoids. She is also deeply committed to improving access to genetic diagnosis, counselling, and precision medicine in underserved communities.

Azhar Hussain, MBBS
Assistant Professor & Director of Research & Teaching Laboratories at MC-PK

Dr Hussain is the Director of Research & Teaching Laboratories at AKU's Medical College, overseeing laboratory operations across AKU campuses. With over 20 years of laboratory management experience, Dr Hussain has led multiple infrastructure upgrades, SOP development, and facility expansions, including current projects at BBS Labs at AKU's Stadium Road campus. His research focuses on cancer biology, particularly dysregulated survival pathways and apoptosis in malignancies such as lymphomas, leukemia, colorectal, thyroid, and ovarian cancers, with publications in leading journals including Blood, Cancer Research, and JCEM.

MEET THE SPEAKERS

Azra Mehmood, PhD
Associate Professor, Centre of Excellence in Molecular Biology, University of Punjab, Lahore, Pakistan;
Team Lead: Stem Cell Therapeutics and Regenerative Medicine Research Group

Dr Mehmood completed her PhD in 2011 from the Centre of Excellence in Molecular Biology (CEMB), Pakistan, with part of her doctoral research conducted at the University of Cincinnati, USA. Her doctoral work focused on the therapeutic potential of endothelial progenitor cells for diabetic heart failure. Since 2018, she has been actively involved in translational research, including clinical trials for skin and ocular regeneration. Her research harnesses adult stem cells from diverse tissues for the regeneration of skin, liver, eye, and pancreas in disease and injury for organ repair. Her work also explores the effects of chemokines, growth factors, antioxidants, co-culture systems, and combinatorial stem cell approaches.

Fawad ur Rehman, PhD
Assistant Professor, AKU-CRM

Dr Rehman holds a PhD in Biomedical Engineering from the Southeast University, Nanjing, China and specialises in nanomedicine and nano-drug delivery systems. His research interest lies in developing Blood-Brain Barrier amenable nanoscale materials-based targeted therapies for brain diseases such as glioblastoma multiforme. At AKU-CRM he is currently leveraging his efforts to harness the potential of lipid nanoparticles and extracellular vesicles for the safe and efficacious delivery of gene editing tools. Before joining CRM, he was a group lead and senior postdoctoral fellow at Henan-MQ International Joint Center for Biomedical Innovation, Henan University, China. He was also a postdoctoral researcher at ZhongDa Hospital, affiliated with the Medical School of Southeast University, Nanjing, China. He also holds proffesional association with the International Society for Extra-cellular Vessicles (ISEV).

MEET THE SPEAKERS

Hammad Hassan, PhD
Assistant Professor, AKU-CRM

With over 15 years of experience in immunology and hematopoiesis, Dr Hassan's research focuses on erythropoiesis in hemoglobinopathies, the development of cancer immunotherapies, and the production of lab-grown red blood cells using immortalized progenitor cell lines, other cell lines, and iPSC models. He has led projects funded by the Pakistan Innovation Fund and the HEC Grand Challenge, and his work has been published in prominent journals such as Nature Immunology and PNAS. Dr Hassan has received the Karl Landsteiner Prize from the Austria society of Immunology. His work bridges foundational science and translational applications to advance therapeutic innovations in hematology and immunology.

Hussain Wahedi, PhD
Associate Professor & HOD Biomedicine the National University of Sciences and Technology (NUST), Islamabad

Dr Wahedi is a molecular cell biologist specializing in regenerative medicine and aging, with extensive expertise in tissue repair, stem-cell–based therapeutics, and molecular mechanisms of cellular rejuvenation. As Associate Professor and Head of the Department of Biomedicine at NUST, his research focuses on identifying molecular and protein targets involved in skin regeneration, wound healing, and age-associated tissue decline. His recent work includes the development of stem-cell–derived extracellular vesicle (exosome)–based scaffolds for enhanced skin repair, as well as efforts in mitochondrial transplantation as a promising strategy to restore cellular bioenergetics in damaged tissues. He has authored several original research articles, secured competitive funding, and filed multiple patents in the area of regenerative medicine and tissue engineering.

MEET THE SPEAKERS

Irfan Khan, PhD
Assistant Professor, AKU-CRM

Dr Khan joined AKU-CRM on a joint appointment with the Department of Ophthalmology & Visual Sciences in March 2024. He has more than 11 years of experience in the field of molecular medicine, stem cells, and tissue engineering research. His research interests focus on understanding and treating retinal and optic nerve degenerative disorders. At AKU-CRM, he is leveraging his efforts to develop a rapid differentiation protocol using a 3D retinal organoid model to investigate signaling and transcription factors in retinal ganglion cells fate specification.

Irshad Hussain, PhD
Professor,
Syed Babar Ali School of Science and Engineering

Dr Hussain is a tenured Professor in the Department of Chemistry & Chemical Engineering at the SBA School of Science & Engineering, LUMS, Pakistan. A founding faculty member of SBASSE, he also served as the founding Chair of the Chemistry Department at LUMS. He founded and directed the Nanobiotech Group at the National Institute for Biotechnology & Genetic Engineering, Faisalabad, and has led several national nanotechnology forums in Pakistan. He currently heads the Functional Nanomaterials Group at LUMS, focusing on nanomaterials for energy, catalysis, biomedical, and environmental applications, with over 185 publications in leading journals. He has chaired national nanotechnology panels, received multiple gold medals, was named a PIFI-Distinguished Scientist in 2024, and holds professorial appointments in China.

MEET THE SPEAKERS

Karim F. Damji, MD, FRCSC, MBA
Professor & Dean, Medical College, and former Inaugural Chair, Department of Ophthalmology & Visual Sciences, AKU

Dr Damji completed medical school at the University of British Columbia (1987), followed by fellowships in ocular pathology, ophthalmic genetics, and glaucoma at UBC and Duke University, and an Executive MBA from Queen's University. He served as a glaucoma specialist and clinician scientist at the University of Ottawa (1996–2008) and as Professor at the University of Alberta (2008–2021), including Chair of Ophthalmology and Visual Sciences (2014–2021). He has led residency programs at both universities and served as President of the Canadian Glaucoma Society and CAUPO. His research focuses on stem cell therapy, teleglaucoma, SLT, MIGS, and glaucoma capacity-building in Sub-Saharan Africa. He has authored over 190 publications, is a senior editor of the Shields Textbook of Glaucoma, and has received multiple awards, including the 2017 UBC Alumni Global Citizenship Award.

Kulsoom Ghias, PhD, SHFEA
The Feerasta Family Endowed Chair & Professor
Department of Biological and Biomedical Sciences

Dr Ghias chairs the Department of Biological and Biomedical Sciences at Aga Khan University (AKU) in Karachi, Pakistan. She completed her PhD and post-doctoral training at Northwestern University's Robert H. Lurie Comprehensive Cancer Center in Chicago, IL, USA. She received her Advanced Diploma in Health Professions Education from AKU and is a Senior Fellow of Advance HE. Her grant-funded research focuses on the onset and progression of solid tumours, and the development of chemoresistance, including the role of viruses and the immune microenvironment. She has supervised post-doctoral trainees and graduate students in the area of cancer biology, bioethics and medical education.

MEET THE SPEAKERS

Luis Pereira de Almeida, PhD
Professor, Faculty of Pharmacy, University of Coimbra, Portugal

Dr Pereira de Almeida is a tenured professor at the Faculty of Pharmacy, University of Coimbra, coordinating the Master's in Pharmaceutical Biotechnology. He conducts research at CNC–Center for Neuroscience and Cell Biology, where he is Principal Investigator and Vice-President, and also leads the Center for Innovative Biomedicine and Biotechnology (CIBB). He earned his PhD at the Gene Therapy Center of Lausanne (CHUV, Switzerland) and completed sabbaticals at CEA, France, and MIT. Former Vice-President of the Portuguese Society for Stem Cells and Cell Therapy, Dr. Luís leads research on gene and stem cell therapies for brain disorders, especially Machado–Joseph disease, with 90+ publications and coordination of over 20 projects, including ViraVector and the JPND ModelPolyQ initiative.

Mansoor Saleh, MD
Professor & Chair
Department of Haematology-oncology, AKUH Kenya

Dr Saleh is the Founding Chair in the Department of Hematology and Oncology and the Founding Director of the Cancer Centre at the Aga Khan University, Nairobi. He received his medical degree from the University of Heidelberg in Germany and conducted his doctoral research at the Max Planck Institute for Medical Research in Heidelberg. From there, Dr Saleh completed his clinical and translational research training in Hematology and Oncology at the University of Alabama Comprehensive Cancer Centre, where he was a tenured Professor of Medicine and Pathology and Director of the First-in-Human Early Drug Development Programme. His area of research and clinical focus is targeted therapy of cancer.

MEET THE SPEAKERS

Muhammad Usman Rashid, PhD, MBBS, FRCPath, UICC Fellow
Head, Basic Sciences Research Department, SKMCH

Dr Rashid is an internationally recognised physician-scientist and human geneticist with over 24 years of experience in translational genomics and precision medicine. He currently serves as Senior Research Scientist and Head of Basic Sciences Research at Shaukat Khanum Memorial Cancer Hospital & Research Centre, where he founded Pakistan's first comprehensive translational cancer genetics programme. His work bridges genomic discovery, functional validation, and clinical translation, with a strong focus on regionally relevant diseases. Professor Rashid has led large international collaborations, published extensively, and brings deep expertise relevant to regenerative and genomic medicine.

Raza Shah, PhD
Professor & Director ICCBS, University of Karachi & Chair UNESCO Bio-organic and Natural Product Chemistry

Dr Shah is a full professor at the International Center for Chemical and Biological Sciences, HEJ Research Institute of Chemistry, University of Karachi, Pakistan. He is also the Head of the Center for Bioequivalence Studies and Clinical Research. He is a recipient of several awards, including the Tamgha-i-Imtiaz Award from the President of Pakistan, the Salam Prize, the Professor Atta ur Rahman Gold Medal, and the Dr. M Raziuddin Siddiqi Prize, by the Pakistan Academy of Sciences, for scientists under 40 years of age, in the field of chemistry. Professor Shah has authored six books and edited four books, in addition to contributing over 350 peer-reviewed journal papers. One of his authored books was declared the best book of 2017 by the Government of Pakistan's Higher Education Commission.

MEET THE SPEAKERS

Salim S Virani, MD, PhD
Professor & Vice Provost Research, Nizar E. Noor Mohammed Mewawalla Endowed Professor in Cardiology, Dep. of Population Health, AKU's MC, Kenya

Dr Virani leads Multilateral-Bilateral Relations at AKU and oversees global research and international collaborations across South Asia, Europe, and East Africa in medicine, nursing, education, Muslim civilizations, media, climate change, and environmental science. His research focuses on cardiovascular disease prevention, and he has authored or coauthored over 800 peer-reviewed publications with more than 180,000 citations. Recognised as a World Expert in Cholesterol by Expertscape (top 0.01%, 2013–2023), he is also listed among the World's Top 2% Most-Cited Scientists by Stanford University. In 2024, he was among the 25 AKU faculty recognised for both single-year and career-long impact, ranking among Pakistan's top 10 researchers. He has served as an author and Chair of major U.S. and European preventive cardiology guidelines.

Salman Kirmani, MBBS, DABMGG, DABP
Professor, Paediatrics & Medicine, Director, Centre of Excellence – Women & Child Health, Parveen Kanji Irshad-ud-din Endowed Chair, WCH, Interim Director, Human Development Program

Dr Kirmani is a Professor at the Aga Khan University, where he serves as Chair of the Division of Women & Child Health and Global Director of the Centre of Excellence in Women & Child Health. A Consultant Medical Geneticist and Paediatric Endocrinologist, his work focuses on rare diseases, neurogenetics, and precision genomics in highly consanguineous and under-represented populations. He trained and worked for over a decade at the Mayo Clinic and holds board certifications in Medical Genetics and Pediatrics in the United States.

MEET THE SPEAKERS

Sami Siraj, PhD
Associate Professor and Director at the Institute of Pharmaceutical Sciences, Khyber Medical University, Peshawar

Dr Siraj holds a PhD in Anticancer Pharmacology from China Pharmaceutical University and completed postdoctoral training in Cancer Cell Biology at the Chinese Academy of Sciences, Beijing. His academic background also includes an MPhil in Genetic Diseases from the University of the Punjab and a Bachelor of Pharmacy from Gomal University. Dr. Siraj has over two decades of experience spanning academia, biomedical research, and pharmaceutical production. His research focuses on anticancer pharmacology, pharmacogenetics, mitophagy, and genetic associations in human disease. He has published in high-impact international journals and actively contributes to curriculum development, scientific workshops, and national and international conferences.

Shaheen Sayed, PhD
Associate Professor & Consultant - Anatomical Pathology and Cytology, Chair, Department of Pathology & Laboratory Medicine, AKU-MC, East Africa

Dr Sayed holds an MBChB and MMed (Pathology) from the University of Nairobi, a FCPath (ECSA), a Fellowship in Nephropathology from the Academic Medical Center, Amsterdam, and a PhD in Anatomical Pathology from the University of Cape Town. She is a Fellow of the Royal College of Pathologists (UK) and serves as the RCPath Country Advisor for Kenya, as well as an International Advisor to the ASCP Global Pathology Committee.

She is a global leader in pathology and oncology, serving on WHO tumour boards and Lancet Commissions. She leads the first clinically annotated biorepository in Kenya, advancing translational research, AI-driven diagnostics, and biomarker studies, while training pathologists and strengthening research capacity across Sub-Saharan Africa.

MEET THE SPEAKERS

Syed Ather Enam, SI., MD., PHD
Professor of Surgery,
Sakarkhanum & Hussain Ebrahim Family Chair & Director of CRM

Dr Enam is the Director of the AKU's Centre for Regenerative Medicine and Stem Cell Research. He is a U.S. Board-certified Neurosurgeon and Professor of Neurosurgery. He also serves as Director of the Center of Oncological Research in Surgery and Scientific Director of Juma Research Laboratories at AKU. He has an FRCS in Neurosurgery from Canada, an FRCS from Ireland, an FRCS from Glasgow (U.K.), and a Fellowship of the American College of Surgeons. He was Chair, Dept of Surgery at AKU for more than 7 years, and before that led the Section of Neurosurgery for 7 years.
Dr Enam has a strong interest in basic science research with a PhD in Neuroscience from Northwestern University Institute of Neuroscience, USA.

Sheerien Rajput, PhD
Assistant Professor, AKU-CRM

Dr Rajput is a cell and molecular biologist and an Assistant Professor at the Centre for Regenerative Medicine and Stem Cell Research, AKU. Her work focuses on the biology of stem cells, cancer stem cells and microenvironments to understand tissue regeneration, disease heterogeneity and therapeutic resistance in cancers. Dr Rajput completed her PhD at the Faculty of Health Sciences, Aga Khan University, where she investigated the crosstalk between Mucin 1 (MUC1) and X-box binding protein 1 (XBP1) in cancers. During her doctoral work, she established Pakistan's first myeloma cell line, AKUMY01, derived from bone marrow.

MEET THE SPEAKERS

Tim Beissert, PhD
Group Lead, Vector Development & Gene Transfer, TRON/UMC Mainz

Dr Beissert studied biology at the Technical University Darmstadt, Germany, where he received his diploma in 1999. Next he did his PhD thesis at the University Clinic Frankfurt, Germany, where he investigated the mechanism of resistance of Philadelphia-chromosome positive leukemia towards Imatinib. After continuing in the field of leukemia as a postdoc for 4 years, he changed his field of research in 2008 by joining the lab of Prof. Dr. Ugur Sahin in Mainz, Germany. Since then, Dr Tim Beissert is working on the improvement of RNA vectors, and since 2010 he is heading the Vectors team at the Mainz-based research Institute TRON. Dr. Tim Beissert and his team have been developing and improving self- and trans-amplifying RNA for its use as a vaccine platform.

Zahra Hasan, PhD
Gulamali Hirji Endowed Professor, Section of Molecular Pathology, Department of Pathology and Laboratory Medicine, AKU

Dr Hasan is a Consultant at the Section of Molecular Pathology, Department of Pathology and Laboratory Medicine, and Director of the Faculty of Health Sciences PhD Program, a Fellow of the Pakistan Academy of Sciences. At the Clinical Laboratories, she works in molecular diagnostics across different disciplines, including genetics and genomics. She believes in the scope for basic science research translated to clinical disease diagnostics and management. Her research has focused on both pathogen and host aspects of disease, identifying biomarkers associated with diseases like COVID-19 and TB. The impact of diabetes is of particular interest in her studies examining disease-associated risk in the population.

Organzing Committee

Conference Chair
Professor Ather Enam, SI

Conference Co-Chair
Dr. Azhar Hussain

Organizing Committee
Chair: Dr Sheerien Rajput
Members:
Ms Sadia Habib
Ms Yasmeen Mughal
Ms Noorjehan Amir Ali
Mr Sujjawal Ahmad

Communication & Promotion Committee
Chair: Dr Ambrin Fatima
Members:
Mr Sujjawal Ahmad
Ms Yasmeen Mughal

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Dr Irfan Khan
Members:
Ms Sadia Habib
Mr Areeb Ahmed
Mr Sujjawal Ahmad
Ms Yasmeen Mughal
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Scientific Committee
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Dr Ather Enam
Dr Azhar Hussain
Dr Hammad Hassan
Dr Irfan Khan
Dr Fawad Ur Rehman
Dr Ambrin Fatima
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Dr Sheerien Rajput
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Dr Ayaz Khan
Mr Anwar Alam
Ms. Susheel Fatima
Ms Tooba Tajammul
Ms Aafia Shahid
Ms Ayesha Fatima
Ms Seema Inayat
Ms Shariqa Khawaja
Ms Maliha Javed
Ms Safana Farooq
Mr Sujjawal Ahmad
Ms Sahar Arshad

Plenary Session
Chair: Dr Hammad Hassan
Members:
Dr Sheerien Rajput
Ms Fatima Iqbal
Ms Noor Fatima
Ms Zarmeen Suhail

Abstract Review Committee
Chair: Dr Satwat Hashmi
Members:
Dr Ambreen Surti
Dr Saadia Saad,
Ms Zainab Saleem
Dr Shafaq Ramzan

Workshop Committee
Chair: Dr Fawad Ur Rehman
Members:
Dr Afsar Mian
Dr Ambrin Fatima
Dr Irfan Khan
Dr Fawad Ur Rehman
Dr Rida e Maria Qazi
Dr Rabbia Muneer
Dr Hammad Yousaf
Mr Shariqa Khawaja
Mr Sadia Habib
Mr Areeb Ahmed
Ms Noorjehan Amir Ali
Dr Sidrah Shams
Dr Ayaz Khan
Mr Asif Shah
Ms Seema Inayat
Mr Anwar Alam
Ms Susheel Fatima
Ms Maliha Javed
Ms Safana Farooq
Ms Shagufta Naz
Ms Kainat Ahmed
Ms Sahar Arshad
Ms Lubaba Binte Khalid
Mr Ifham Ali
Ms Ayesha
Ms Reena Zaman
Ms Perveen Bano
Ms Sahida Bano
Mr Alan Nazir
Ms Aafia Shahid
Mr Zohaib
Mr Sajid Ali
Mr Areeb Masood
Mr Tariq Wasim
Mr Mohid Qazi
Ms Fatima Iqbal
Ms Zarmeen Suhail
Ms Zainab Saleem
Ms Noor Fatima

CONFERENCE PROGRAMME

PRE-CONFERENCE CME ACTIVITIES
FEBRUARY 2-5, 2026
VENUE: AKU CENTRE FOR REGENERATIVE MEDICINE AND STEM CELL RESEARCH, DR PARVEEN KANJI LABORATORY, JUMA BUILDING FIRST FLOOR

Workshop	Activity Director(s)
WORKSHOP 1 Cell and Gene Therapy: Research and Applications Date: February 2-3, 2026 Time: 9:00am to 5:00pm	Dr Afsar Mian Dr Ambrin Fatima Dr Irfan Khan Dr Fawad Ur Rehman
WORKSHOP 2 CAR-T Cell Therapy: Prospects of Design and Application Date: February 4, 2026 Time: 9:00am to 5:00pm	Dr Hammad Hassan
WORKSHOP 3 The Art and Science of 3D Bioprinting Date: February 5, 2026 Time: 9:00am to 5:00pm	Dr Sheerien Rajput

CONFERENCE PROGRAMME
DAY 1 – FRIDAY, FEBRUARY 6, 2026
VENUE: AKU AUDITORIUM

Time	Duration	Session / Proceedings	Speaker
08:00 am – 09:00 am	60 min	Registration	-
09:00 am – 09:05 am	5 min	Welcome & Introduction	-
Theme 1: Stem Cells – Bench to Bedside			
09:05 am -10:50 am (Pakistan Time; GMT +5)			
Chair: Professor Ather Enam | Co-Chair: Dr Afsar Mian			
09:05 am – 09:30 am	25 min	Keynote Address **"Reimagining Glaucoma Care: From Preventing Damage to Restoring Vision"**	Prof. Karim F. Damji, Dean MC, AKU
09:30 am – 09:50 am	20 min	Plenary Session **"Stem Cells Mediated Tissue Repair and Regeneration"**	Dr Azra Mehmood, CEMB, UoP
09:50 am – 10:05 am	15 min	Thematic Talk: **"Blood in a Dish: A Step Towards Manufactured Blood Products"**	Dr Hammad Hassan, CRM, AKU
10:05 am – 10:40 am	35 min	Oral Talks	
		Transplantation of human Amniotic Epithelial Stem Cells (hAESCs) from human amniotic membrane attenuates liver fibrosis in vivo	Aliya Jabeen, NUST, Islamabad
		Directed Differentiation of iPSCs into Retinal Ganglion Progenitors	Dr Rabbia Muneer, AKU
		Drug Screening on BACE2 Gene Expression on Alzheimer's Disease Patient- Specific Neuronal Cells Derived from Induced Pluripotent Stem Cells	Dr Aniqa Batool, DUHS
10:40 am – 10:50 am	10 min	Distribution of Souvenirs and Certificates	
10:50 am - 11:05 am	15 min	Tea Break (Venue: Male Hostel Garden Area)	
11:05 am - 11:25 am	20 min	Poster Evaluation Session (Venue: Medical College Courtyard)	

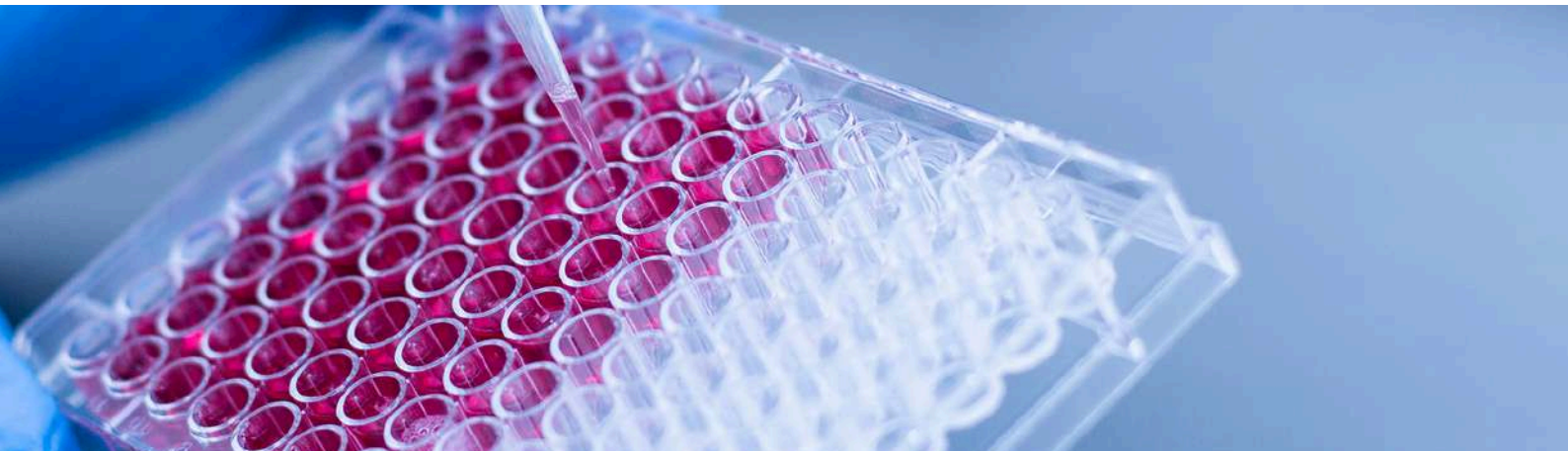
Time	Duration	Session / Proceedings	Speaker
Theme 2: Regenerative Medicine & Tissue Engineering			
11:30 am – 01:25 pm (Pakistan Time; GMT +5)			
Chair: Dr Azhar Hussain | Co-Chair: Dr Hammad Hassan			
11:30 am – 12:15 pm	45 min	Keynote **"RNA-Based Gene Transfer and Application"**	Dr Tim Beissert TRON/UMC Mainz
12:15 pm – 12:40 pm	25 min	Keynote Address **"Preconditioning Mesenchymal Stem Cells with Small Molecules: Strategies for Improved Cardiac Regeneration"**	Professor. Asmat Saleem PCMD, ICCBS, KU
12:40 pm – 1:00 pm	20 min	Plenary Session **"Stem-Cell derived Exosomes: An Emerging Therapeutic Strategy for Skin Regeneration and Aging"**	Dr. Hussain Wahedi ASAB, NUST
01:00 pm – 01:15 pm	15 min	Thematic Talk **"Translating Stem Cell-Derived Extracellular Vesicles for Ophthalmic Regeneration: A Preclinical Study"**	Dr Irfan Khan CRM, AKU
01:15 pm - 01:25 pm	10 min	Distribution of Souvenirs and Certificates	
01:25 pm - 02:30 pm	65 min	Lunch and Juma Break (Venue: Male Hostel Garden Area)	
02:30 pm - 02:50 pm	20 min	Poster Evaluation Session (Venue: Medical College Courtyard)	

Time	Duration	Session / Proceedings	Speaker
Theme 3: Genetics, Genomics & Gene Therapy			
02:55 pm – 05:40 pm (Pakistan Time; GMT +5)			
Chair: Dr Satwat Hashmi | Co-Chair: Dr Irfan Khan			
02:55 pm – 03:20 pm	25 min	Keynote: **“Fulfilling the Precision Medicine Promise in LMICs”**	Professor Salman Kirmani WCH, AKU
03:20 pm – 03:40 pm	20 min	**Plenary Session: "Old Drug, New Hope: Clinical Efficacy and Molecular Insights of Thalidomide in β-Thalassemia"**	Professor Sami Siraj, KMU
03:40 pm – 03:55 pm	15 min	**Thematic Talk: “Using Host Transcriptional Responses to Investigate Pathogen Associated Virulence”**	Professor Zahra Hasan PLM, AKU
03:55 pm – 04:10 pm	15 min	**Thematic Talk: "Gene Editing Therapies for Beta-Thalassemia and Sickle Cell Disease"**	Dr Afsar Mian CRM, AKU
04:10 pm – 04:25 pm	15 min	**Thematic Talk: “The Pakistani Variome: Unlocking the Genetics of Neurodevelopmental Disorders”**	Dr Ambrin Fatima BBS, AKU
04:25 pm - 05:00 pm	35 min	Oral Talks	
		Integrated Clinical, Genetic, and Functional Approaches Diagnose Eight Limb-Girdle Muscular Dystrophy Families and identify Four Novel Variants	Dr Hammad Yousaf, AKU
		Individuals with latent Mycobacterium tuberculosis infection and diabetes display increased inflammatory cytokines, downregulated immunity and dysregulation of metabolic pathways	Dr Kiran Iqbal, AKU
		Investigating Clinical Outcomes in Post-Renal Transplant Recipients Receiving Tacrolimus: A Pharmacogenomic Perspective in the Pakistani Population	Dr Tooba Noor, DUHS

Time	Duration	Session / Proceedings	Speaker
05:00 pm - 05:30 pm	30 min	**Panel Discussion:** Moderators: Professor Salman Kirmani & Professor Natasha Ali	
05:30 pm – 05:40 pm	10 min	Distribution of Souvenirs and Certificates	

INAUGURAL SESSION
FRIDAY, FEBRUARY 6, 2026 | 06:00 PM – 09:00 PM
VENUE: AKU AUDITORIUM

Time	Duration	Session / Proceedings	Lead / Speaker
06:00 pm – 06:05 pm	5 min	**Tilawat & National Anthem**	
06:05 pm – 06:15 pm	10 min	Welcome Remarks & Conference Overview by Professor Ather Enam, Director, AKU-CRM	
06:15 pm – 06:25 pm	10 min	Address by Dean Medical College, AKU, Professor Karim F. Damji	
06:25 pm – 06:35 pm	10 min	Address by Vice Provost, Research, AKU, Professor Salim S. Virani	
06:35 pm – 06:55 pm	20 min	Guest Address by Professor Luis Pereira de Almeida, University of Coimbra	
06:55 pm – 07:15 pm	20 min	Guest Address by Dr Tim Beissert, TRON/UMC Mainz	
07:15 pm – 07:25 pm	10 min	Address by Chair, Department of Biological and Biomedical Sciences, AKU, Professor Kulsoom Ghias	
07:25 pm onwards	Dinner Venue: Poolside, AKU Sports & Rehabilitation Centre		



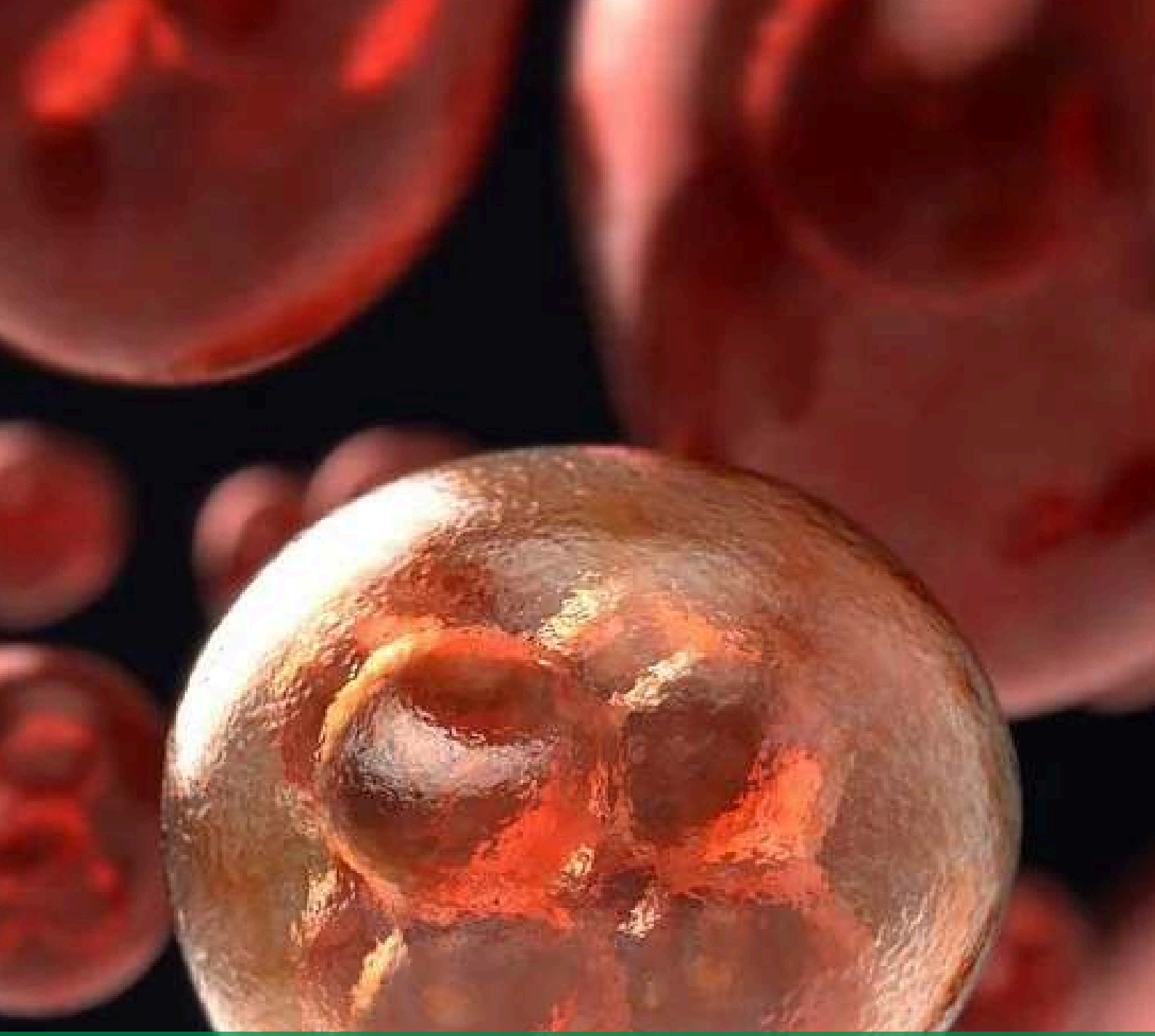
DAY 2 – SATURDAY, FEBRUARY 7, 2026
AKU AUDITORIUM

Time	Duration	Session / Proceedings	Lead / Speaker
Theme 4: Cancer Biology & Precision Medicine			
09:30 am -12:30 pm (Pakistan Time; GMT +5)			
Chair: Professor Natasha Ali | Co-Chair: Dr Sheerien Rajput			
09:30 am – 10:15 am	45 min	**Keynote Address** **"Beyond Repair: Gene Therapy"**	Professor Luis Pereira de Almeida, University of Coimbra
10:15 am – 10:40 am	25 min	***Keynote Address:*** **"Cancer Clinical Trials at the Aga Khan University Hospital Nairobi – Are We Targeting the Cancer Stem Cell?"**	Prof. Mansoor Saleh AKUH, Kenya
10:40 am – 10:55 am	15 min	Tea Break (Venue: Male Hostel Garden Area)	
10:55 am – 11:15 am	20 min	***Plenary Session:*** **"Pioneering Precision Oncology in Pakistan: Nation-Led Large-Scale Genetic Profiling and Its Translational Impact"**	Dr. Muhammad Usman Rashid, SKMCH
11:15 am – 11:30 am	15 min	**Thematic Talk:** **"microRNAs in Brain Tumours: Small Regulators, Large Consequences"**	Prof. Ather Enam, SI, CRM, AKU
11:30 am – 11:45 am	15 min	**Thematic Talk:** **"From Sample to Science: Establishing Biobanks, the AKUHN Experience"**	Dr. Shaheen Sayed AKU-MC, Kenya
11:45 pm – 12:20 pm	35 min	Oral Talks	
		Genomic Landscape and Association of Selected Stem Cell Markers in Pancreatic Ductal Adenocarcinoma	Dr Saleema Mehboob Ali, AKU
		Helix-2–TAT Peptide–Engineered Mesenchymal Stem Cells as a Therapeutic Strategy for Philadelphia Chromosome–Positive Leukemia	Dr Rida-e-Maria Qazi, AKU
		Z-36, a novel Bcl-xL inhibitor, induces oxidative stress mediated non-apoptotic and non-autophagic cell death in colorectal cancer HCT-116 cells	Almuayyad Gajani, AKU

Time	Duration	Session / Proceedings	Lead / Speaker
12:20 pm – 12:30 pm	10 min	Distribution of Souvenirs and Certificates	
12:30 pm- 01:00 pm	30 min	Poster Evaluation Session (Venue: Medical College Courtyard)	
01:00 pm - 02:00 pm	60 min	Lunch and Prayer Break	
Theme 5: Nanomedicine, Extracellular Vesicles & Advanced Drug Delivery			
02:05 pm -04:50 pm (Pakistan Time; GMT +5)			
Chair: Professor Mansoor Saleh | Co-Chair: Dr Shaheen Sayed			
02:05 pm – 02:30 pm	25 min	**Keynote Address "Customised Drug Delivery System"**	Professor Raza Shah HEJ, KU
02:30 pm – 02:50 pm	20 min	**Plenary Session "Functional Nanomaterials - Controlling the Size and Surface Chemistry for Biomedical Applications"**	Professor Dr Irshad Hussain, LUMS
02:50 pm- 03:05 pm	15 min	**Thematic Talk "Exploring Engineered Exosomes and Liposomes- Regenerative and Drug Delivery Potential"**	Dr Fawad Ur Rehman CRM, AKU
03:05 pm – 04:15 pm	70 min	Oral Talks	
		Isolation of CD34+ Hematopoietic Stem Cells using antibody-functionalized Iron Oxide Nanoparticles	Aiman Aslam, AKU
		Comparative Evaluation of Non-Viral Nanocarriers for siRNA and mRNA Delivery in Hematopoietic and Stem Cell Models	Shariqa Khawaja, AKU
		Nanoscale Lipid-Drug Conjugates: An Alternative and Efficient Approach for Drug Delivery	Dr Mujeeb Ur Rehman, ICCBS, University of Karachi

Time	Duration	Session / Proceedings	Lead / Speaker
03:05 pm – 04:15 pm	70 min	Oral Talks	
		Tissue engineered multifunctional chitosan-modified polypropylene hernia mesh loaded with bioactive phyto-extracts	Dr Faiza Sharif, COMSATS University of Islamabad, Lahore Campus
		Three-Dimensional Spatial Organization Modulates Gene Expression in Mesenchymal Stem Cells Cultured on Human Tissue-Derived Scaffold	Faiza Chaudhary, ICCBS, University of Karachi
		Characterization of Human Tissue–Derived Scaffold: MSC Compatibility, Multi-Omics Profiling, and Regenerative Potential	Dr Omair A. Mohiuddin, DUHS
		Optimization of Alginate Capsules to Generate a Hematopoietic Stem Cell Bone Marrow Niche	Shermeen Tanveer, Univeristy of Silesia, Poland
04:15 pm – 04:45 pm	30 min	Panel Discussion, Moderators: Professor Kulsoom Ghias & Dr Munira Moosajee	
04:45 pm – 04:55 pm	10 min	Distribution of Souvenirs & Certificates	
04:55 pm-05:00 pm	05 min	Vote of Thanks	Dr Sheerien Rajput CRM, AKU
05:00- 05:20 pm	Announcement of the Poster Winners and Runner ups; Distribution of Souvenirs and Certificates to Organizers and Facilitators		
5:20-onwards	Tea and Exits (Venue: Male Hostel Garden Area)		

ABSTRACT BOOK



THEME 1: STEM CELLS: FROM BENCH TO BEDSIDE

Abstract ID: 5

Title: Transplantation of human Amniotic Epithelial Stem Cells(hAESCs) from human amniotic membrane attenuates Liver fibrosis in vivo

Authors: Aliya Jabeen, Dr. Hussain Mustatab Wahedi
Affiliation: National University of Sciences and Technology(NUST), Islamabad

Background:
Liver fibrosis is one of the major health challenges, which, if left untreated, leads to cirrhosis and hepatocellular carcinoma (HCC). Human amniotic epithelial stem cells (hAESCs) have antifibrotic and anti-inflammatory effects due to their pluripotent nature and immunomodulatory effects.

Purpose:
This study aimed to check the antifibrotic and anti-inflammatory potential of hAESCs by restoring the CCL4-induced liver fibrosis in a mouse model.

Study Design:
This study was designed to investigate the isolation, proliferation, and characterization of human Amniotic Epithelial Stem Cells (hAESCs) through in vitro experiments. Additionally, an innovative liver fibrosis mouse model was established using carbon tetrachloride (CCL_4) to induce fibrosis. In vivo experiments were then performed to evaluate the antifibrotic potential of hAESC transplantation, assessing their therapeutic effects on liver regeneration.

Methods:
hAESCs were isolated by trypsinization of human amniotic membrane and characterized by measuring the mRNA expression of E-cad, Nanog. The expression of CD105 was also assessed to confirm the negligible presence of mesenchymal stem cells. For transplantation, hAESCs were administered via the intravenous route. To assess liver function, serum levels of biomarkers such as ALT, ALP, and Bilirubin were measured. The anti-inflammatory (IL-6 and IL-10) and the antifibrotic effect (α-SMA and TGF-β1) of hAESCs were measured by checking the mRNA expression levels. Morphological changes and collagen deposition in the fibrotic liver were evaluated through histopathological analysis to confirm the therapeutic effects of hAESC transplantation on liver fibrosis.

Results:
The higher mRNA expression of E-cadherin confirmed that the majority of the isolated cells were hAESCs. Transplantation of hAESCs resulted in a significant improvement in liver function, as indicated by the enhanced serum levels of ALT, ALP, and Bilirubin. Additionally, hAESCs transplantation downregulated the expression of IL-6, α-SMA, and TGF-β1, while upregulating the expression of IL-10, suggesting a potent anti-inflammatory and antifibrotic effect. Histological analysis revealed that hAESCs transplantation reduced collagen deposition, notably restoring the hepatic architecture and alleviating hepatocyte necrosis.

Conclusion:
hAESCs' Transplantation ameliorates liver fibrosis by modulating the serum level of liver function biomarkers, the expression of cytokines, and profibrotic gene expression. These findings indicate that hAESCs have therapeutic potential for treating liver fibrosis.

Abstract ID: 12
Title: Directed Differentiation of iPSCs into Retinal Ganglion Progenitors
Authors: Rabbia Muneer, Karim Damji, Irfan Khan.
Affiliation: Aga Khan University, Karachi

Background:
Human induced pluripotent stem cells (iPSCs) provide a well-established model to study retinal development and neurodegeneration. Retinal diseases such as glaucoma and age-related macular degeneration lead to progressive loss of neurons and vision impairment, emphasizing the need for effective regenerative strategies. Differentiation of iPSCs into retinal neuronal lineages enables investigation of retinal ganglion cell biology and disease mechanisms. This study focuses on inducing neuronal differentiation from human iPSCs and confirming lineage commitment through morphological, molecular, and immunocytochemical characterization.

Objective:
To differentiate human iPSCs into retinal ganglion progenitor cells (RGPCs) in vitro and validate lineage specification through morphology, immunocytochemistry, and gene expression profiling.

Methods:
Human iPSCs were maintained in Essential 8 medium and subsequently differentiated using a neuronal induction medium for 30 days. Morphological changes were monitored at regular intervals by phase-contrast microscopy. Immunocytochemistry was performed using antibodies against RBPMS, NF-H, Nestin, ISL-1, and GFAP to confirm the expression of retinal progenitor and neuronal markers. In parallel, RNA was isolated and subjected to qPCR to analyse germ layer markers (ectoderm, mesoderm, endoderm), neuronal transcription factors (ASCL1, NeuroD1, NeuroG2), and the pluripotency-associated marker THY1 (CD90). Gene expression was normalized to GAPDH, and relative fold changes were calculated using the 2^–ΔΔCt method. Experiments were performed in triplicate, and data presented as mean ± SD.

Results:
The differentiated cells exhibited morphological features characteristic of RGPC. Immunocytochemistry confirmed robust expression of RGPC markers RBPMS, NF-H, Nestin, ISL-1, and GFAP, while qPCR analysis demonstrated significant upregulation of ectodermal markers and early retinal transcription factors, including ASCL1 ($p < 0.01$), NeuroD1, and NeuroG2 ($p < 0.001$). In contrast, pluripotency-associated genes such as THY1 (CD90) were significantly downregulated ($p < 0.001$). These findings indicate efficient lineage commitment of iPSCs toward RGPCs in vitro.

Conclusion:
This study demonstrates the successful in vitro differentiation of human iPSCs into RGPCs. The generated RGPCs provide a valuable platform for studying retinal development, modelling retinal diseases, and may serve as a potential source for future cell-based therapies for retinal degeneration.

Abstract ID: 6
Title: Drug Screening on BACE2 Gene Expression on Alzheimer's Disease Patient-Specific Neuronal Cells Derived from Induced Pluripotent Stem Cells
Authors: Aniqa Batool, Mohsin Wahid, Naila Shahbaz, Sumbul Shamim, Osama Shahid
Affiliation: Dow University of Health Sciences, Karachi

Objective:
Gene expression-based drug screening for Alzheimer's disease (AD).

Methods:
Peripheral blood cells collected from an Alzheimer's disease (AD) patient was reprogrammed into induced pluripotent stem cells (iPSCs) using an episomal plasmid. The iPSCs were differentiated into neural stem cells, and these were directed towards neuronal cells using specified media and growth factors. Cells were characterized using immunofluorescence for protein expression of specified markers. These invitro neuronal cells were used to screen drugs for AD by investigating six different pharmacological drugs, including amlodipine, candesartan, empagliflozin, metformin, memantine, and rosuvastatin, on beta-amyloid converting enzyme 2 (BACE2) gene expression using quantitative real-time polymerase chain reaction (qPCR).

Results:
The one-way ANOVA revealed a significant difference in BACE2 expression, $P < 0.001$. The Tukey test showed that empagliflozin ($P < 0.001$) was statistically significant. qPCR revealed upregulation of the BACE2 gene by metformin and rosuvastatin, although it was statistically nonsignificant. In contrast, the BACE2 gene was downregulated with amlodipine, candesartan, metformin, and memantine treatment.

Conclusion:
Empagliflozin has been shown to have a statistically significant effect on BACE2 gene expression, indicating its potential as a promising therapeutic agent for AD, although more studies would be required to explore its potential further.

Abstract ID: 7
Title: Effects of ROCK inhibitor Y-27632 on BACE2 gene expression in neuronal cells derived from Alzheimer's patients
Authors: Aniqa Batool, Mohsin Wahid
Affiliation: Dow University of Health Sciences, Karachi

Objective:
This study aims to investigate the effects of the Rho-associated coiled-coil containing protein kinase (ROCK) inhibitor Y-27632 on beta-amyloid converting enzyme 2 (BACE2) gene expression in neuronal cells derived from Alzheimer's patients. The findings suggest that Y-27632 may offer a potential novel therapeutic strategy for Alzheimer's disease.

Materials and Methods:
Episomal reprogramming of peripheral blood mononuclear cells generated induced pluripotent stem cells (iPSCs). The iPSCs were differentiated into neural stem cells using neuronal-specific media and growth factors, further directed toward neuronal cells. Cells were characterized using immunofluorescence for protein expression of specific markers for iPSCs (TRA-160 and OCT-4), Neural Stem Cells (NESTIN and SOX-2), and neuronal cells (MAP-2). These invitro-derived neuronal cells were used to screen the effect of ROCK inhibitor Y-27632, and BACE2 gene expression was measured via quantitative real-time polymerase chain reaction (qPCR).

Results:
qPCR showed significant upregulation of BACE2 gene expression upon Y-27632 treatment. The data were normalized using the TBP housekeeping gene, and an independent sample t-test confirmed a statistically significant increase in BACE2 expression ($P < 0.05$).

Conclusion:
The study suggests ROCK inhibitor Y-27632 may serve as a neuroprotective agent by enhancing BACE2 expression, underscoring its potential in personalized treatment strategies for Alzheimer's disease.

Abstract ID: 60
Title: Antioxidant Effect of Patuletin on Oxidative Stress Damage in Human Mesenchymal Stem Cells.
Authors: Erum Munir, Nazia Ahmed, Sana Eijaz & Nasirullah
Affiliation: University of Karachi

Background:
Stem cells are pluripotent cells found in various tissues, with the ability to self-renew and differentiate into multiple lineages. Patuletin, a rare flavonol within the flavonoid family, exhibits strong antioxidant activity through free radical scavenging. This study investigated the antioxidant and anti-apoptotic effects of patuletin on mesenchymal stem cells (MSCs) derived from human umbilical cord tissue under oxidative stress. Its influence on the proliferation of stressed MSCs was also evaluated.

Methods:
Oxidative stress was induced using hydrogen peroxide (H_2O_2). Cell viability and proliferation were determined using the MTT assay. RNA was extracted with the Gene GET RNA Purification Kit, and real-time PCR was performed to analyze the expression of SOD (chromosome 21), HIF1α (chromosome 14), P53 (chromosome 17), PCNA (chromosome 20), and GPX1 (chromosome 3). Protein expression levels of Ki-67 and P53 were quantified by flow cytometry.

Results:
Patuletin treatment significantly improved MSC proliferation and antioxidant activity while reducing apoptosis in cells exposed to oxidative stress. Gene expression analysis revealed up -regulation of antioxidant and proliferation-associated markers, along with suppression of pro-apoptotic genes.

Conclusion:
Patuletin enhanced the survival, proliferation, and antioxidant capacity of human umbilical cord-derived MSCs under oxidative stress, suggesting its potential as a protective agent in stem cell-based therapies

Abstract ID: 16
Title: Anti-fibrotic Efficacy of Adipose Tissue Derived Mesenchymal Stem Cells in Preclinical Models of Pulmonary Fibrosis: A Systematic Review and Meta-Analysis
Authors: Hisaal Jabeen, Malayka Ali, Fariha Anum, Aisha Ishaque
Affiliation: Ziauddin Universilty

Background and Objectives:
Pulmonary fibrosis (PF) is a chronic progressive lung disease characterized by excessive extracellular matrix deposition and irreversible scarring, for which current antifibrotic therapies only slow disease progression. Adipose-derived mesenchymal stem cells (AD-MSC) possess immunomodulatory and regenerative properties, but their overall antifibrotic effectiveness in preclinical models remains unclear. This review aimed to evaluate the antifibrotic efficacy of AD-MSC-based interventions and their derivatives in preclinical models of pulmonary fibrosis.

Methods:
The review was prospectively registered in PROSPERO (CRD420251119193). Literature searches were conducted in Google Scholar, PubMed, and ScienceDirect up to April 2025 using structured Boolean strings. Eligible studies included original, peer-reviewed experimental investigations assessing MSC-based therapies in idiopathic pulmonary fibrosis, silicosis, or bleomycin-induced fibrosis using in-vivo, in-vitro, or ex-vivo models. Non-MSC interventions, non-PF conditions, reviews, non-peer-reviewed sources, grey literature, and non-English studies were excluded. Risk of bias for in-vivo studies was assessed using SYRCLE's tool, and in-vitro study quality was evaluated using QUIN. Narrative and quantitative synthesis was performed using a random-effects model to pool mean differences (MD) in Ashcroft scores; heterogeneity was quantified using Cochran's Q and I^2 statistics.

Results:
Out of 397 records, 19 studies met the inclusion criteria (8 in-vivo, 2 in-vitro, 9 combined vivo-in vitro studies). In-vivo studies were rated low or unclear risk with no high-risk domains; in-vitro studies were rated low to medium risk. Meta-analysis showed a pooled MD of –1.35 (95% CI –3.19 to 0.49), indicating lower Ashcroft scores in AD-MSC-treated groups, although not statistically significant ($z = 1.44$, $p = 0.15$). Heterogeneity was considerable ($I^2 = 99\%$). Despite this, all individual studies showed directionally reduced fibrosis in treated groups, supporting a consistent qualitative antifibrotic effect. Across diverse preclinical models, AD-MSC therapies consistently reduce fibrosis while modulating core IPF pathways such as Smad, NF-κB, ERK and key miRNA networks. Paracrine derivatives, extracellular vesicles and conditioned medium in particular, offer scalable and low-risk alternatives that often match whole-cell MSCs. Despite model variability, the uniform direction of benefit highlights strong translational potential, particularly for low- and middle-income settings where existing antifibrotics are limited by high cost and accessibility barriers.

Conclusions:
Collectively, current evidence positions AD-MSCs and their derivatives as promising antifibrotic candidates with strong potential for clinical translation, especially given their consistent effects across varied preclinical pulmonary fibrosis models.

Abstract ID: 20
Title: Human Kidney Organoids in Diabetic Nephropathy Research: A Scoping Review
Authors: Nashmiya Khan; Ramsha Haider; Safa Irfan Shah; Daniya Jawed; Wajeeha Siddiqui; Shifa Imran; Zoha Iftikhar
Affiliation: Karachi Medical and Dental College

Background:
Diabetic nephropathy (DN) is a major cause of chronic kidney disease and renal failure. Traditional models, including animal studies and 2D cell cultures, often fail to capture key aspects of human renal physiology or DN pathogenesis. Kidney organoids—three-dimensional structures derived from pluripotent stem cells—have emerged as human-relevant platforms capable of modeling kidney development, disease mechanisms, and therapeutic responses.Introduction: This scoping review maps current evidence on the use of kidney organoids for modeling DN, summarizing technological advances, experimental applications, mechanistic insights, and remaining challenges.

Methods:
A scoping review approach was used to synthesize peer-reviewed literature describing kidney organoid generation, characterization, and application in DN research. Sources included studies on differentiation protocols, vascularization strategies, organoid-on-chip platforms, CRISPR gene editing, drug screening approaches, and omics-based analyses. Extracted themes captured advances, applications, and knowledge gaps.

Results:
Substantial progress has been made in employing kidney organoids to model DN. Organoids recapitulate nephron-like segments, stromal components, and—when integrated with microfluidic or vascularization approaches—early features of renal microvasculature. Exposure to diabetic stressors (e.g., hyperglycemia, angiotensin II) induces DN-like phenotypes, including podocyte injury, basement membrane alterations, endothelial dysfunction, and early fibrosis. Novel platforms combining organoids with CRISPR editing, microphysiological systems, and multi-omics technologies facilitate mechanistic studies, drug discovery, and personalized modeling. Despite these advances, limitations persist, including fetal-like maturation, incomplete vascular networks, variability between batches, and limited modeling of advanced DN features such as proteinuria and severe fibrosis. Discussion: Kidney organoids offer customizable, human-derived systems that address major limitations of traditional DN models. Their capacity to model early pathogenesis, incorporate patient-specific genetics, and enable high-throughput experimentation makes them powerful tools for DN research. Continued improvement in vascularization, maturation, and protocol standardization will enhance their translational value.

Conclusion:
Kidney organoids represent a rapidly evolving platform for DN modeling, supporting deeper mechanistic understanding, therapeutic screening, and personalized approaches. Ongoing innovations are expected to improve their physiological relevance and expand their utility in both basic and translational research.

Abstract ID: 42
Title: mRNA-Enhanced Stem Cell Therapies: A New Frontier in Epilepsy Treatment
Authors: Syeda Zakia, Muskan Masroor, Muzammil Javed, Waniya Adil, Maryam Saeed, Amna Yousuf, Dr. Saima Mahmood Malhi
Affiliation: Dow University of Health Sciences, Karachi

Background & Objectives:
Epilepsy affects over 50 million people worldwide, with nearly 30% developing drug-resistant epilepsy. Current treatments control seizures but do not repair neuronal loss, interneuron dysfunction, neuroinflammation, or blood–brain barrier disruption. Stem cell therapies offer regenerative potential, but poor graft survival, limited differentiation, and safety concerns limit clinical use. This review explores mRNA-engineered stem cells as a promising regenerative strategy for epilepsy.

Methods:
Literature from 2015–2025 was reviewed, yielding 93 articles, of which 75 were included after screening. A structured review of preclinical, translational, and early clinical studies of mesenchymal stem cells, induced pluripotent stem cell–derived neurons, and GABAergic progenitors, alongside emerging mRNA-based stem cell engineering was conducted. Inclusion criteria covered studies on stem cell therapy for epilepsy and mRNA-based stem cell approaches. Excluded were articles unrelated to epilepsy or CNS disorders, irrelevant to stem cells or mRNA technologies, duplicates, or those with insufficient data.

Results:
None of the studies directly examined mRNA-engineered stem cell therapy in epilepsy. However, 29 articles (38.7%) provided indirect evidence, including mRNA-based stem cell engineering, delivery to neural progenitors, and conceptual frameworks relevant to epilepsy. Preclinical studies made up 42.7%, clinical studies 12.0%, and reviews/meta-analyses 45.3%. Models included in vivo (41.3%), in vitro (24.0%), and combined approaches (18.7%). Preclinical epilepsy studies reported seizure reduction, restored inhibitory neurotransmission, reduced neuroinflammation, and improved neuronal survival, while clinical studies showed preliminary safety and modest benefit. mRNA-based studies enhanced stem cell survival, differentiation, and immunomodulation, supporting future translational potential in epilepsy.

Conclusions:
Although stem cell therapies are promising for epilepsy, clinical use is limited by safety and biological challenges. mRNA-engineered stem cells, though not yet studied in epilepsy, may enhance survival, differentiation, and immunomodulatory function, offering a potential regenerative approach for drug-resistant cases.

Abstract ID: 51
Title: Stem Cell Therapies for Ischemic Stroke Recovery Across Acute and Chronic Phases: Mechanisms, Safety, and Clinical Evidence.
Authors: Muhammad Abdullah, Dr. Sania Nisar
Affiliation: The Aga Khan University, Karachi

Background & Objectives:
Ischemic stroke remains a leading cause of death and permanent disability worldwide. While acute reperfusion therapies save lives, most survivors are left with chronic deficits due to limited endogenous repair. Stem cell therapy has emerged as the most promising neurorestorative strategy capable of promoting long-term recovery beyond the acute phase.

Methods:
Comprehensive narrative review of preclinical and clinical studies (January 2020–November 2025) identified through PubMed, Scopus, Web of Science, and Google Scholar. Focus was placed on mesenchymal stem cells (MSCs), neural stem cells (NSCs), induced pluripotent stem cell derivatives, and bone marrow mononuclear cells (BMMNCs) in ischemic stroke, with special emphasis on safety, efficacy, delivery routes, timing, and chronic-phase outcomes.

Results:
Preclinical models consistently show robust reductions in infarct volume, enhanced angiogenesis, synaptogenesis, and sensorimotor recovery lasting >12 months, driven primarily by paracrine mechanisms. Over 500 patients have been treated in clinical trials, predominantly with MSCs and BMMNCs. Across intravenous, intra-arterial, and intracerebral routes, stem cell therapy demonstrates an outstanding safety profile.

Conclusion:
Stem cell therapy is safe, biologically active, and capable of driving meaningful functional recovery across all stroke phases. Optimized early intervention with MSCs currently offers the strongest signal, while next-generation delivery platforms are unlocking the long-elusive potential of chronic stroke treatment. Large, standardized phase III trials are now warranted to translate this regenerative approach into routine clinical practice.

Abstract ID: 68

Title: Outcomes of haploidentical hematopoietic stem cell transplantation in low- and middle income countries: A systematic review and meta-analysis

Authors: Rida Shakeel, Muhammad Abdullah, Sania Nisar, Zain Amjad, Ghulam Hassam u din tarrar, Hassan Amin

Affiliation: Bahria University Health Sciences Campus, Karachi

Background & Objectives:

Haploidentical hematopoietic cell transplantation (haplo-HCT) has emerged as a vital alternative for patients lacking matched donors, particularly in the treatment of hematologic malignancies and selected non-malignant disorders. Advances in conditioning regimens, graft sources, and graft-versus-host disease (GVHD) prophylaxis have expanded its feasibility in low and middle income countries (LMICs). This systematic review summarizes real-world evidence on indications, transplant strategies, and survival outcomes of haplo-HCT in LMIC settings.

Methods:

A comprehensive systematic search of PubMed, ClinicalTrials.gov, Scopus, and Web of Science was conducted for studies published up to June 2025. Eligible studies reported post haplo-HCT outcomes from LMICs in adult populations. Extracted data included patient demographics, disease indications, conditioning intensity, graft source, GVHD incidence, relapse, engraftment kinetics, and survival outcomes. Risk of bias was assessed using the modified Newcastle Ottawa Scale and ROB2 tool. Meta analyses were performed using random-effects models in R.

Results:

Twenty four studies encompassing 2,248 patients were included. The patients' age ranged from 0 to 79 years, with follow up periods spanning 1–83 months. Among studies reporting sex (n=1,915), 63.3% of recipients were male. Disease-specific data demonstrated acute leukemia as the predominant indication (72.4%), followed by aplastic anemia, lymphoma, and myelodysplastic syndromes. Peripheral blood stem cells were the primary graft source, and reduced intensity or non myeloablative conditioning regimens were favored over myeloablative approaches. Post-transplant cyclophosphamide was widely utilized for GVHD prophylaxis. Pooled overall survival was 0.52 at 1 year (95% CI: 0.41–0.63; I^2=87.5%) and 0.43 at 2 years (95% CI: 0.36–0.53; I^2=83.1%). One-year disease-free survival was 0.63 (95% CI: 0.50–0.75). Acute GVHD incidence within 90–100 days was 0.37, and relapse occurred in approximately one-third of patients. Substantial heterogeneity was observed across studies.

Conclusions:

Haploidentical hematopoietic cell transplantation is increasingly implemented in LMICs, primarily for acute leukemia, using peripheral blood grafts, reducedintensity conditioning, and post transplant cyclophosphamide based GVHD prophylaxis. Survival outcomes remain heterogeneous and modest, underscoring the need for standardized protocols, strengthened supportive care infrastructure, and prospective multicenter trials to optimize outcomes in resource-limited settings. Keywords: haploidentical transplantation; low and middle income countries; overall survival; graft versus host disease; real-world evidence.

Abstract ID: 70
Title: Stem Cell Policies in Pakistan: Navigating Ethics and Regulatory Frameworks
Authors: Sujjawal Ahmad, Maryam Huda, Azhar Hussain, Kulsoom Ghias, Ather Enam, Karim F. Damji, Irfan Khan
Affiliation: The Aga Khan University, Karachi

Background:
Stem cell research (SCR) and regenerative medicine (RM) are rapidly advancing fields offering potential treatments for a wide range of diseases previously considered incurable. The United States (US) and European Union (EU) have established comprehensive regulatory frameworks to govern these innovations, while Pakistan's regulatory environment remains fragmented, outdated, and without clarity. The growing global trend of unregulated interventions has emerged as a public health concern, which underscores the urgency of policy reform in Pakistan, where demand for such therapies is rising.

Objective:
This study aims to generate evidence-informed recommendations for strengthening the policy framework for SCR and therapy in Pakistan. The objectives were (1) to map the landscape of SCR and therapy in Pakistan with attention to its policy context, drivers, and key actors, and (2) to analyze Pakistan's policy and regulatory framework in comparison with established international frameworks of the US and EU.

Methods:
A qualitative research design was employed, using the Walt and Gilson Policy Triangle as a conceptual framework to examine context, content, processes, and actors. Data sources included document analysis of regulatory frameworks and policy guidelines issued by the Drug Regulatory Authority of Pakistan (DRAP), National Bioethics Committee (NBC), United States Federal Drug Regulatory Authority (FDA), and the European Union's European Medicines Agency (EMA). 16 Key informant interviews (KII) and 04 focus group discussions (FGDs) were conducted with researchers, clinicians, bioethicists, social scientists, and relevant institutional experts. The data of KIIs and FGDs were thematically analyzed.

Results:
The study revealed a fragmented and reactive policy landscape. Key gaps include outdated and non-binding guidelines, weak regulatory and institutional infrastructure, limited GMP-certified facilities, inadequate funding, and insufficient oversight of early commercialization and direct-to-consumer marketing of stem cell interventions. Public awareness is low, and cultural and societal barriers persist. Comparison with the FDA and EMA frameworks showed Pakistan lacks product-specific classification and regulation and structured monitoring, and key gaps in the existing guidelines across preclinical, clinical, and marketing phases.

Conclusions:
The issues brought to light by this study called for the need for systemic big R reform, however the way forward requires incremental steps towards a coherent, proactive, and risk-based governance. This will ultimately place the innovative culture in the country in terms of responsible and ethical research that contributes to improving the quality of life of the millions of people suffering from diseases.

Abstract ID: 74
Title: CD34+ Hematopoietic Stem Cells: under the Lens of Magnetic Cell Sorting Enrichment, Expansion & Characterization for gene therapy purposes
Authors: Shagufta Naz, Hafiz Ghufran, Safana Farooq, Areeb Ahmed, Sadia Habib, Aafia Shahid Arain, Anwar Alam, Fawad-ur-Rehman, Afsar Mian
Affiliation: The Aga Khan University, Karachi

Background:
$CD34^+$ hematopoietic stem and progenitor cells (HSPCs) serve as the primary target for ex vivo gene editing due to their ability to self-renew and reconstitute the entire blood system. Advances in CRISPR-Cas9 and other genome engineering tools have enabled precise correction or modulation of disease-causing genes within these cells. This approach holds significant therapeutic promise for hemoglobinopathies, including β-thalassemia and sickle cell disease, by restoring normal hemoglobin production after transplantation back into the patient.

Objective:
This study aimed to isolate, culture, and characterize $CD34^+$ hematopoietic stem cells, obtained via magnetic-activated cell sorting (MACS), from peripheral blood samples of transfusion-dependent β-thalassemia patients.

Methodology:
Mononuclear cells were isolated by density gradient centrifugation, and $CD34^+$ cells were enriched using magnetic microbeads. Cultures were maintained in cytokine-rich medium for 14 days to evaluate growth kinetics, phenotype, and gene expression. The expression of stem cell markers was confirmed by qPCR and immunocytochemical staining.

Results:
On average, $0.1 \times 10^6 \pm 0.1$ $CD34^+$ cells were obtained from $3.8 \times 10^6 \pm 2.4$ MNCs. Upon culture, cell numbers increased from $0.23 \times 10^6 \pm 0.2$ at day 0 to $4.25 \times 10^6 \pm 4.4$ at day 14. Cytokine-enriched medium supported $CD34^+$ cells, as indicated by flow cytometry analysis. The cultured cells expressed CD34 (87.3 ± 2.4 %) and CD117 (66.9 ± 7.3%) markers, with the highest proportion of $CD34^+CD117^+$ cells observed at 90hrs or 3.75 days, followed by a gradual decline in subsequent days. The results from qPCR and immunocytochemical staining was found consistent with results obtained via flow cytometry.

Conclusion:
$CD34^+$ hematopoietic stem cells can be efficiently isolated from peripheral blood of transfusion-dependent β-thalassemia patients using MACS and expanded in cytokine-enriched culture. The observed expression dynamics of CD34 and CD117 suggest early proliferation followed by gradual differentiation, highlighting the potential of these cells as a target population for ex vivo gene editing–based therapeutic strategies for hemoglobinopathies.

THEME 2: REGENERATIVE MEDICINE AND TISSUE ENGINEERING

Abstract ID: 58

Title: Tissue engineered multifunctional chitosan-modified polypropylene hernia mesh loaded with bioactive phyto-extracts

Authors: Faiza Sharif, Sadaf Nosheen, Hamid Mukhtar, Sajjad Haider, Rawaiz Khan.
Affiliation: COMSATS University Islamabad, Lahore Campus

Background & Objectives:
Surface modified tissue engineered polypropylene / PP hernia meshes were fabricated by incorporating Bacterial cellulose / BC and chitosan / CS and phytochemical extracts. Under current practice, hernia and other traumatic injuries to the abdominal organs are clinically treated with surgical meshes. Often the foreign body reaction and infections result in relapse in patients which dictates additional reparative surgical procedures and pain. To improve the outcome of clinical restorative procedures new biomaterials with improved characteristics are required.

Methods:
The functionalized meshes were physically and chemically characterized using SEM, mechanical testing, FTIR and XRD. The antimicrobial activity was qualitatively and quantitatively tested using E. coli and S. aureus strains of bacteria. In vitro biocompatibility and wound healing effect of the modified meshes were performed using NIH3T3 fibroblast cell lines. Furthermore, tissue engineering potential of the meshes was evaluated using confocal fluorescent microscopy. In vivo implantation of the meshes was performed in male wistar rats for 21 days. Therefore, PP meshes with sustained drug delivery system augmented with anti-inflammatory and anti-microbial characteristics were developed.

Results:
The coatings hereby not only increased the tensile strength of meshes but also prevented the modified meshes from causing infection. Current study resulted in CS-BC bioactive PP meshes loaded with phytochemicals which showed anti-inflammatory, antibacterial and wound healing potential.

Conclusions:
These meshes can be valuable to lessen the post-surgical complications of implanted PP mesh and thus reduce rejection and recurrence.

Abstract ID: 61
Title: Three-Dimensional Spatial Organization Modulates Gene Expression in Mesenchymal Stem Cells Cultured on Human Tissue-Derived Scaffold
Authors: Faiza Chaudhary, Marium Mansuri, Muhammad Shakeel, Omair Anwar Mohiuddin
Affiliation: International Center for Chemical and Biological Sciences, University of Karachi

Background & Objectives:
The therapeutic potential of mesenchymal stem cells (MSCs) is largely mediated by their secretory factors and paracrine signaling, which has gained considerable interest due to its potential implications in regenerative medicine. Conventional two-dimensional (2D) culture systems fail to recapitulate the native physiological microenvironment of cells, causing genotypic changes that limit their therapeutic capabilities. Whereas, three-dimensional (3D) culture systems provide a biomimetic microenvironment that allow robust cell-cell and cell-matrix communication and potentially boosts their therapeutic efficacy. Herein, the present study utilizes decellularized human amniotic membrane (D-hAM) as a 3D scaffold for culturing human umbilical cord-derived MSCs (hUC-MSCs) to investigate the influence of D-hAM microarchitecture on cellular behaviour and transcriptomic profile of hUC-MSCs.

Methods:
The in vitro biocompatibility of D-hAM with hUC-MSCs was assessed by scanning electron microscopy (SEM). Furthermore, cell viability, proliferation, and differentiation assays were performed to analyze cellular behaviour. The gene expression profile of D-hAM-seeded hUC-MSCs (D-hAM-MSCs) was evaluated by RNA sequencing followed by gene ontology (GO) and pathway enrichment analyses.

Results:
The surface topography of D-hAM-MSCs using scanning electron micrographs displayed favourable cell attachments. The viability and proliferation analyses demonstrated sustained cellular viability and consistent increase in proliferation over a period of 10 days. Furthermore, D-hAM-MSCs demonstrated selective lineage differentiation into adipocytes. Transcriptomic profiling revealed major gene expression differences amongst both the groups. Out of 12,563 identified genes, 956 were significantly upregulated in D-hAM-MSCs, while 1,716 genes were observed to be downregulated. Prominently, genes associated with ECM organization, tissue remodeling, cell adhesion, and migration, were upregulated with the concurrent downregulation of inflammation-associated genes. The transcriptomic changes in D-hAM-MSCs indicate robust ECM remodeling, adaptation to hypoxia-induced oxidative stress, immunomodulation, and metabolic reprogramming towards cellular survival. These findings were supported by GO and pathway enrichment analyses, which identified significant upregulated canonical pathways and biological processes involved in signal transduction, cell polarity, locomotion, and morphogenesis, with downregulation of cellular respiratory metabolism and protein synthesis. Collectively, the results demonstrate a progressive shift of cells towards a more quiescent and less metabolically active state.

Conclusions:
The current findings highlight the influence of D-hAM spatial architecture and the role of ECM in mediating the regenerative potential of hUC-MSCs, and facilitate designing more efficacious and robust tissue engineering constructs for clinical translation.

Abstract ID: 82

Title: Characterization of Human Tissue–Derived Scaffold: MSC Compatibility, Multi-Omics Profiling, and Regenerative Potential

Authors: Kainat Ahmed, Haadia Tauseef, Faiza Chaudhary, Asmat Salim, Muhammad Shakeel, Munazza Raza Mirza, Omair A. Mohiuddin
Affiliation: Dow University of Health Sciences, Karachi

Background:
Extracellular matrix (ECM)–based scaffolds are valuable in tissue engineering because they preserve the native structural, mechanical, and physiological microenvironment of tissues, thereby supporting cell adhesion and matrix-related activities. These scaffolds are prepared through a process known as decellularization, in which tissues are treated to remove cellular components while retaining the ECM architecture and composition. Decellularized tissue–based scaffolds can subsequently be repopulated with cells to study cell–ECM interactions, support in vitro tissue engineering, and enable in vivo regenerative applications.

Objectives:
We developed a decellularized human amniotic membrane (d-HAM) using a salt-based decellularization method. The objectives of this study were to characterize the d-HAM, evaluate its compatibility with mesenchymal stem cells (MSCs), and assess its wound-healing potential in vivo.

Methods:
The d-HAM was characterized using liquid chromatography–mass spectrometry (LC–MS) to define its proteomic profile and infer potential effects on cells seeded onto the scaffold. The compatibility of human MSCs with d-HAM was evaluated using cell viability assays and imaging techniques. Transcriptomic analysis was performed on MSCs cultured on the scaffold for ten days to assess gene expression changes resulting from interactions with ECM components. Finally, the regenerative potential of the scaffold was evaluated using a mouse model of full-thickness skin wounds.

Results:
Proteomic analysis demonstrated that d-HAM retained key ECM components while cellular proteins were largely depleted. The scaffold supported MSC attachment and proliferation while maintaining the cells in a relatively quiescent state. In vivo, d-HAM significantly promoted wound regeneration in the mouse full-thickness skin wound model.

Conclusions:
This human tissue–derived scaffold retains essential structural ECM molecules required for MSC attachment and viability. Furthermore, d-HAM modulates the gene expression profile of MSCs compared to conventional two-dimensional culture. The scaffold is biocompatible and effectively promotes wound healing in a full-thickness skin wound model in mice.

Abstract ID: 83
Title: Optimization of Alginate Capsules to Generate a Hematopoietic Stem Cell Bone Marrow Niche
Authors: Shermeen Tanveer, Zabreen Mustafa & Witold Nowak
Affiliation: University of Silesia, Katowice, Poland

Background:
The complex microenvironment of hematopoietic bone marrow niche maintains hematopoiesis. It has a heterogeneous landscape which is difficult to replicate. Niche supporting cells like Mesenchymal stem cells (MSCs) and hematopoietic cells characterize the niche. Alginate can be used to replicate this environment through encapsulation of bone marrow cells and polymer microparticles.

Objectives:
1. Optimize alginate capsules taking under consideration cytotoxicity and robustness.
2. Encapsulate unprocessed bone marrow cells inside capsules effectively to ensure cell survival and long term culture.
3. Evaluate niche markers through comparison with 2D culture and characterization techniques to validate replication of niche.

Methods:
Primary mouse bone marrow cells were isolated and encapsulated within three-dimensional alginate capsules to replicate key features of the hematopoietic stem cell (HSC) bone marrow niche. Alginate capsules were formed via ionic crosslinking using calcium chloride, following an optimized droplet-based protocol to ensure capsule robustness and minimize cytotoxicity. Polycaprolactone (PCL) microparticles, functionalized with collagen type I, were incorporated to mimic extracellular matrix (ECM) cues associated with the endosteal niche.

Results:
The optimized calcium-crosslinked alginate capsules were spherical, mechanically stable up to five days, and non-cytotoxic, as evidenced by sustained cell viability even after partial capsule rupture. Capsule diameter increased by approximately 13% over five days, indicating ion exchange and swelling behavior. XPS analysis confirmed calcium release from the crosslinked alginate network. Confocal imaging demonstrated successful cell encapsulation and proliferation in 3D, with cells exhibiting predominantly round morphologies and limited elongation near PCL microparticles. While hematopoietic ($CD45^+$) cells proliferated robustly, stromal ($CD146^+$) cell presence and attachment to PCL microparticles were limited. Gene expression analysis showed no detectable expression of key niche factors (CXCL12, CSF3), indicating that a fully functional bone marrow-like niche was not established. Overall, the results suggest that while alginate capsules effectively support cell survival and 3D culture, unprocessed bone marrow is insufficient for robust niche replication without prior cell enrichment.

Conclusions:
• Calcium crosslinking effectively encapsulates cells and poses no toxicity.
• Lack of cell adherence to PCLs
• Unprocessed Bone marrow is not suitable for bone marrow replication.
• Bone marrow cells should be processed with magnetic sorting or hypoxia culture.
• Endothelial markers should be investigated

Abstract ID: 29

Title: Regenerative Rejuvenation Through Klotho mRNA: A Polymer-Enabled Strategy to Counter Cellular Aging

Authors: Syed Aqib Ali Zaidi, Dezhong Zhou and Guangqian Zhou
Affiliation: Shenzhen University, China

Background & Objectives:
Cellular aging is driven by senescence, mitochondrial dysfunction, oxidative stress, and dysregulated calcium homeostasis, collectively impairing tissue regeneration. Klotho, a key longevity protein, counteracts these pathways, but its age-related decline accelerates degeneration. mRNA-based therapy offers a transient, non-integrating strategy to restore Klotho and promote cellular rejuvenation. This study aimed to evaluate the regenerative potential of engineered IVT Klotho mRNA delivered through a biodegradable polymer-based delivery system.

Methods:
Chemically optimized IVT Klotho mRNA was formulated into nanoparticles using a hyperbranched poly(β-amino ester) (HPAE) carrier to enable efficient cellular uptake. Delivery performance was assessed across multiple cell types, including induced mesenchymal stem cells (iMSCs). Rejuvenation and regenerative outcomes were evaluated through senescence markers, SA-β-gal activity, mitochondrial function, oxidative stress indicators, antioxidant responses, and calcium homeostasis in senescent and Klotho (-/-) iMSCs.

Results:
Klotho mRNA treatment produced robust regenerative effects, significantly lowering p53, p21, and p16, reducing SA-β-gal activity, restoring mitochondrial membrane potential, and enhancing antioxidant capacity (SOD2, catalase). mRNA delivery also reduced intracellular ROS and normalized calcium balance, key determinants of cellular health and regenerative function. Notably, young iMSCs remained unaffected, highlighting the selective and context-specific rejuvenation achieved by Klotho mRNA therapy. The HPAE carrier enabled efficient delivery with minimal cytotoxicity.

Conclusions:
This work demonstrates that Klotho mRNA therapy can reverse multiple hallmarks of cellular aging and restore regenerative function, establishing a strong foundation for mRNA-based rejuvenation strategies. The biodegradable HPAE carrier provided effective delivery, supporting the broader application of polymer-assisted mRNA therapeutics in age-related degeneration.

Abstract ID: 17
Title: N-Acetylcysteine as a Neuroprotective Agent: Combating Oxidative Stress in Retinal Degeneration
Authors: Kainat Ahmed, Rabbia Muneer, Karim F. Damji, Irfan Khan
Affiliation: The Aga Khan University, Karachi

Background:
Oxidative stress (OS) plays a central role in damaging retinal pigment epithelium (RPE) cells, a characteristic of retinal degenerative diseases. Sodium iodate ($NaIO_3$), a potent oxidative agent which targets RPE cells, making it a potential retinal degeneration disease model.Objectives: The current study investigates the therapeutic potential of N-Acetylcysteine (NAC), a well-known antioxidant, in counteracting $NaIO_3$ induced oxidative damage in ARPE-19 cells.

Methods:
Using ARPE-19 cells, we evaluated protective efficiency of NAC through a comprehensive analysis of cell viability, reactive oxygen species (ROS) production, mitochondrial membrane potential, colony forming ability, cell migration, cell cycle distribution, and gene expression related to OS, inflammation, autophagy, and cell cycle regulation.

Results:
Exposure to $NaIO_3$ activated a cascade of cellular dysfunctions in ARPE-19 cells, including increased ROS production, reduced mitochondrial membrane potential, and weakened proliferative and migratory capacities as well as significant dysregulation of genes associated with cellular stress responses. Moreover, treatment with NAC effectively restored these effects by lowering ROS levels, restoring mitochondrial membrane potential, improving cell viability, and maintaining colony forming and migration ability. NAC also inhibited apoptosis and normalized the expression of genes involved in OS, inflammation, and autophagy.

Conclusion:
Our findings determine role of NAC against OS in ARPE-19 cells and highlight its potential as a therapeutic approach to combat OS driven retinal degeneration.

Abstract ID: 54
Title: Standardising Micro and Nano Fat Grafting in Resource-Limited Settings: A Reproducible Protocol and Early Clinical Outcomes
Authors: Farhat Ul Ann Tayyaba,Samina Ijaz,Shazma Seher
Affiliation: Quaid E Azam Medical College, Bahawalpur

Background & Objectives:
Autologous fat grafting is widely used in plastic and reconstructive surgery due to its versatility and cost-effectiveness, particularly in resource-limited settings. However, variability in harvesting, processing, and injection techniques leads to inconsistent outcomes, especially in regenerative applications such as scar remodeling. This study aimed to standardize a reproducible protocol for micro- and nano-fat graft preparation and to evaluate early clinical outcomes in facial rejuvenation and post-burn and post-traumatic scar management, focusing on scar pliability, texture, and colour.

Methods:
A prospective clinical study was conducted on 20 patients undergoing facial rejuvenation, contour correction, or treatment of post-burn and post-traumatic scars. Fat was harvested predominantly from the lower abdomen using a low-pressure technique with 10-mL syringes and blunt, multi-port cannulas (3–5 mm diameter). A buffered tumescent solution containing low-dose lidocaine (≤0.05%) and epinephrine (1:1,000,000) was infiltrated 10–15 minutes prior to harvest to minimize cellular trauma. Harvested fat was processed using a modified Coleman technique with centrifugation at approximately 50 g for 3 minutes to obtain purified microfat. Nano-fat was prepared through mechanical emulsification and filtration without enzymatic digestion. Fat was injected in small aliquots across multiple tissue planes using a retrograde technique. Cellular viability was assessed in selected samples using trypan blue exclusion assay. Clinical outcomes were evaluated using standardized pre- and post-operative photographs and patient satisfaction scores.

Results:
Trypan blue exclusion assays demonstrated approximately 150,000–200,000 viable nucleated cells per millilitre of processed fat. Clinically, patients undergoing facial rejuvenation showed consistent improvement in contour and skin quality, with patient satisfaction reaching up to 90%. In post-burn and post-traumatic scars, micro-fat grafting resulted in mechanical softening and improved pliability, while nano-fat application was associated with improved surface texture and partial normalization of scar pigmentation during early follow-up.

Conclusions:
This study demonstrates that a standardized, low-cost protocol for micro- and nano-fat grafting can be safely and reproducibly implemented in resource-limited settings, yielding meaningful aesthetic and regenerative outcomes. The combined use of micro- and nano-fat offers a practical, biologically plausible approach for improving scar pliability, texture, and colour, supporting its role as a scalable regenerative adjunct in plastic and reconstructive surgery.

Abstract ID: 67

Title: Oral Mesenchymal Stem Cells for Tissue engineering: Comparative Isolation and Stemness Profiling of hGMSCs and hPDLSCs

Authors: Asma, Nazia Ahmed, Shakila Nazeer, Lubna Khan
Affiliation: Dow University of Health Sciences, Karachi

Background & Objectives:
Human gingival mesenchymal stem cells (hGMSCs) and human periodontal ligament stem cells (hPDLSCs) are promising adult stem cell populations with significant regenerative potential in craniofacial and periodontal tissue engineering. Despite their common neural crest origin, variations in their biological behavior necessitate a comparative evaluation of their isolation efficiency and stemness characteristics. Objective: To compare gingival and periodontal ligament derived mesenchymal stem cells with respect to their stemness attributes and regenerative potential using comprehensive morphological, functional, and immunophenotypic characterization.

Methods:
Human gingival tissue and periodontal ligament samples were obtained from systemically healthy donors undergoing routine dental procedures. Stem cells were isolated using the tissue explant technique and expanded under standard culture conditions. Morphological characteristics were assessed through phase-contrast microscopy. Multipotency was evaluated via trilineage differentiation into osteogenic, adipogenic, and chondrogenic lineages using standard induction protocols. Immunophenotypic characterization was performed using flow cytometry to assess the expression of mesenchymal stem cell surface markers (CD73, CD90, CD105) and the absence of hematopoietic markers (CD34, CD45). Quantitative data were expressed as mean ± standard deviation, and statistical comparisons between hGMSCs and hPDLSCs were performed using student t- tests, with $p < 0.05$ considered statistically significant.

Results:
Both hGMSCs and hPDLSCs demonstrated plastic adherence and a fibroblast-like spindle-shaped morphology. Trilineage differentiation assays confirmed their multipotent potential in both groups. Flow cytometric analysis revealed high expression of mesenchymal stem cell markers and minimal expression of hematopoietic markers, with no statistically significant differences observed between hGMSCs and hPDLSCs ($p > 0.05$).

Conclusion:
hGMSCs and hPDLSCs both cell types fulfill the minimal criteria for mesenchymal stem cells, exhibiting similar morphological features, immunophenotypic profiles, and multilineage differentiation potential. These findings support their suitability for regenerative and translational applications in periodontal and craniofacial tissue engineering.

Abstract ID: 69
Title: Identification and Characterization of Natural Biomaterial for Three-Dimensional (3D) Cell Culture and Bioprinting Applications
Authors: Zarmeen Suhail, Sheerien Rajput
Affiliation: The Aga Khan University, Karachi

Background & Objectives:
Conventional 2D cell culture systems fail to adequately recapitulate in vivo microenvironments & cellular interactions. While 3D culture systems offer greater physiological relevance, their adoption is limited by the high cost & limited accessibility of biomaterials, particularly in low- & middle-income countries.
Objective: This study aims to evaluate the biological feasibility of a natural biomaterial (hereafter referred to as Factor-X) as a 3D cell culture platform & develop a Factor-X based bioink suitable for extrusion-based 3D bioprinting applications.

Methods:
Factor-X was sourced from pathogen-free biological material & processed aseptically to obtain a clear, homogeneous formulation. Samples were mechanically processed under controlled conditions & stored at −80 °C until use. Biocompatibility was evaluated using GFP-transfected MCF-7 cells cultured either encapsulated within Factor-X or on Factor-X–coated substrates, without addition or replacement of external culture media; uncoated substrates served as controls. Cellular behavior, morphology, & viability were monitored using widefield fluorescence microscopy, confocal imaging with 3D reconstruction, & live time-lapse imaging. H&E staining assessed cell distribution & structural organization. Cell recovery methods were optimized to enable RNA & protein isolation for downstream molecular analyses. Following biological validation, Factor-X was combined with appropriate materials to enable hydrogel formation and extrusion for 3D bioprinting applications.

Results:
Factor X formed a consistent web-like lattice network when coated on slides, comprising tri-, tetra-, & penta-sided structures. H&E staining revealed diverse morphologies, including X-, Y-, & curved line-like patterns. MCF7 cells cultured on Factor X-coated slides for 20 days exhibited organized lattice networks with lumen-like structures & enhanced cell–cell contacts, whereas cells on uncoated slides showed compromised proliferation. Immunocytochemical analysis demonstrated mixed cytoplasmic & membranous expression of epithelial membrane antigen, supporting Factor X as a biological substrate. In unsupplemented Factor X gel, GFP+ MCF7 cells remained viable & proliferative for 14 days without additional media, forming organized 3D structures. 3D reconstruction confirmed cell growth in X, Y, & Z dimensions, with secondary structures reaching 100–120µm in height. Time-lapse imaging on Factor X-coated dishes revealed 3D bodies measuring up to 342µm (X), 276µm (Y), & 56 µm (Z). RNA & protein isolated from cells cultured in Factor X gel showed prominent 28S/18S and protein bands, indicating compatibility with molecular analyses. Factor X, combined with gelatin & xanthan gum, exhibited shear-thinning behavior & suitable extrusion for 3D bioprinting, followed by glutaraldehyde crosslinking.

Conclusion:
Overall, Factor X supports 2D & 3D MCF7 cell growth & exhibits modifiable viscosity for bioink formulation in 3D bioprinting applications.

Abstract ID: 81

Title: Regenerative Small-Molecule Cytokinins Originate from Diet-Gut Microbiome Interactions in Mammalian Hosts

Authors: Eman M. Othman, Thomas Dandekar, and Muhammad Naseem
Affiliation: Zayed University, Abu Dhabi

Background & Objectives:

Cytokinins (CKs) are adenine-derived plant hormones with undefined origin, distribution, and functions in mammalian systems. Despite their widespread presence across various forms of life (1, 2), the physiological role of CKs beyond the kingdom plantae remains a terra incognita. However, the exogenous application of CKs holds strong therapeutic potential, including their ability to mitigate oxidative stress (3), ameliorate various pathophysiological conditions (4), and attenuate inflammation (5). They show promise in neurodegenerative diseases such as Huntington's disease (HD) (6,7) and Familial Dysautonomia (FD) (8), and also hold potential as potent cosmeceuticals (9). CKs also contribute to muscle growth (10, 11), anti-senescence processes (12), and longevity (13-15).

Methods:

First and foremost, a comprehensive investigation into the origin of endogenous CK production is essential for elucidating its biological significance in mammalian systems. Using integrated metabolomics, microbiome, and metagenomics approaches, we systematically analyzed the production of CK in mammals.

Results:

Serum profiling across five animal species revealed the consistent detection of multiple CK derivatives, which were significantly lower compared to those in plants. Species-specific differences, such as reduced trans-zeatin in mice and lower kinetin in humans, further suggest divergent regulatory patterns. In mice, CKs were present in kidney, heart, and liver, demonstrating systemic distribution. Dietary manipulation through starvation significantly reduced CK abundance across serum, colon, faeces, and urine, confirming diet as a major contributor to the mammalian CK pool. Meta-omics analysis of gut microbiomes identified CK-related genes across multiple microbial taxa. Germ-free mice experiments exhibited substantially diminished CK levels compared to conventionally raised counterparts.

Conclusion:

Collectively, our findings identify CKs as diet- and microbiome-modulated metabolites in mammals, warranting future investigation to elucidate their physiological significance in mammalian biology.

Abstract ID: 2
Title: Efficacy of Autologous Platelet-Rich Plasma Treatment in Retinitis Pigmentosa: A Systematic Review
Authors: Ulfat Anjum, Chen-Wei Pan, Andrzej Grzybowski and Carla Lanc**a**
Affiliation: University of Lahore

Background:
Retinitis pigmentosa (RP) is a prevalent retinal dystrophy causing lifelong vision loss. Autologous Platelet-Rich Plasma (aPRP) treatment has been described as an emerging treatment to reduce the permanent retinal cell death.

Objective:
This study aimed to review the current literature about the effect of aPRP on visual functions in RP, highlighting research gaps.

Methods:
A comprehensive literature search was conducted following the PRISMA guidelines using relevant keywords from the inception until November 2024. Cohort, case-control, and clinical trial studies were included, and animal studies were excluded. Risk of bias was assessed using the Cochrane tools.

Results:
Seven articles were found (two clinical trials and five non-random interventional studies). The outcome measures were based on best corrected visual acuity (BCVA; n=7), electroretinogram (n=4), visual field (VF; n=6), optical coherence tomography (n=4), microperimetry (n=4), and fixation stability (n=2). All studies indicated a potential benefit of aPRP, and no major adverse events were reported. Several studies lacked controls and reported short-term preservation or improvement of visual functions such as VF, microperimetry, and electrophysiological responses.

Conclusion:
Changes in BCVA were not clinically meaningful. Moreover, most studies were from a single country, limiting generalizability and urging the need for multicenter trials.

Abstract ID: 8
Title: Investigating the Neurogenic Potential of Stem Cells in Neurodegenerative Diseases: Exploring Transcriptional, Molecular, and Environmental Inducers of Adult Neurogenesis
Authors: Abdullah Hadi and Safaa Batool
Affiliation: International Center for Chemical and Biological Sciences, University of Karachi

Background and objective:
Neurodegenerative disorders such as Alzheimer's disease (AD), Parkinson's disease (PD) and Huntington's disease (HD) are characterised by progressive neuronal loss, synaptic dysfunction and limited regenerative capacity in the adult brain. Adult neurogenesis occurs primarily in the subventricular zone (SVZ) and the subgranular zone (SGZ) of the hippocampus, but declines markedly during ageing and is compromised further in neurodegenerative conditions. Stem cell therapy using neural stem cells (NSCs) or induced-pluripotent-stem-cell derived NSCs (iPSC-NSCs) offers a promising route to replenish neuronal populations and restore connectivity. However, successful functional integration demands an understanding of the endogenous neurogenic signalling environment and how it can be optimized.

Methods:
A systematic search of PubMed, Scopus, and Web of Science was performed using terms related to adult neurogenesis, neural stem cells, and neurodegenerative disease. Eligible peer-reviewed mammalian studies on adult neurogenesis or stem-cell–based modulation were included. After screening and full-text review, data on interventions, neurogenesis markers, and functional outcomes were extracted and synthesized thematically.

Results:
Enhancing adult neurogenesis in neurodegenerative models consistently required coordinated modulation of transcriptional factors (NeuroD1, Sox2, Ascl1), key signaling pathways (Wnt/β-catenin, SHH, Notch), and neurotrophic support (BDNF, GDNF), together with improvements in the host microenvironment. Evidence showed that exercise, environmental enrichment, and reduced inflammation further promoted NSC differentiation and integration. Overall, findings across studies indicate that combinatorial strategies including, integrating stem cell preconditioning with targeted regional delivery and host niche modulation may yield the most robust neurogenic and functional outcomes.

Conclusions:
Adult neurogenesis represents a promising regenerative strategy for neurodegenerative diseases, with the strongest evidence supporting stem-cell approaches enhanced by transcriptional programming, signaling modulation, and environmental enrichment. Despite feasibility, challenges remain, including hostile disease niches and limited integration. The proposed study on pre-conditioned NSCs in AD and PD models aligns with current research needs and may advance more effective neurodegenerative therapies.

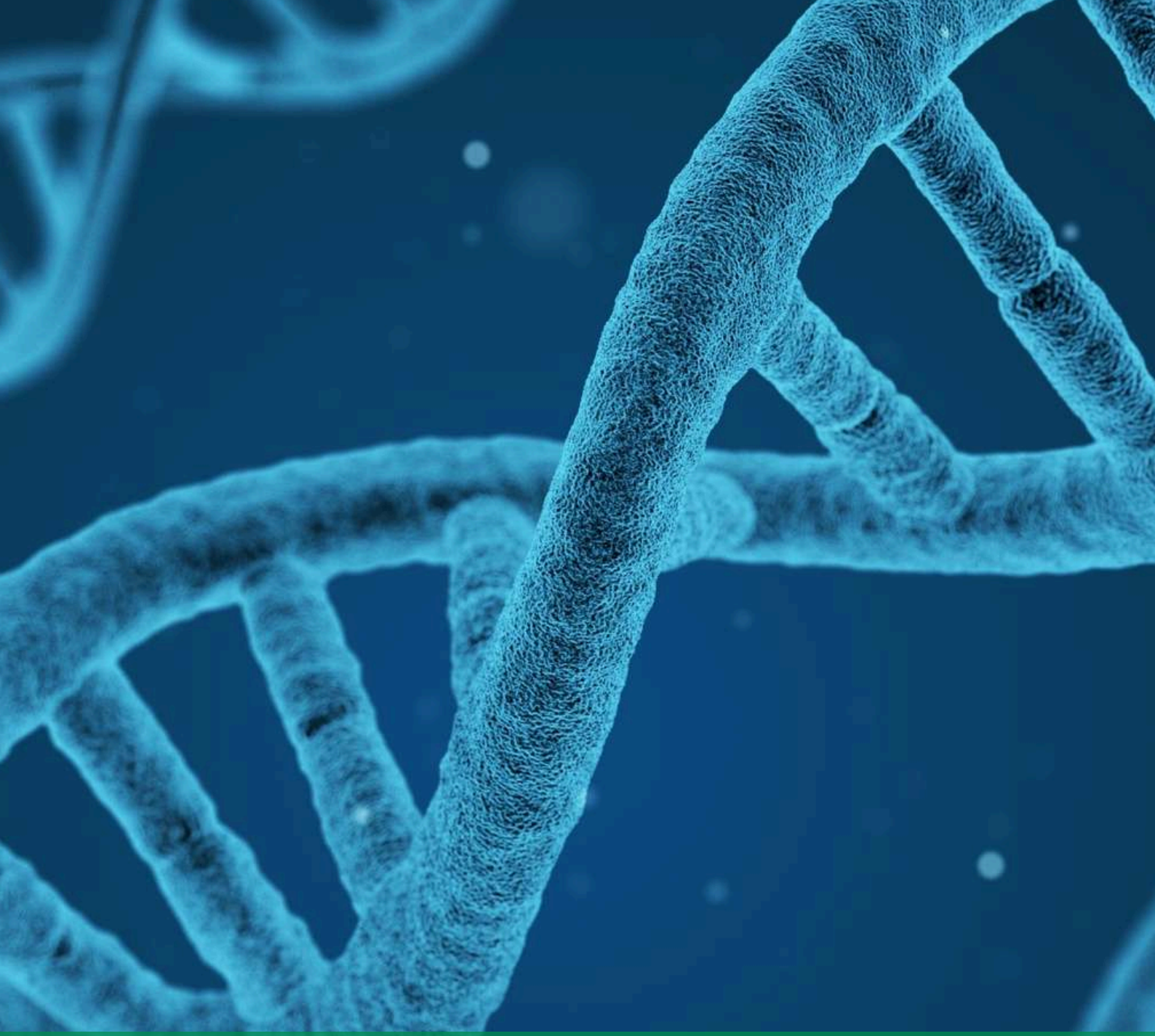
Abstract ID: 27
Title: Messenger RNA Loaded Immunomodulatory Biomaterials for Enhanced Healing of Diabetic Wounds
Authors: Amna Ali, Javeria Iqbal
Affiliation: Dow University of Health Sciences, Karachi

Background & Objectives:
Diabetic wounds fail to heal due to persistent inflammation, oxidative stress, and impaired angiogenesis. Messenger RNA-based therapies offer targeted ways to modulate immune pathways and stimulate tissue repair, but their effectiveness depends on biomaterials that protect and deliver messenger RNA within the harsh wound environment. This systematic review aimed to evaluate current evidence on biomaterial-based messenger RNA delivery systems designed to modulate immune responses and enhance healing in diabetic wounds.

Methods:
A systematic search was conducted across PubMed, Scopus, Web of Science, and Google Scholar using predefined terms related to messenger RNA delivery, biomaterials, immunomodulation, and diabetic wound healing. Studies were included if they used messenger RNA-loaded biomaterials in diabetic wound models and reported immunological or regenerative outcomes. Data extracted included biomaterial type, messenger RNA cargo, immune effects, angiogenesis levels, wound closure rates, and safety findings. The review followed the PRISMA framework.

Results:
Seventy-seven eligible studies were identified, predominantly preclinical work conducted between 2022 and 2025. Most used lipid nanoparticles, hydrogels, or microneedle-based dressings to deliver messenger RNA encoding interleukin-4, interleukin-10, vascular endothelial growth factor-A, or other regenerative molecules. Across studies, these systems consistently promoted macrophage transition from inflammatory to healing phenotypes, reduced oxidative stress, improved vascular formation, and accelerated wound closure in diabetic rodent models. However, no human studies were found. Evidence gaps included the absence of clinical trials, limited long-term safety data, lack of standardized outcome measures, and scarce head-to-head comparisons between delivery platforms.

Conclusions:
Messenger RNA-loaded immunomodulatory biomaterials represent a promising strategy for treating diabetic wounds by targeting key immune and regenerative pathways. Current research supports their therapeutic potential, but clinical translation requires standardized testing, comparative evaluations, and long-term safety validation. This review highlights the most effective biomaterial messenger RNA combinations and outlines research priorities for future development.



THEME 3: GENETICS/GENOMICS & GENE THERAPY

Abstract ID: 46

Title: Integrated Clinical, Genetic, and Functional Approaches Diagnose Eight Limb-Girdle Muscular Dystrophy Families and Identify Four Novel Variants

Authors: Hammad Yousaf, Lubaba Bintee Khalid, Rafia Zafar Ghumman, Hijab Zahra, Shaheer Hassan, Haq Nawaz Khan, Bilal Ahmad Mian, Asmat Ali, Mathias Toft, Zafar Iqbal, Ambrin Fatima

Affiliation: The Aga Khan University, Karachi

Background and objectives:

Limb girdle muscular dystrophies (LGMD) are genetically heterogeneous group of neuromuscular disorders characterized by overlapping clinical features that includes but not limited to progressive weakness and wasting of the proximal muscles, elevated creatine kinase (CK) levels, motor and skeletal anomalies. Due to the overlapping continuum of phenotypic outcomes with variable severity, diagnosis solely based on clinical features is often prone to errors. Incorporation of genetic investigations to aid diagnosis/ differential diagnosis have been accelerating the diagnostic process, enabling genetic counselling and timely palliative care. The current study was designed to investigate eight families presenting with features of LGMD.

Methods:

We performed exome sequencing (ES), Sanger confirmation, in-silico pathogenicity assessments, combined with functional analysis using RT-qPCR, Western blotting, Immunocytochemitry, to identify the underlying genetic cause of the disease in these families.

Results:

We investigated 24 affected individuals (20 males, 8 females) from nine unrelated families with the age of onset ranging from 0.8 years to 30 years. Our cases universally presented progressive muscle weakness and wasting, elevated CK level, movement difficulties including gait anomaly (24/24), hypotonia (22/22; information for 2 is unavailable- n/a), decreased grip strength (21/24), and motor delay (10/24). ES identified homozygous missense variant, COL12A1: p.(Val1051Leu) in family A. Primary fibroblasts from patient showed low COL12A1 abundance as compared to controls. Family B and C harbor compound heterozygous CAPN3: p.(Arg490Pro); p.(Arg490LeufsTer87) variants while family D segregates biallelic CAPN3: p.(Arg490LeufsTer87) variant. Family E and F carry homozygous splicing variants in DYSF: c.342+1G>A and c.2163-1G>T, respectively. Affected individuals in family G and H possess biallelic LAMA2: p.(Arg2869Glufs*34) and SGCA: p.(Pro55Thrfs*49) respectively. All variants in these families are classified as pathogenic according to the ACMG variant classification criteria.

Conclusion:

Taken together, this study utilized ES to provide genetic diagnosis to the hard-to-diagnose families. This study will help in genetic counselling, parental decision-making, and extended cascade testing to identify the carrier status of the family members.

Abstract ID: 76

Title: Individuals with latent Mycobacterium tuberculosis infection and diabetes display increased inflammatory cytokines, downregulated immunity and dysregulation of metabolic pathways

Authors: KI Masood, M Yameen, J Ashraf, N Ram, N Saifullah, B Jamil, M Irfan, Q Masood, A Ajmal, R Hussain, HM Dockrell, M Rottenberg, JM Cliff and Z Hasan
Affiliation: The Aga Khan University, Karachi

Background:
Latent Mycobacterium tuberculosis (Mtb) infection is asymptomatic and characterized by immune reactivity to Mtb antigens. Type II diabetes (DM) is a common comorbidity associated with worsened tuberculosis (TB) outcomes, with less known about the interactions between DM and latent TB infection (LTBI). DM and TB are both highly prevalent in Pakistan.

Methods:
Participants included individuals with and without DM. LTBi were identified by IGRA test. Th1/ Th2 cytokines were measured in assay supernatants of M. tuberculosis -stimulated in blood from LTBI, DM and DM-LTBI and healthy endemic controls (HC) using a Luminex-based assay. Blood transcriptomics were studied using microarrays followed by GO and KEGG pathways analysis.

Results:
TB-antigen stimulated IL-2, TNF- , GM-CSF and IL-13 were raised in DM-LTBI compared with LTBI group after xx antigen stimulation. Host blood transcriptome analysis of the four groups revealed 359 differentially expressed genes (DEGs); 187 DEGs between LTBI and EC; 181 DEGs between DM and EC, with 13 DEGs between DM-LTBI and EC groups. Compared with EC, LTBI and DM groups both displayed downregulation of antigen presentation pathways (HLA-DQA1, HLADRB5, JCHAIN) and upregulation of inflammatory genes. In DM, immunity related genes (GNLY, KLRC3, PRF1) were downregulated. Comparison between LTBI-DM and LTBI revealed 321 up- and 12 downregulated DEGs, with increased of host adaptive (HLA-DR/DQ), innate immunity (LY96, FCAR, GYPA, IL1R, TREM1) and inflammatory (MMP9, TLRs) pathways with suppression of cell cycle associated genes (AHCTF1, ENSA). Within LTBI, comparison of transcripts from those with uncontrolled DM HDM) with those with controlled DM (CDM) revealed 333 DEGs. HDM was associated with reduction in protein synthesis and oxidative phosphorylation, accompanied with increased cellular migration, catabolic processes and activation of snRNAs. Dysregulated pathways observed in DM and DM-LTBI were common to those in published datasets

Conclusions:
Our novel data show that LTBI and DM synergistically increase host inflammatory and metabolic processes whilst reducing innate immunity. This dysregulation is enhanced in those with uncontrolled hyperglyemia. This likely results in increased risk of developing TB and emphasizes the need for diabetes control in a TB endemic population.

Abstract ID: 59

Title: Investigating Clinical Outcomes in Post-Renal Transplant Recipients Receiving Tacrolimus: A Pharmacogenomic Perspective in the Pakistani Population

Authors: Tooba Noor, Anwar Ali, Quratulain Amir, Muhammad Tassaduq Khan, Farina Hanif
Affiliation: Dow University of Health Sciences, Karachi

Background & Objectives:
Tacrolimus is the drug of choice immunosuppressant in renal transplant (RTx) recipients, but due to its narrow therapeutic index, wider inter and intra patient variability and difficult pharmacogenomics, optimized dose of tacrolimus is difficult to achieve. The aim of this study is to investigate frequency of CYP3A5 and ABCB1 polymorphisms in Pakistani renal transplant patient and their effect on Tacrolimus blood trough levels and the renal functions in terms of clinical outcomes and adverse reactions and rejection.

Methods:
This retrospective cross-sectional study was conducted at Renal transplant Unit of Dow University Hospital, DUHS. A total of 83 subjects who have received renal transplant and on tacrolimus for more than one year have been recruited for genotyping analysis (via primer specific PCR, Gel electrophoresis and RFLP) of CYP3A5 and ABCB1 polymorphisms in association with pharmacokinetic details of tacrolimus and relevant clinical outcomes.

Results:
In this study, 79.8% (n = 64) were males, the median duration since transplantation was 28 months while, 15.6% (n = 10) were pre-emptive transplant. Post transplant Diabetes Mellitus was common in 57.83% (n = 48) of subjects. The mean daily dose of tacrolimus was 2.5 ± 0.5mg, and mean tacrolimus blood trough levels were 7.6 ± 3.7 mg/dl, 7.1 ± 2.1 mg/dl and 5.9 ± 3.9 mg/dl at 0, 3 and 6 months, respectively. All subjects were tested CYP3A5*1/3 (non-expressors) genotype while homozygous mutants ABCB1 rs1045642 and rs2032582 have considerably higher blood trough concentration levels as compared to heterozygous or homozygous wild type. Significant association of higher neutrophils/ platelets ratio, higher CRP levels, frequent infections were reported in homozygous mutants ABCB1 rs2032582 while CYP3A5*1/3 non expressors has significantly increased fasting blood sugar levels, hypertriglyceridemia and hyperuricemia.

Conclusions:
Our findings served as a pilot study relating to pharmacogenomics of CYP3A5 and ABCB1 polymorphisms of Pakistan population with renal transplant. We found significant influence of these polymorphisms in achieving targeted tacrolimus blood trough levels leading to abnormally higher rate of adverse effects which may affect long term graft functions. It is highly recommended that timely detection of theses notable polymorphisms may reduce the titration time in tacrolimus dose optimization thus reducing side effects and improving the outcomes.

Abstract ID: 38
Title: Transcriptomic Profiling Identifies RELN–PDE3A as a Peripheral Blood Gene Signature in Pakistani Children with Autism Spectrum Disorder
Authors: Farah Wazir, Saira Amir, Nazia Bibi, Sumreena Mansoor, Aisha Naeem and Muhammad Jawad Khan
Affiliation: COMSATS University, Islamabad

Background:
Autism Spectrum Disorder (ASD) is a complex neurodevelopmental condition with rising prevalence and limited molecular diagnostic tools, particularly in low-resource settings. Identifying blood-based biomarkers may support early screening and culturally relevant interventions. This study integrates bioinformatics and wet-lab validation to identify peripheral gene-expression signatures associated with ASD in Pakistani children.

Objective:
To identify differentially expressed genes (DEGs) linked to ASD and validate a diagnostic gene signature using peripheral blood samples from Pakistani case–control sibling pairs.

Methods:
Public microarray datasets (GSE143155, GSE65106, GSE162954) were analyzed using GEO2R with Benjamini–Hochberg FDR correction ($p < 0.05$; $|\log_2 FC| \geq 1$). Functional enrichment (DAVID, KEGG) and STRING-based protein–protein interaction (PPI) analyses were applied to prioritise hub genes. Peripheral blood RNA from 40 ASD children and 40 neurotypical sibling controls was analyzed through SYBR-green RT-qPCR. Fold-changes were computed using the ΔΔCt method, and significance was assessed with Mann–Whitney U tests (FDR-adjusted).

Results:
Bioinformatics screening identified 67 overlapping DEGs across datasets, converging on PI3K–Akt signaling, ECM–receptor interaction, and morphine signaling pathways. Seven hub genes (RELN, PDE3A, COL6A1, NFKB1, PRKCA, TGFA, GABRA1) were shortlisted. RT-qPCR validation revealed significant upregulation of RELN (4.3-fold; FDR = 0.049) and PDE3A (4.8-fold; FDR = 0.041), while remaining genes showed non-significant trends. Gene-expression differences were independent of sex, socioeconomic status, screen exposure, and clinical severity. A two-gene diagnostic model (RELN + PDE3A) achieved an AUC > 0.80, indicating strong predictive potential.

Conclusion:
This study identifies a reproducible RELN–PDE3A peripheral blood signature capable of distinguishing ASD children from matched sibling controls. The findings highlight dysregulated PI3K–Akt and cyclic-nucleotide pathways in ASD and present a promising low-cost biomarker pair suitable for validation in multicentre South-Asian cohorts. These results advance the development of accessible precision-diagnostic tools for ASD in resource-limited settings.

Abstract ID: 43
Title: Cadmium Sulphate Induces Pro-Atherogenic Phenotypic Switching in Human Aortic Smooth Muscle Cells
Authors: Shafaq Ramzan, Brishna Khan, Mati-Ur-Rehman, Satwat Hashmi
Affiliation: The Aga Khan University, Karachi

Background & Objectives:
Vascular smooth muscle cells (VSMCs) are essential for maintaining vascular integrity. Their ability to transition from a quiescent, contractile state to a synthetic, migratory, and proliferative phenotype is a critical driver of atherosclerosis. Environmental cadmium, common in cigarette smoke and industrial emissions, is epidemiologically associated with coronary artery disease, yet its mechanistic impact on VSMC behavior remains unclear. This study investigated whether cadmium sulphate ($CdSO_4$) promotes phenotypic switching in human aortic smooth muscle cells (HASMCs) and explored the molecular and functional consequences.

Methods:
HASMCs from ATCC were cultured under standard conditions and exposed to $CdSO_4$. Cell viability was assessed using CCK-8, and three concentrations (1 µM, 15 µM, 25 µM) were selected for downstream experiments. Targeted qPCR assessed expression of contractile markers (ACTA2, TAGLN), osteogenic marker (BMP2), and inflammatory marker (CD68). Functional assays at 1 µM $CdSO_4$ included scratch-wound migration and Ki-67 immunostaining to evaluate proliferation. Global molecular changes were assessed using transcriptomic profiling.

Results:
$CdSO_4$-treated HASMCs exhibited clear phenotypic switching. Targeted qPCR showed upregulation of BMP2 and CD68 and downregulation of ACTA2 and TAGLN, indicating loss of contractile identity and acquisition of osteogenic and inflammatory characteristics. Functional assays demonstrated enhanced migration and increased proliferation, consistent with a synthetic, pro-atherogenic phenotype. Transcriptomic analysis performed at 1 µM $CdSO_4$ revealed extensive differential gene expression, including upregulation of genes associated with migration, inflammation, and proliferation (e.g., CDH6, HGF, F2RL1), supporting the observed functional changes.

Conclusions:
Cadmium sulphate drives phenotypic switching in HASMCs, evidenced by loss of contractile markers, upregulation of osteogenic and inflammatory genes, enhanced migration and proliferation, and broad transcriptomic reprogramming. These findings provide mechanistic insight into the pro-atherogenic effects of environmental cadmium and highlight the importance of mitigating heavy metal exposure to prevent vascular remodeling and atherosclerosis.

Abstract ID: 45

Title: A homozygous founder variant in DDHD2 causes hereditary spastic paraplegia in three consanguineous Pakistani families.

Authors: Bilal Ahmed Mian, Hammad Yousaf, Rafia Zafar Ghumman, Zafar Ali, Asmat Ali, Farhan Bahadur Ali, Ghazala Zafar, Mathias Toft, Ambrin Fatima, Zafar Iqbal
Affiliation: The Aga Khan University, Karachi

Background/Objective:
DDHD2 (DDHD Domain Containing 2) is implicated in autosomal recessive spastic paraplegia 54 (MIM: 615033). The disease spectrum includes delay in psychomotor development, intellectual disability and early-onset spasticity in lower limbs. This study was designed to identify the underlying genetic cause of three Pakistani families with rare hereditary spastic paraplegia.

Methods:
We performed detailed clinical assessment and exome sequencing (ES) to identity plausible disease-causing variants. Segregation of the prioritized variant was confirmed by Sanger sequencing. Further, we also performed haplotype analysis to assess the founder effect of the identified variant.

Results:
We identified seven cases from three consanguineous Pakistani families presenting developmental disability including peripheral neuropathy, muscular atrophy, and developmental delay. ES identified a founder homozygous missense variant in DDHD2 (NM_015214.3): c.2065G>T:p.D689Y (classified as likely-pathogenic as per ACMG criteria i.e. PM1, PM2, PP1, PP3) as the most likely cause of the disease. The variant exists in a 2.06 Mb stretch shared among three families and segregates with the disease in recessive pattern. Since DDHD2 is known to cause spastic paraplegia 54, we compared the clinical presentation of our cases with the reported phenotype. MRI findings showed no morphological anomalies of the brain as compared to thin corpus callosum and white matter anomalies as already reported.

Conclusion:
Our findings add to the mutation spectrum of DDHD2-related condition. Moreover, we have initiated a set of functional experiments to unravel the plausible pathogenicity of the identified variant in this study.

Abstract ID: 49
Title: Homozygous ASTN1 Nonsense Variant Associated with Severe Neurodevelopmental Phenotypes in a Consanguineous Pakistani Family
Authors: Muhammad Saad Waheed Abbasi, Lubaba Bintee Khalid, Sajid Ali, Matias Toft, Zafar Iqbal, Ambrin Fatima
Affiliation: The Aga Khan University, Karachi

Background & Objectives:
ASTN1 encodes astrotactin-1, a neuronal adhesion protein essential for glial-guided neuronal migration during brain development. Pathogenic variants in ASTN1 have been rarely reported, and genotype–phenotype correlations remain poorly defined. We report a family-based case series describing a homozygous nonsense variant in ASTN1 identified in multiple affected siblings. The objective of this study was to characterize the phenotypic spectrum and provide a genetic diagnosis to support ASTN1 as a candidate gene for neurodevelopmental disorders.

Methods:
This family-based case report includes six siblings from a consanguineous family, four of whom were affected. Exome sequencing was performed. Variant filtering and identification were performed using standard bioinformatic pipelines and public variant databases. Clinical evaluation was based on detailed medical history. Reverse genotyping and segregation analysis are pending, and Sanger sequencing is planned for validation.

Results:
All four affected siblings presented with childhood-onset severe neurodevelopmental impairment. Core clinical features included global developmental delay, severe intellectual disability, abnormal gait with spasticity, and variable seizure history. Speech impairment was profound, with absent speech in one individual at 11 years of age. Sleep disturbances were also reported. Notably, one affected sibling did not have a history of seizures, highlighting intra-familial phenotypic variability. Two siblings in the family were unaffected. A homozygous nonsense variant in ASTN1: c.C3334T; p.R1112X (NM_001286164) was identified, previously reported once in ClinVar as likely pathogenic. This is the first reported Pakistani family with a homozygous ASTN1 loss-of-function variant.

Conclusions:
This report expands the phenotypic spectrum associated with homozygous loss-of-function variants in ASTN1. The presence of a shared homozygous nonsense variant in four affected siblings from a single consanguineous family supports ASTN1 as a strong candidate gene for severe neurodevelopmental disorders. The combination of familial clustering, phenotypic diversity and absence of OMIM annotation underscores the need for additional case studies, segregation analyses and functional validation to definitively establish ASTN1’s role in human neurodevelopment.

Abstract ID: 77
Title: Deciphering COVID-19 Severity through Mitochondrial DNA in Pakistani population: A Machine Learning Approach to Predicting COVID19 severity
Authors: Sayed Ali Raza Bukhari, Yusra Abdul Rehman, Javaria Ashraf, Asghar Nasir, Saad Farooq, Zahra Hasan
Affiliation: The Aga Khan University, Karachi

Background & Objectives:
The clinical trajectory of COVID-19 varies significantly between individuals, suggesting a strong underlying genetic component. Emerging evidence points to the mitochondrial genome (mtDNA) as a key regulator of the host immune response and metabolic resilience. This study aimed to identify specific mtDNA variants associated with disease severity and to develop a predictive machine learning tool to distinguish between Non-Severe (Asymptomatic/Mild) and Severe (Moderate/Critical) outcomes.

Methods:
Mitochondrial DNA was sequenced and analyzed from four clinical groups: Healthy Controls (n=40), Asymptomatic/Mild (n=64), Moderate (n=43), and Critical/Severe (n=40) COVID-19 subjects. Haplogroups and mitochondrial genome variants were identified. In significantly different variants we implemented a Random Forest (RF) classification model using an 80/20 train-test split. To enhance clinical utility, groups were merged into a binary classification: Non-Severe (HC+AMD) and Severe (MOD+CS). Predictive performance was evaluated using Receiver Operating Characteristic (ROC) analysis and confusion matrices, while feature importance ranking identified the primary genetic drivers of severity.

Results:
Haplogroup analysis showed major haplogroup M to be negatively associated with COVID-19 and CS disease. Whereas, haplogroup U showed a positive association with CS disease. Haplogroup W was associated with an increased AMD COVID-19. The binary ML model demonstrated robust performance, achieving an estimated accuracy of 75-82% and an AUC-ROC > 0.85, indicating high discriminatory power. Variants in MT-ATP6 was identified as the strongest independent predictor of severe disease (Risk Weight: +3.8), followed by 16311T>C in the D-loop. Conversely, variant 14128A>G (MT-ND5) emerged as a significant protective marker (Weight: -3.5) prevalent in the non-severe cohort. ROC analysis confirmed that the model effectively captures the "metabolic bottleneck" associated with hospitalization risk.

Conclusions:
This study demonstrates that mitochondrial variants are potent independent predictors of COVID-19 severity. The development of a weighted scoring tool provides a potential framework for early clinical screening, allowing for the identification of high-risk individuals based on their mitochondrial genetic "signature". Since there is a limited dataset depending on the overall population. Large scale data will be required to make firm decisions from this pilot study.

Abstract ID: 21

Title: Early Synaptic Dysfunction in C9orf72 ALS/FTD: Insights from Human Organoid Models

Authors: Daniya Jawed, Ramsha Haider, Nashmiya Khan, Safa Irfan Shah, Shifa Imran, Wajeeha Siddiqui, Zoha Iftikhar
Affiliation: Karachi Medical and Dental College

Background:
Hexanucleotide repeat expansions in C9orf72 are the most common genetic cause of amyotrophic lateral sclerosis (ALS) and frontotemporal dementia (FTD). Organoid technology enables modeling of early human-specific synaptic and developmental abnormalities, providing insights into disease mechanisms and therapeutic targets.

Introduction:
Traditional models incompletely capture human-specific synaptic processes disrupted in C9orf72 ALS/FTD. iPSC-derived cerebral, spinal, and neuromuscular organoids allow study of early pathogenic events, including synaptic dysfunction that may precede neurodegeneration. This review maps organoid research, summarizes synaptic and molecular abnormalities, and highlights translational insights.

Methods:
A literature search (2015–2024) was conducted using Consensus, PubMed, and Google Scholar with keywords: “C9orf72,” “ALS,” “FTD,” “organoids,” “iPSC,” and “synaptic dysfunction.” Studies using human iPSC-derived organoids reporting synaptic, neuronal, or molecular outcomes were included. Data on organoid type, functional assays, pathological findings, and therapeutic testing were extracted and synthesized qualitatively.

Results:
Seventeen studies were included. Across cerebral, spinal, and neuromuscular organoids, early and progressive synaptic dysfunction was consistently observed, including reduced excitatory synapses, altered synaptic protein expression (synapsin, SYP, SHANK2), impaired excitability, and disrupted network activity. Organoids from presymptomatic carriers showed early molecular pathology, indicating synaptic impairment precedes neurodegeneration. Mechanistic insights implicated C9orf72 haploinsufficiency and toxic gain-of-function mechanisms, including RNA foci, dipeptide repeat proteins, and altered neural progenitor composition. Several studies demonstrated therapeutic effects, such as restoration of synaptic function following unfolded protein response inhibition.

Discussion:
Organoid models reveal early, cell type–specific synaptic dysfunction in C9orf72 ALS/FTD driven by developmental and molecular mechanisms. They capture presymptomatic stages inaccessible in patients and support preclinical drug testing. Limitations include iPSC variability, incomplete maturation, and limited modeling of glial or systemic interactions, but these models remain crucial for understanding disease onset and identifying therapeutic targets.

Conclusion:
Organoid studies show that early synaptic dysfunction is a hallmark of C9orf72 ALS/FTD, preceding neuronal loss and driven by both loss- and gain-of-function mechanisms. Human organoids are valuable for elucidating disease onset and guiding development of targeted therapies.

Abstract ID: 30
Title: Genetic Profiling and Community-Based Initiatives for Limb Girdle Muscular Dystrophy in Pakistan
Authors: Ghulam Ali
Affiliation: Muscular Dystrophy Pakistan, Jacobabad

Background & Objectives:
Limb Girdle Muscular Dystrophy (LGMD) is a rare genetic disorder with diverse clinical presentations. In Pakistan, there is limited data on genetic variants, awareness, and early diagnosis strategies. This study aims to profile the genetic landscape of LGMD patients, assess the impact of awareness initiatives, and implement early diagnostic approaches through newborn screening.

Methods:
Data from 200 MD patients were collected via surveys, case studies, and literature review. LGMD Awareness Day was conducted on 30 September 2025 under the umbrella of EveryLife Foundation for Rare Diseases. Genetic testing campaigns were coordinated with Aga Khan University Hospital, Karachi, to ensure accurate subtype diagnosis. Early diagnostic strategies include initiating a newborn screening program for Muscular Dystrophies.

Results:
Genetic profiling identified diverse LGMD mutations, highlighting the need for precise diagnosis. Awareness campaigns enhanced patient engagement and understanding of disease management. Collaboration with AKUH facilitated accurate diagnosis and genetic counseling. Planned newborn screening is expected to enable early identification and intervention for at-risk infants.

Conclusions:
Integrating genetic profiling with community awareness and early diagnostic programs improves identification, care, and management of LGMD patients. Collaboration with national and international organizations enhances the translational impact of rare disease research.

Abstract ID: 37

Title: Clinical and Molecular Characterization of Pakistani Families with Sanfilippo and Morquio Syndromes

Authors: Shumaila Zulfiqar, Farheen Nasir Awan, Liza Eiman, Maria Asif, Muhammad Sajid Hussain, Niklas Dahl, Shahid Mahmood Baig, Hirotsugu Oda
Affiliation: Kinnaird College for Women, Lahore

Background & Objectives:
Mucopolysaccharidoses (MPS) are rare lysosomal storage disorders caused by defective glycosaminoglycan-degrading enzymes, leading to multisystem disease. Limited genetic data exist for Pakistani populations, which exhibit high consanguinity. This study aimed to characterize the clinical features and identify pathogenic gene variants in two unrelated consanguineous Pakistani families presenting with MPS.

Methods:
Six affected individuals from two families underwent comprehensive clinical evaluation. Whole-exome sequencing (WES) was performed, followed by segregation analysis via Sanger sequencing. In silico structural modeling assessed the functional impact of identified variants. Pathogenicity was evaluated using ACMG criteria and multiple bioinformatic tools.

Results:
Family 1 (three affected siblings) presented with developmental regression, severe intellectual disability, behavioral disturbances, and mild facial dysmorphisms. WES revealed a homozygous missense variant in SGSH (c.548G>A; p.Cys183Tyr) within the sulfatase catalytic domain, predicted to destabilize protein structure and disrupt enzymatic function. Family 2 (three affected siblings) showed classical skeletal dysplasia consistent with Morquio syndrome. A homozygous splice-site variant in GALNS (c.423-1G>A) segregated with disease and is predicted to cause exon skipping and loss of function. Both variants segregated with disease in their respective families, and bioinformatic analyses supported their pathogenicity.

Conclusions:
This study expands the genetic spectrum of MPS in Pakistan by reporting a previously uncharacterized SGSH variant and confirming a regional GALNS variant. The findings highlight the value of genomic diagnostics for early detection, accurate classification, and genetic counseling in populations with high consanguinity. Integration of clinical assessment with molecular analysis enables informed reproductive planning and improved patient management.

Abstract ID: 44
Title: Whole-Exome Sequencing Identifies Adjacent Pathogenic TTN Variants in a Family with Adult-Onset Hypertrophic Cardiomyopathy
Authors: Shafaq Ramzan, Ayesha Tahir
Affiliation: The Aga Khan University, Karachi

Background & Objectives:
Hypertrophic cardiomyopathy (HCM) is a genetically heterogeneous cardiac disorder most often caused by mutations in sarcomeric genes. While truncating variants in the TTN gene are a major cause of dilated cardiomyopathy, missense variants in specific titin regions have also been implicated in HCM, though their pathogenic role remains unclear. The objective of this study was to identify the genetic basis of adult-onset HCM in a Pakistani family with a history of sudden cardiac death.

Methods:
A consanguineous Pakistani family with four affected individuals was clinically evaluated using echocardiography and electrocardiography. Whole-exome sequencing was performed on two affected and one unaffected family member, followed by variant filtering under an autosomal dominant inheritance model. Candidate variants were assessed using multiple in silico pathogenicity prediction tools. Segregation analysis and variant validation were carried out by Sanger sequencing in all available family members.

Results:
Two novel heterozygous missense variants in exon 28 of the TTN gene—c.6230C>T (p.Ala2077Val) and c.6232C>T (p.Pro2078Ser), affecting adjacent codons in the Z-disc/I-band transition region were identified. Both variants co-segregated with the HCM phenotype, were absent in unaffected individuals, and were not reported in public databases. The affected amino acids were highly conserved across species. In silico prediction tools (PolyPhen-2, CADD, DANN, MutationTaster, and GERP) consistently classified both variants as deleterious. Clinically, affected individuals presented with adult-onset mild HCM, with a mean left ventricular end-diastolic diameter of 46 ± 9 mm and a mean ejection fraction of 38 ± 12%.

Conclusions:
This study identifies two novel adjacent TTN missense variants likely exerting a synergistic effect in causing mild, adult-onset hypertrophic cardiomyopathy. These findings expand the mutational spectrum of TTN associated with HCM and highlight the importance of detailed genetic and clinical evaluation in familial cardiomyopathies.

Abstract ID: 71

Title: A Homozygous Canonical Splice-Site Variant in SAMHD1 Expands the Phenotypic Spectrum of Aicardi-Goutières Syndrome Type 5: A Case Report from a Consanguineous Family

Authors: Zehra Zonash, Javeria Manzoor, Hammad Yousaf, Mathias Toft, Zafar Iqbal, Ambrin Fatima.

Affiliation: The Aga Khan University, Karachi

Background:

Aicardi-Goutières syndrome type 5 (AGS5) is a rare pediatric-onset monogenic interferonopathy caused by loss-of-function variants in SAMHD1. Affected individuals typically present in infancy with microcephaly, leukodystrophy, intracranial calcifications, developmental delay, and spasticity. However, phenotypic heterogeneity and atypical systemic manifestations have been increasingly recognized.

Methods:

We performed whole exome sequencing, In silico pathogenicity prediction, and Sanger sequencing in a Pakistani family.

Case presentation:

We report a consanguineous Pakistani family with two siblings affected by AGS5. The proband, a 9-year-old female, presented with congenital-onset neurodevelopmental delay, microcephaly (head circumference 46 cm), dysarthria, bilateral lower-limb muscular atrophy, and mild planovalgus deformity. She never attained independent ambulation and had only rudimentary speech. Notably, spasticity, seizures, and aggressive behavior were absent. Whole-exome sequencing identified a homozygous canonical splice-site variant in SAMHD1 (NM_015474: exon13: c.1503+1G>A), predicted to abolish normal splicing and classified as pathogenic under ACMG criteria (PVS1, PM2, PP1). Segregation analysis confirmed autosomal recessive inheritance, with both parents heterozygous carriers and three affected siblings, one of whom had died in early childhood.

Conclusion:

This report expands the mutational and phenotypic spectrum of SAMHD1-related AGS. The identification of a canonical splice-site variant in a consanguineous South Asian family underscores the importance of considering AGS in children with congenital microcephaly and progressive neurodevelopmental impairment, even in the absence of seizures or spasticity.

Abstract ID: 75

Title: Integrative Clinical and Genomic Characterization of Familial Hypercholesterolemia (FH) in Pakistan: Toward Population-Tailored Diagnostics and Therapy

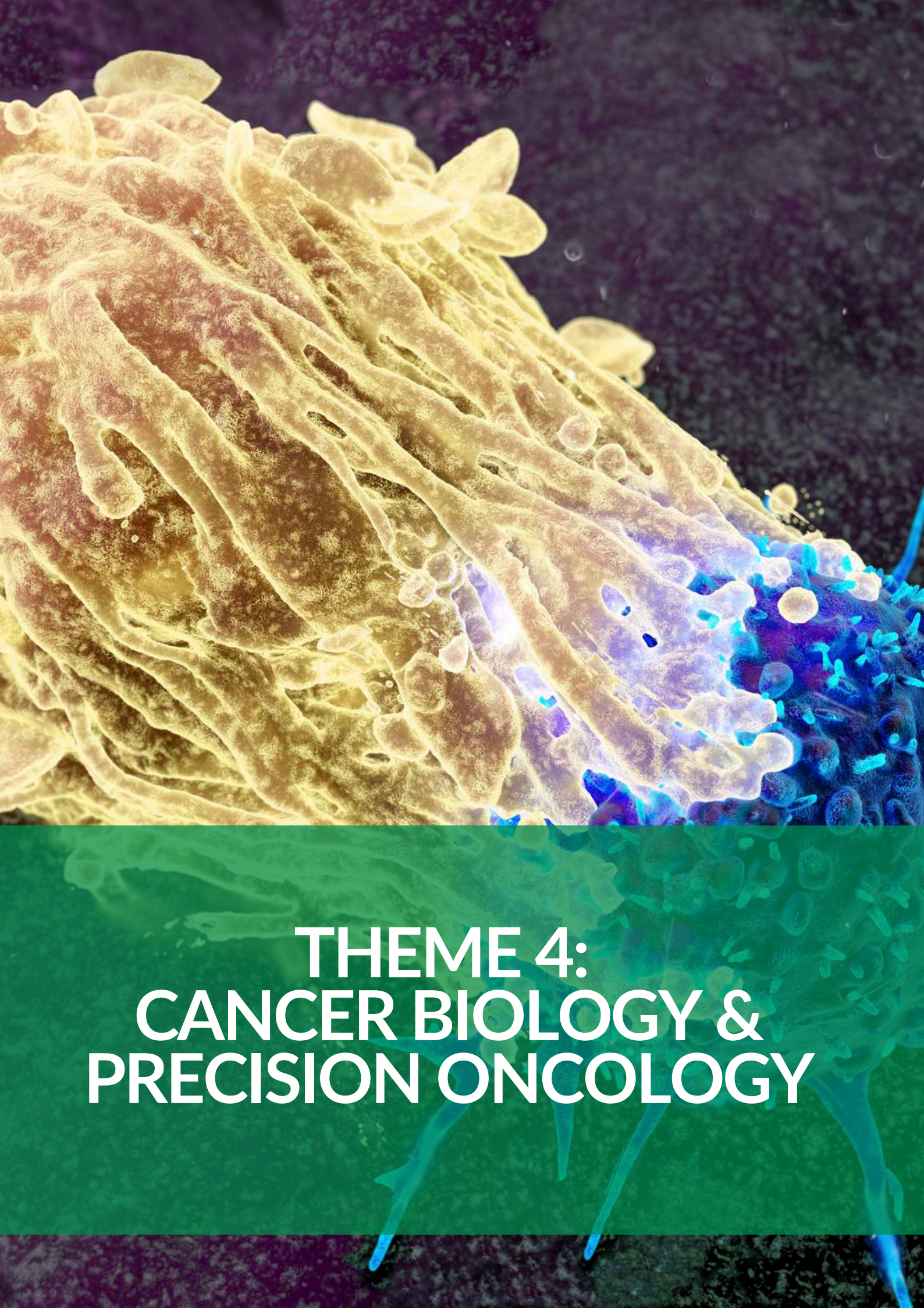
Authors: Ayaz Khan, Umer Naeem Effindi, Haq Nawaz Khan, Hafsa Majid. Salman Kirmani, Fawad Ur Rehman, Saleem Verani, Aysha Habib khan, and Afsar Ali Mian.
Affiliation: The Aga Khan University, Karachi

Rationale and Objectives:
Familial hypercholesterolemia (FH) is an inherited metabolic disorder caused by impaired clearance of LDL cholesterol, leading to severe dyslipidemia and premature coronary heart disease. Current FH variant databases are overwhelmingly Eurocentric, limiting diagnostic accuracy and therapeutic relevance for ~80% of the global population, including South Asian cohorts. This study aims to comprehensively characterize the clinical and genetic landscape of FH in Pakistan to inform population-specific diagnosis and precision therapies.

Methodology:
Between January and December 2024, we conducted systematic clinical screening at Aga Khan University Hospital, identifying 309 Pakistani patients meeting stringent FH criteria. Whole-exome sequencing was performed on nine multiplex consanguineous families, with two affected individuals sequenced per family. Variants in FH-associated genes (LDLR, APOB, PCSK9, LDLRAP1) were prioritized using population-matched and clinical annotation filters.

Results:
We observed an estimated FH prevalence of ~1:575 among screened individuals, with a median age of 29 years. Clinically, patients presented severe phenotypes: claudication (68.8%), tendon xanthomas (47.5%), and xanthelasmas (42.6%), all significantly correlated with elevated LDL-C ($p < 0.05$). Genetically, we identified several rare homozygous and compound heterozygous variants in LDLR, including splice-site mutations (c.2312-1G>A, c.1060+2T>G) and missense variants (p.Cys148Tyr, p.Cys104Tyr). A likely founder mutation (p.Asp354Gly) and a VUS (p.Gly549Arg) awaiting Sanger validation were also detected. Additionally, we observed an APOB variant of uncertain significance (p.Glu2414Val) and a homozygous frameshift mutation in LDLRAP1 (p.Gly25fs), underscoring the prominence of recessive inheritance patterns in this consanguineous population.

Conclusion:
This integrated clinical and genomic profiling of the largest reported Pakistani FH cohort establishes a critical foundation for developing tailored diagnostic panels and precision therapies, including CRISPR-Cas9, prime editing, and antisense oligonucleotides optimized for the genetic architecture of South Asian populations.



THEME 4: CANCER BIOLOGY & PRECISION ONCOLOGY

Abstract ID: 47

Title: Genomic Landscape and Association of Selected Stem Cell Markers in Pancreatic Ductal Adenocarcinoma

Authors: Saleema Mehboob Ali, SM Adnan Ali, Yumna Adnan, Zubair Ahmad, Iqbal Azam
Affiliation: The Aga Khan University, Karachi

Background and objective:
Pancreatic ductal adenocarcinoma (PDAC) is among the most aggressive malignancies, characterized by poor prognosis and limited therapeutic options. Molecular biomarkers are central to precision oncology; however, their expression and prognostic relevance may vary across populations. To date, the molecular and stemness-associated biomarker landscape of Pakistani patients with PDAC remains largely unexplored. This objective of the study is to characterize key genetic alterations and associated cancer stem cell (CSC) markers in PDAC from the Pakistani population and to evaluate their prognostic relevance.

Methods:
This study extends our pilot PDAC investigation, which identified pathogenic variants in KRAS, TP53, BRCA1 and APC using an 88-gene panel. Based on these findings, we designed a targeted analysis in a statistically powered cohort. A total of 109 histologically confirmed PDAC Formalin-fixed paraffin-embedded (FFPE) tumor samples were obtained. Genomic DNA was extracted, followed by primer-specific PCR for KRAS, TP53, BRCA1 and APC and Sanger sequencing. In parallel, three cancer stem cell markers (CD24, CD44 and CD133) were selected based on their functional relevance to the pathways involving the identified genes. These markers were evaluated using immunohistochemistry to explore associations between genetic alterations and stemness-related phenotypes.

Results:
A total of 59 genetic variants were identified across the selected regions four genes, of which 22% were classified as pathogenic. Pathogenic variants in TP53 were most frequent (75.2%), with all affected cases harboring a recurrent TP53 c.730G>A variant. Multivariate analysis identified TP53 c.730G>A as an independent prognostic marker (adjusted HR: 2.428; 95% CI: 1.330–4.435; p = 0.004). BRCA1 alterations showed significant associations with concurrent mutations in KRAS (p = 0.001), TP53 (p = 0.008) and APC (p = 0.000). Independent mutations in TP53 (p = 0.001) and KRAS (p = 0.001) were significantly associated with overall survival. Among CSC markers, CD24 expression was significantly associated with the TP53 pathogenic variants, while CD133 expression showed a significant association with BRCA1 genetic alterations. No statistically significant association was observed for CD44. None of the CSC markers was found to be associated with overall survival of the patient.

Conclusion:
The pathogenic variant TP53 c.730G>A showed an exceptionally high frequency and was also found to be an independent prognostic biomarker in the cohort. This finding shows that TP53 c. 730G>A may be a plausible biomarker in the Pakistani PDAC population. Moreover, the significant associations between key genetic alterations and CSCs (CD24 and CD133) highlight a potential link between genomic instability and stemness-driven tumor biology. These results support integrating genomic and stem cell markers into precision oncology strategies.

Abstract ID: 79

Title: Helix-2–TAT Peptide–Engineered Mesenchymal Stem Cells as a Therapeutic Strategy for Philadelphia Chromosome–Positive Leukemia

Authors: Rida-e-Maria Qazi, Zahra Sajid, Shariqa Khawaja, Rena Zaman, Seema Inayat, Irfan Hussain, Sujjawal Ahmed, Syed Muhammad Areeb Ahmed, Tooba Tajammul, Fawad Ur Rehman and Afsar Ali Mian

Affiliation: The Aga Khan University, Karachi

Background:

Philadelphia chromosome–positive (Ph+) leukemia is a hematologic malignancy driven by the constitutively active BCR-ABL fusion oncoprotein. Tyrosine kinase inhibitors (TKIs) are the current standard of care and have significantly improved patient outcomes; however, long-term TKI therapy is limited by adverse effects, the development of drug resistance, and the need for lifelong treatment. These limitations necessitate the development of alternative therapeutic strategies. BCR-ABL oncogenic activity depends on tetramer formation mediated by its N-terminal coiled-coil (CC) domain. Disruption of this structural interaction represents a promising non–kinase-based therapeutic approach. Previously, we demonstrated that competitive peptides, including the helix-2 (H2) peptide, can interfere with CC-domain–mediated tetramerization and suppress BCR-ABL oncogenic function. To enhance intracellular delivery, HIV-TAT–tagged H2 peptides were developed. Aims: The aim of this study was to establish and evaluate a novel therapeutic strategy for Ph+ leukemia using mesenchymal stem cells (MSCs) engineered to continuously secrete HIV-TAT–tagged H2 peptides targeting BCR-ABL tetramerization.

Methods:

MSCs were genetically modified to constitutively secrete HIV-TAT–tagged H2 peptides designed to disrupt BCR-ABL tetramerization through its N-terminal CC domain. The therapeutic efficacy of engineered MSCs was assessed in vitro and in vivo using Ph+ leukemia models, with analyses of leukemia cell proliferation, apoptosis, specificity of peptide delivery, and durability of therapeutic effects.

Results:

H2-TAT peptides were predominantly released as soluble molecules, with a fraction associated with extracellular vesicles. These secreted peptides effectively inhibited the proliferation and viability of Ph+ leukemic cells in vitro. In murine leukemia models, treatment with H2-TAT–secreting MSCs significantly improved survival and reduced splenomegaly.

Conclusion:

Our findings demonstrate that MSC-based paracrine delivery of H2-TAT peptides represents a novel and effective strategy for targeting BCR-ABL–driven leukemia. Continuous peptide secretion by engineered MSCs enables sustained disruption of BCR-ABL function and suppresses disease progression in preclinical models. This approach offers a clinically translatable alternative or complement to TKI therapy, with the potential to overcome drug resistance, reduce systemic toxicity, and minimize lifelong treatment dependence.

Abstract ID: 24

Title: Z-36, a novel Bcl-xL inhibitor, induces oxidative stress mediated non-apoptotic and non-autophagic cell death in colorectal cancer HCT-116 cells

Authors: Almuayyad Gajani, Mahwish Fatima, Mati Ur Rehman
Affiliation: The Aga Khan University, Karachi

Background:
Colorectal cancer (CRC) is among the most prevalent malignancies worldwide and a leading cause of cancer-related mortality. Resistance to apoptosis remains a major therapeutic barrier, often mediated by the overexpression of anti-apoptotic Bcl-2 family proteins such as Bcl-xL. Z-36, a novel indolin-2-one derivative and inhibitor of Bcl-2/Bcl-xL, has been reported to trigger autophagy-associated and caspase-independent cell death in other cancer models. However, its mechanistic role in mediating cell death via ROS remains unclear. This study is, therefore, aimed to investigate the cytotoxic potential of Z-36 and elucidate whether its effects on human colorectal carcinoma HCT-116 cells involve reactive oxygen species (ROS)-mediated non-apoptotic cell death.

Methods:
HCT-116 cells were treated with varying concentrations of Z-36 (0–10 μM) for 48 hr. Cytotoxicity was quantified by CCK-8 assay, and morphological alterations were evaluated by phase-contrast and Giemsa staining. Apoptotic status was analyzed using Annexin V–FITC/Propidium Iodide (PI) double staining via flow cytometry, while cell-cycle distribution was assessed by PI-based DNA content analysis. The involvement of oxidative stress was confirmed by pre-treatment with the ROS scavenger N-acetyl cysteine (NAC). Western blotting was performed to determine the expression of caspase-3 proteins to evaluate the apoptotic treated cells.

Results:
Z-36 significantly reduced HCT-116 cell viability in a dose-dependent manner. Microscopic examination revealed characteristic cytoplasmic vacuolization beginning at 2.5 μM and intensifying at 5 μM, while severe cell rounding and detachment occurred at 10 μM. Annexin V–FITC/PI analysis demonstrated a dose-dependent increase in apoptotic cell populations, whereas cell-cycle analysis showed accumulation in the Sub-G1 phase, reflecting DNA damage and growth inhibition. Co-treatment with NAC completely suppressed vacuolization and preserved cell morphology, confirming the ROS-dependent nature of Z-36-induced cytotoxicity. Western blot analysis revealed unaltered pro-caspase-3 with no detectable cleavage, indicating caspase-independent cell death.

Conclusion:
Collectively, these findings demonstrate that Z-36 at the dose of 5 μM induces ROS-mediated, non-apoptotic cell death in HCT-116 colorectal cancer cells independent of caspase activation. The ability of NAC to rescue cells from Z-36-induced damage highlights oxidative stress as the central mechanism driving its cytotoxicity.

Abstract ID: 11

Title: E3 ubiquitin ligase TRIP12 controls protein translation and promotes cancer cell survival via a potential mTOR/G3BP1 nexus

Authors: Amira Kohil, Munia Hamdan, Adam Dowle, Lutfiye Ozer, Kripa Keyan, Aiman Bushra, Emma Embarek, Bini Sreenesh, Ayman Al Haj Zen, Omar Khan

Affiliation: Hamad Bin Khalifa University, Doha, Qatar

Background:

Thyroid Hormone Receptor Interacting Protein 12 (TRIP12) is an E3 ubiquitin ligase associated with neurological disorders, including Clarke-Baraitser syndrome and autism spectrum disorder, as well as various cancers. Its role varies depending on the biological context, with recent research indicating its involvement in the DNA damage response, TGF-β, WNT signaling, and chemotherapy responses. This study examines the role of TRIP12 in regulating protein translation through mTOR signaling.

Methodology:

We utilized quantitative proteomics to investigate the function of TRIP12 in protein translation across multiple human cancer cell lines. By genetically inhibiting TRIP12, we assessed protein translation using puromycin and its analogue, O-propargyl-puromycin (OPP). We evaluated cellular characteristics, including size, migration, and tumor spheroid formation, and analyzed mTOR signaling by examining the phosphorylation of p70 S6 kinase and S6 Ribosome. We also studied the interaction between TRIP12 and G3BP1, a negative regulator of mTOR, using co-immunoprecipitation followed by wester blotting.

Results:

Inhibition of TRIP12 significantly decreased protein translation across various cell lines, resulting in smaller cell sizes, reduced migration, and impaired formation of tumor spheroids. TRIP12-deficient cells showed persistently lower mTOR activity, evident from decreased phosphorylation of p70 S6 kinase and S6 Ribosome. Mechanistically, TRIP12 depletion resulted in less monoubiquitination of G3BP1, strengthening its association with mTOR. Co-deletion of G3BP1 and TRIP12 mitigated the phenotypes caused by the absence of TRIP12.

Conclusion:

Overall, TRIP12 is crucial for cellular protein translation via the mTOR/G3BP1 pathway, underscoring its potential as a therapeutic target in cancers that rely on mTOR signaling.

Abstract ID: 4

Title: Organotin(IV) Derivatives Enhance Endocrine Drug Response in Breast Cancer Cells

Authors: Yumna Yousaf, Rumeza Hanif
Affiliation: National University of Sciences and Technology, Islamabad

Background and Objectives:
Approximately 80% of breast cancer cases are hormone receptor-positive and receive endocrine therapy as the first line of treatment. But therapy resistance limits the efficacy of endocrine drugs. Co-targeting breast cancer cells with other chemotherapeutic agents alongside commercially used endocrine drugs has gained attention as a potent remedy for resistance.

Methods:
We aimed to investigate the effect of Organotin (IV) derivatives of 4-(2-thienyl) butyric acid on the survival of breast cancer cells and their potential in combination therapy. The cytotoxicity profile was quantified in established ER+/PR+ breast cancer and TNBC cell lines.Clonogenic Survival and cell migration were assessed in breast cancer cells lines. The DNA fragmentation assay was used to study the effect of selected derivatives on DNA integrity in cancer cells.

Results:
Two of the chosen derivatives inhibited cell proliferation in a dose-dependent manner. These derivatives synergistically inhibited cell survival and migration in combination with fulvestrant, palbociclib and letrozole in breast cancer cells. Induction of apoptosis was confirmed in treated cells through DNA fragmentation.

Conclusion:
Our results showed that Organotin (IV) derivatives of 4-(2-thienyl) butyric acid induce apoptosis and synergistically inhibit cell proliferation, survival and migration in breast cancer cells. These findings make them a potent candidate for combination therapies against endocrine resistant breast cancers.

Abstract ID: 22
Title: Integrative Computational Approach for Exploring the Mechanism of RhoA Inhibition in Aggressive Cancer
Authors: Mishal Dar, Dr Afifa Navid
Affiliation: Forman Christian College, University, Lahore

Background & Objectives:
RhoA, a small GTPase of the Rho family, is a central regulator of actin cytoskeleton dynamics, cell migration, and transcriptional control, making it a key driver of cancer metastasis and invasion. However, its compact, stable surface and lack of conventional binding pockets have rendered it historically "undruggable." This study aimed to identify druggable sites on RhoA and evaluate covalent inhibitor candidates using integrated computational approaches.

Methods:
Computational modelling, covalent docking, and long-timescale molecular dynamics (MD) simulations were employed. Potential binding sites were predicted in silico, followed by docking of Rhosin analogues selected based on reported potency and SAR evidence. MD simulations (100 ns) were performed to assess interaction stability, residue-level contributions, and covalent bond persistence.

Results:
Among the screened analogues, UC-177619 exhibited the strongest performance, with a docking affinity of −5.17 and stable covalent attachment at the noncanonical Cys107 site. MD analysis demonstrated minimal atomic fluctuation across the trajectory. Stabilizing interactions were observed with Asp78, Ala178 (37% occupancy), and Gln180 (34% occupancy), including persistent hydrogen bonding that enhanced ligand retention within the pocket. These cooperative interactions between Cys107 and neighbouring residues supported the structural viability of the covalent complex.

Conclusions:
This study identifies Cys107 as a promising covalent inhibition site on RhoA and demonstrates the feasibility of targeting RhoA through unconventional, multi-residue binding motifs. These findings challenge the long-held assumption of RhoA's "undruggability" and provide a computational foundation for the development of therapeutics against aggressive, treatment-resistant cancers driven by RhoA dysregulation.

Abstract ID: 32

Title: Promoter Methylation–Driven Epigenetic Silencing as a Biomarker in Head and Neck Cancer

Authors: Madiha Kanwal, Ghulam Haider, Abdul Hameed, Asmat Salim, Sitwat Zehra, Tuba Shakil Malick, Saima Saleem

Affiliation: Salim Habib University, Karachi

Background & Objectives:

Head and neck squamous cell carcinoma (HNSCC) is the seventh most common cancer worldwide and ranks second in incidence in Pakistan. The Hippo signaling pathway kinase MST1, a pro-apoptotic protein, regulates cell growth through phosphorylation. Epigenetic silencing of the MST1 gene may lead to cellular dysregulation, potentially contributing to HNSCC progression. This study investigates the association of MST1 gene methylation with HNSCC and its impact on gene expression.

Methods:

A total of 413 subjects (313 HNSCC patients and 100 controls) were analyzed for DNA methylation status of the MST1 gene. DNA methylation analysis was conducted via DNA extraction, bisulfite conversion, methylation-specific PCR, and gel electrophoresis. Gene expression analysis was performed in CAL-27 (cancer) and HGF-1 (normal) cell lines through tissue culturing, RNA extraction, and RT-PCR. Genetic analysis aimed to detect any start codon mutation in MST1 using PCR, restriction fragment length polymorphism, and sequencing.

Results:

MST1 promoter methylation was detected in 86% of HNSCC samples and in both cell lines. Statistical analysis (χ^2 test) revealed a significant association between MST1 promoter methylation and HNSCC (χ^2=34.7, PATA) was identified in MST1.

Conclusions:

The findings suggest that MST1 promoter methylation may contribute to HNSCC by down-regulating MST1 expression, highlighting its potential as a biomarker and therapeutic target for HNSCC. Understanding this epigenetic modification could provide valuable insights for precision oncology approaches in HNSCC.

Abstract ID: 33

Title: Integrated In Silico–In Vitro Discovery of Novel Aromatase Inhibitors for Breast Cancer Therapy

Authors: Hassan Khan, Atif Hussain, Mehwish Kalam, Humaira Zafar
Affiliation: International Center of Chemical and Biological Science, University of Karachi

Background & Objectives:
Breast cancer progression is closely linked to estrogen production, making aromatase a critical therapeutic target. However, current aromatase inhibitors (AIs) often cause resistance and adverse effects. This study aimed to identify new, potentially safer AIs using a combined in silico and in vitro approach.

Methods:
A library of selected compounds was screened through molecular docking, followed by molecular dynamics simulations to assess complex stability. ADME/Tox analysis was performed to evaluate drug-likeness. Top candidates were then tested in vitro using aromatase inhibition assays to determine IC_{50} values. Cytotoxicity was assessed on MCF-7 cells using the MTT assay.

Results:
Docking studies identified several compounds with strong predicted binding affinities and key interactions within the aromatase active site. MD simulations confirmed stable ligand enzyme complexes. ADME/Tox profiling indicated favorable pharmacokinetic properties. In vitro assays showed that nine lead compounds exhibited strong aromatase inhibition, with IC_{50} values comparable to Letrozole, Anastrzole and Exemestane. MTT results indicated selective cytotoxicity toward MCF-7 cells.

Conclusions:
The combined computational and experimental workflow successfully identified promising new aromatase inhibitor candidates. These findings support further structural optimization and in vivo evaluation to develop more effective breast cancer therapies.

Abstract ID: 40
Title: Vanillin Effects on Wnt/β-Catenin Signaling and Proliferation in CAL-27 Oral Squamous Cell Carcinoma
Authors: Maazah Muhammad Ali, Dr Rehan Imad
Affiliation: Ziauddin University, Karachi

Background and Objectives:
Oral squamous cell carcinoma (OSCC) is a major health challenge in Pakistan, with limited therapeutic options for advanced stages as well as high recurrence rates. The Wnt/β-catenin pathway plays a central role in OSCC progression by driving cell proliferation, survival, and metastatic behavior. Vanillin, a naturally occurring phenolic compound commonly classified among bioactive flavonoid-like molecules, has been reported to exhibit anti-proliferative and pro-apoptotic effects in several cancer models. However, its functional impact on the CAL-27 OSCC cell line, particularly through modulation of Wnt/β-catenin signaling, remains insufficiently explored. This gap limits our understanding of whether Vanillin may serve as a potential pathway-targeted therapeutic candidate. Hence, there are two main objectives for this study, namely, 1. To evaluate the anti-proliferative and growth-inhibitory effects of Vanillin on the CAL-27 OSCC cell line. 2. To determine whether Vanillin modulates key components of the Wnt/β-catenin signaling cascade in CAL-27 cells.

Methods:
CAL-27 cells will be exposed to Vanillin at increasing concentrations to assess its dose-dependent cytotoxic and anti-cancer effects. Standard in-vitro assays including MTT will be run for cell viability. Cell cycle analysis will be carried out using autophagy and apoptosis assays. Migration assays will also be used to quantify phenotypic responses. Expression profiling of Wnt/β-catenin pathway regulators will be performed using real-time quantitative PCR (qPCR). These analyses aim to establish whether Vanillin interferes with pathway activity and cell cycle regulation in OSCC.

Results:
Expected results include determining the half-maximal inhibitory concentration (IC50) of Vanillin in CAL-27 cells and identifying whether its anti-proliferative effects correlate with altered expression of Wnt/β-catenin pathway genes. These findings will help clarify whether Vanillin acts as a molecular pathway modulator in OSCC.

Conclusions:
This study will provide an early in-vitro exploration of Vanillin's therapeutic potential in OSCC by investigating both its cytotoxic effects and its influence on Wnt/β-catenin signaling. The results will contribute to the growing body of work on flavonoids as potential pathway-targeted agents in cancer biology and support further pre-clinical and pre-translational work on Vanillin-based interventions for precision oncology.

Abstract ID: 63
Title: Impact of 3D spheroids in regulating chemoresistance of 5-Flourouracil and curcumin in liver cancer
Authors: Rameen Matloob, Muhammad Ikram
Affiliation: COMSATS University Islamabad, Abbottabad Campus

Background and Objectives:
Cancer, a highly dangerous disease, poses a significant threat to millions of people worldwide. Unfortunately, many cases of cancer, particularly in developing countries like Pakistan, are detected at advanced stages, leading to millions of deaths. Moreover, the existing cancer treatments are prohibitively expensive, making them inaccessible to majority of the population in Pakistan. Additionally, the current chemotherapeutic agents face challenges due to the development of resistance. Hence, there is an urgent need to discover a new, innovative, and affordable treatment approach for this deadly ailment. In this study, we successfully established a sophisticated scaffold model using chitosan, acetic acid, polyvinyl alcohol, marine collagen and hydroxypropyl methylcellulose (CAPCH) to simulate the microenvironment and chemoresistance of human hepatoma cells.

Methods:
Solvent casting and lyophilization were used to create CAPCH scaffolds with different hydroxypropyl methylcellulose concentration. Characterization of prepared scaffold was performed using FTIR, XRD, Porosity analysis and Swelling behavior test to confirm its physical characteristics. We employed MTT assay and optical microscopy to assess cell growth, proliferation, and spheroid formation in the composite Scaffold.

Results:
The results revealed that our Scaffold system effectively promoted cell growth and proliferation. Based on these evaluations, we assessed the efficacy of the scaffold system in promoting cancer growth and stimulating chemoresistance phenomenon in HepG2 cell lines. Evaluation of chemoresistance, stemness and cancer progression related genes MDR, MMP, CD-133 and CD44 gens were upregulated and proapoptotic PPR and p53 genes were downregulated by reverse transcriptase PCR (RT-PCR) in 3D CAPCH in vitro hepatoma model compared to 2D culture.

Conclusion:
This study demonstrates that three-dimensional (3D) culture more accurately mimics the tumor microenvironment and enhances chemoresistance compared to conventional models. A composite biopolymeric scaffold supported HepG2 spheroid formation and exhibited increased resistance to 5-fluorouracil and curcumin. Furthermore, this data can offer valuable techniques for developing three-dimensional scaffolds that are functional and biocompatible for culturing various cells.

Abstract ID: 65
Title: New Insights Regarding Potential Diagnostic and Prognostic Biomarkers of Breast Cancer
Authors: Yasir Hameed, Samina Ejaz, and Shufang Liang
Affiliation: The Islamia University of Bahawalpur

Background and Objectives:
Breast cancer (BC) is one of the most prevalent malignant tumors in women worldwide, with a significant morbidity and mortality rate. The present study was launched to investigate the BC-associated hub genes using in-silico approach.

Methods:
GSE10810 and GSE29431 were retrieved from the Gene Expression Omnibus (GEO) database to identify the differentially expressed genes (DEGs). The pathway and functional enrichment analyses of DEGs were performed using DAVID. Protein-Protein interaction (PPI) network was constructed and visualized through STRING and Cytoscape to identify the hub genes. The identified hub genes were further analyzed to document their response in Kaplan-Meier (KM) survival curve analysis using KM plotter. UALCAN was used to validate the differential expression of the hub genes at both mRNA and protein level, and to check their correlation with promoter's methylation status. Finally, the CTD was utilized to construct the gene-drug interaction networks of the identified hub genes.

Results:
In total 449 DEGs were detected including 151 up-regulated and 298 down-regulated genes. The identified DEGs were enriched in various cancer-related biological functions and pathways. The identified six hub genes (CDK1, FN1, AURKA, CCNB2, BIRC5, and TOP2A) were correlated with worse overall survival (OS) and various other features of the BC patients.

Conclusion:
This extensive study helped to document the prominent role of the identified hub genes in stimulating breast tumor growth through the activation of Maturation Promoting Factor (MPF), PI3K/AKT signaling, and an anti-apoptotic factor like BIRC5 that could be targeted for devising effective treatment strategies.

Abstract ID: 10
Title: Targeting Extrachromosomal DNA in Cancer: A Narrative review on Precision Oncology
Authors: Fareeda Brohi, Biruk D.Ayalew, Lareb Asad
Affiliation: Peoples University of Medical And Health Sciences For Women, Nawabshah

Introduction:
The first Identification of Extrachromosomal DNA (ecDNA) occurred during the mid-20th century when scientists spotted double minutes as tiny acentric chromatin bodies in cancer cell metaphase spreads (1). Scientists initially viewed these cytogenetic anomalies as strange yet they contained amplified oncogenes which later established their direct connection to cancer progression (2). The occurrence of ecDNA spans multiple cancer types because it serves as a prevalent pattern in diverse malignancies thus demonstrating its widespread clinical importance.

Methodology:
We comprehensively search the literature using the PubMed, Cochrane, and Scopus databases from inception to December 2024, for the following keywords,The literature search encompassed English-language review articles, case reports, randomized control trials, and cross-sectional studies. Publications from the past ten years were prioritized, but no articles from older eras that receive frequent citations were left out. We examined the tables of references in all subject-to-analysis articles, reviews, and book chapters in order to locate additional papers that might have slipped through the first search. The objective of this narrative review is to synthesize current evidence on the molecular mechanisms underlying ecDNA formation, including chromothripsis, breakage–fusion–bridge cycles, and replication stress, and highlight how these events reform cancer genome architecture. We further discuss the rapid advances in detection methods, ranging from next-generation and single-cell sequencing to CRISPR-based capture techniques and liquid biopsy approaches, which have improved the identification and characterization of ecDNA in clinical samples. Increasing evidence links ecDNA to poor prognosis and therapy failure, underscoring its value as a prognostic biomarker and therapeutic target. Finally, we explore emerging strategies aimed at disrupting ecDNA maintenance and function, which may open new avenues in precision oncology. Overall, ecDNA research is redefining our understanding of cancer evolution and offers promising opportunities for translational applications.

Conclusion:
Extrachromosomal DNA (ecDNA) has emerged as an important driver of tumor heterogeneity, oncogene amplification, and therapeutic resistance, redefining our knowledge of cancer biology. ecDNA is now recognized as a clinically significant biomarker and therapeutic target. Advances in sequencing, imaging, and computational tools have enabled exact ecDNA detection, while preclinical studies highlight its potential as sensitivity for targeted therapies,Major challenges remain in clarifying mechanisms of ecDNA biogenesis, integration, and immune interactions, as well as in translating preclinical findings into clinical practice despite the advances,Future research integrating liquid biopsy, single-cell profiling, and novel therapeutics promises to accelerate the clinical utility of ecDNA-directed strategies

Abstract ID: 53
Title: Implantable Bioengineered Encapsulated Asparaginase Cell Factories as a Safer, less Infectious and Affordable Alternative for Acute Lymphoblastic Leukemia
Authors: Tayba Arab Farooqi
Affiliation: Jinnah Sindh Medical University, Karachi

Background:
Acute lymphoblastic leukemia (ALL) is the most common childhood cancer and remains a major cause of pediatric cancer mortality in low- and middle-income countries (LMICs). While cure rates exceed 85–90% in high-income countries, LMIC outcomes often remain below 40% due to treatment abandonment, limited supportive care, and restricted access to essential agents such as PEG-asparaginase. Salvage therapies for relapsed or refractory disease including CAR-T cell therapy and hematopoietic stem-cell transplantation (HSCT) are available but are prohibitively expensive, deeply immunosuppressive, and rarely accessible in LMIC settings.

Methods:
A narrative review of global ALL treatment strategies, survival disparities, and the pharmacologic role of asparaginase was conducted. Drawing on established encapsulated cell-therapy technologies used in diabetes management, this proposal outlines a conceptual, implantable bioengineered, retrievable cell-factory to secrete PEG-asparaginase or a low-immunogenic equivalent.

Comparative Insights:
Reduced infection risk compared with CAR-T: PEG-asparaginase selectively targets leukemic blasts lacking asparagine synthetase while sparing normal B cells and other lymphocytes with endogenous asparagine synthesis. Unlike CAR-T, it does not cause B-cell aplasia, hypogammaglobulinemia, or IVIG dependence, avoiding profound adaptive immunosuppression and associated infection burden. Reduced infection risk compared with HSCT: HSCT requires central venous catheters, prolonged neutropenia, and repeated inpatient procedures, major drivers of life-threatening nosocomial infections in immunocompromised children. A subcutaneous implant eliminates these requirements and reduces exposure to hospital-acquired infectious risk. Proposed Cell-Factory Design and Safety Framework The system employs human-derived producer cells, minimizing xenogeneic immune reactions and improving long-term compatibility. These cells must be resistant to self-depletion through asparagine synthetase (ASNS) overexpression, and secretion efficiency can be enhanced using intracellular trafficking pathways to prevent accumulation-induced toxicity. Encapsulation would employ biocompatible semi-permeable materials, allowing for nutrient and enzyme diffusion while preventing immune cell penetration. Drug-inducible expression systems, inducible suicide switches, and surgical retrievability from subcutaneous implantation sites, would enable reversibility and patient protection.

Conclusion:
A bioengineered implant using controllable expression for long-term enzyme delivery and retrievability option can avoid CAR-T–related IVIG dependence and HSCT hospitalization/catheter burdens, with minimal infection risk and improved and affordable suitability for LMIC deployment.

Abstract ID: 62

Title: Safety and Efficacy Of Camrelizumab Based Regimens In Triple Negative Breast Cancer : A Systematic Review and Meta Analysis

Authors: Abdelrahman Elhalbawy, Hakim Ullah Wazir, Muhammad Maaz, Muhammad Uzair, Rabia Javed, Kashif Ali, Muhammad Hisham alamin, Nora Iman
Affiliation: Liaqat University Of Medical And Health Sceinces, Jamshoro

Background:
Triple-negative breast cancer (TNBC) is an aggressive subtype of breast cancer associated with limited treatment options and a high risk of recurrence.Studies suggest that the addition of immune checkpoint inhibitors to combination therapies may enhance treatment efficacy. While PD-L1 inhibitors such as atezolizumab, avelumab, and durvalumab have been evaluated in TNBC, camrelizumab, a newer PD-1 inhibitor, has not yet been assessed in this context. Therefore, we conducted this meta-analysis to evaluate safety and efficacy of camrelizumab-based regimens in the management of TNBC.

Methodology:
We conducted a thorough search on PubMed, Cochrane Library, Embase, Scopus and Clinicaltrials.gov till April 2025. After screening, data extraction and Rob quality assessment of RCTs, analysis was performed using RevMan software. Mantel-Haenszel model was used for dichotomous outcomes. The outcomes are presented as Proportions or Standard mean differences (SMD) with 95% confidence intervals (CI), were calculated using a random-effects model. The Higgins I-square statistic was calculated to assess statistical heterogenicity. A p value of <0.05 indicates significance of results.

Results:
A total of ten studies, 9 single arm RCTs And 1 Observational Study involving 598 patients with triple-negative breast cancer (TNBC).The pooled prevalence of complete response (CR) was (0.21, 95% CI: 0.03–0.39), disease control rate (DCR) (0.76, 95% CI: 0.62–0.90), objective response rate (ORR) (0.62, 95% CI: 0.42–0.81), partial response (PR) (0.52, 95% CI: 0.27–0.77), stable disease (SD) (0.24, 95% CI: 0.12–0.37) and progressive disease (PD) (0.13, 95% CI: 0.03–0.23. The mean progression-free survival (PFS) was 7.81 months.Treatment-related adverse events (TRAEs) of any grade (1.00, 95% CI: 0.99–1.00).

Conclusion:
Camrelizumab-based regimens suggest a promising therapeutic option for patients with triple-negative breast cancer (TNBC), demonstrating encouraging efficacy outcomes and a manageable safety profile. These findings support the continued exploration of camrelizumab in the treatment of TNBC. Further research is needed to refine therapy and enhance outcomes in TNBC.

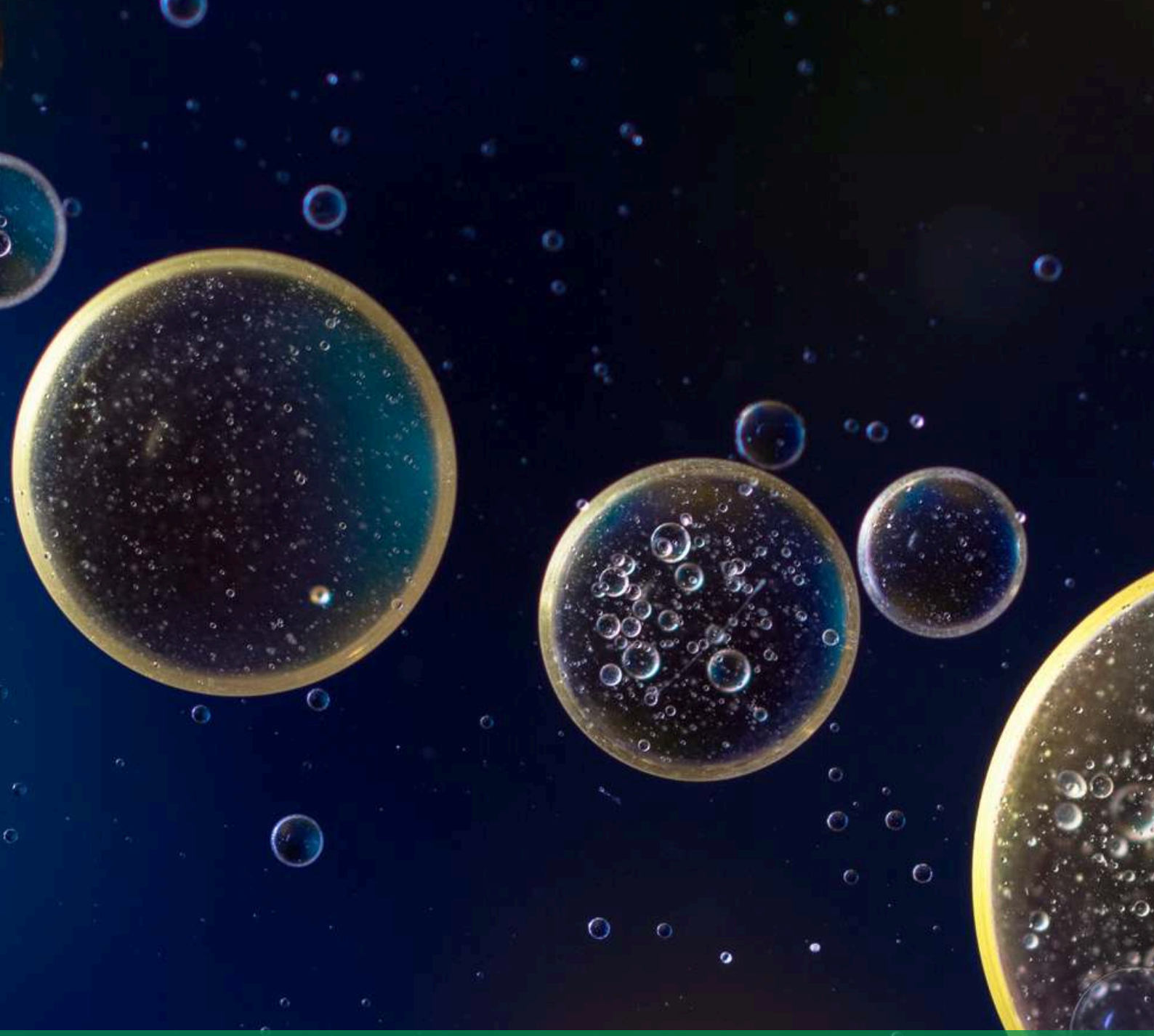
Abstract ID: 66
Title: Biomarkers of Tumor-Associated Heterogeneity and Clinical Outcomes of Breast Cancer Therapy
Authors: Duaa e Fathah, Samina Ejaz, Romena Qazi
Affiliation: The Islamia University of Bahawalpur

Background & Objectives:
Breast cancer (BC) is a highly heterogeneous disease and thus leads to variations in patient-to-patient clinical features and clinical outcomes of treatment. Therefore, developing treatment strategies based on heterogeneity-specific features of BC patients is an important requirement of personalized treatment.

Methods:
We systematically explored the literature databases like SciVerse Scopus ® (Elsevier Properties S. A, USA), Web of Science® (Thomson Reuters, USA), and PubMed. Only 56 studies were filtered out of the total published articles available on these search engines which satisfied the inclusion criteria of the study. The published literature reported various macromolecules that are altered in tumor cells and serve as contributing factors in the progression of cancer. Moreover, the studies highlighted the fact that such molecules can be employed as diagnostic and prognostic biomarkers for early detection of BC.

Results:
The literature suggested that many biomarkers are exclusively useful for diagnosing BC more accurately. Due to their ability to elicit differential behavior in BC patients of diverse clinicopathological features, these biomarkers can be targeted during personalized treatment strategies, and may help in efficient monitoring of chemotherapy progress to improve patients' survival rates.

Conclusions:
This systematic review is an attempt to enhance the understanding and synthesize more information based on patients' reproductive age, menopausal status, family history, and gender. Various molecular markers are highlighted that can help in diagnosis, prognosis and monitoring response of patients to therapy. More research is needed to better understand the heterogeneous features of breast tumors and their variable response to chemotherapy.



THEME 5: NANOMEDICINE, EXTRACELLULAR VESICLES & ADVANCED DRUG DELIVERY

Abstract ID: 25

Title: Isolation of CD34+ Hematopoietic Stem Cells using antibody-functionalized Iron Oxide Nanoparticles

Authors: Aiman Aslam, Rida e Maria Qazi, Irfan Hussain, Afsar Ali Mian, Fawad Ur Rehman
Affiliation: The Aga Khan University, Karachi

Background & Objectives:
Magnetic-activated cell sorting (MACS) allows the sorting of targeted cells by labelling them with iron oxide nanoparticles (IONPs). In Pakistan, researchers are dependent on importing costly MACS kits with limited shelf life for cell isolation. The import process is beset with long shipment delays. This strains research budgets and delays experimental workflow. Moreover, there is a lack of research exploring IONP-based isolation strategies in Pakistan.
Objectives: (1) To synthesize IONPs (2) To modify IONPs with Polyethylene glycol (PEG) (3) To functionalize IONPs surface with anti-CD34 antibodies (4) To investigate cell isolation efficiency of antiCD34-functionalized IONPs

Methods:
IONPs were synthesized via co-precipitation method, coated with PEG and functionalized with anti-CD34 antibody. Characterization was performed using DLS, TEM, and FTIR. Optimum experimental conditions were optimized using Kasumi-1 cells. IONPs-induced ROS production was assessed via DCFH-DA assay. Anti-CD34 antibody conjugation was performed via BCA protein estimation kit. Isolation efficiency of antiCD34-functionalized IONPs was investigated in a pure culture of HSCs as well as in a heterogeneous cell culture.

Results:
IONPs were successfully synthesized and validated using DLS, TEM, and FTIR. Surface modification was done with PEG, and confirmed via FTIR. IONPs were conjugated with anti-CD34 antibody, and effective conjugation was confirmed using a BCA protein estimation assay. Optimization of experimental conditions using Kasumi-1 cells determined 1 mg as the optimum IONP concentration for cell isolation, 45 minutes as the optimum incubation time, and 40 minutes as the optimum magnetic isolation time. DCFH-DA assay revealed that PEG coating reduced ROS generation in the cells, making the IONPs biocompatible. MACS sorting showed higher isolation efficiency of CD34+ cells by antiCD34-functionalized IONPs as compared to non-functionalized IONPs in pure HSCs culture. AntiCD34-functionalized IONPs also showed 44.4% of relative cell isolation efficiency compared to a commercial MACS kit.

Conclusion:
AntiCD34-functionalized IONPs have the potential to serve as a cost-effective alternative to commercial MACS kit with further improvements in isolation efficiency. In future, nanoparticles can be functionalized with different antibodies and applied to isolate other important cell types.

Abstract ID: 73

Title: Comparative Evaluation of Non-Viral Nanocarriers for siRNA and mRNA Delivery in Hematopoietic and Stem Cell Models

Authors: Shariqa Khawaja, Rida e Maria Qazi, Fawad Ur Rehman, Afsar Mian
Affiliation: The Aga Khan University, Karachi

Background:
Efficient and safe delivery of RNA-based therapeutics remains a central challenge for gene modulation and CRISPR-based strategies, particularly in hematopoietic stem cells (HSCs). Non-viral nanocarriers represent a versatile alternative to viral vectors; however, their performance across different stem and hematopoietic cell types requires systematic evaluation.

Methods:
A head-to-head analysis of four non-viral delivery platforms lipid nanoparticles (LNPs), exosomes, iron oxide nanoparticles (IONPs), and TiO_2GO was conducted using fluorescently labeled siRNA (siRNA-Cy5) and mRNA encoding enhanced green fluorescent protein (eGFP) as model cargos. Nanocarriers were physicochemically characterized by dynamic light scattering, including assessment of Z-average hydrodynamic diameter before and after RNA loading. Cellular uptake, delivery efficiency, and functional expression were evaluated in K562 cells, mesenchymal stem cells (MSCs) and hematopoitic stem cells (HSCs).

Results:
All nanocarrier platforms exhibited nanoscale size distributions, with observable cargo-dependent increases in hydrodynamic diameter following RNA loading. Delivery efficiency and cellular uptake varied across cell types and carrier systems. Functional delivery was confirmed through siRNA-Cy5 internalization and mRNA-mediated eGFP expression in all tested models, revealing platform and cell type dependent differences in uptake and expression profiles. Cytocompatibility was assessed using MTT assays, which demonstrated minimal cytotoxicity and preserved cell viability at functionally relevant doses across all nanocarriers.

Conclusion:
This comparative study highlights the diverse delivery behaviors of non-viral nanocarriers in hematopoietic and stem cell systems. The findings provide an experimental framework to inform the rational selection and optimization of RNA delivery platforms for gene modulation and CRISPR-based therapeutic applications.

Abstract ID: 84

Title: Nanoscale Lipid-Drug Conjugates: An Alternative and Efficient Approach for Drug Delivery

Authors: Mujeeb-ur-Rehman, Franceline Reynaud, Nicolas Tsapis, Saadat Hussain, Aqsa Arif, Rudaba Saleem, Elias Fattal

Affiliation: International Center for Chemical and Biological Sciences, University of Karachi

Background:

In an attempt to tune drug release and subsequent pharmacokinetics once administered intravenously, we have synthesized three lipid-drug conjugates (LDCs) of dexamethasone (DXM) each possessing a different lipid-drug chemical linkage: namely ester, carbamate and carbonate.

Methods:

These LDCs were thoroughly characterized before being turned into nanoscale particles by an emulsion-evaporation process using DSPE-PEG_{2000}-OMe as the only excipient. Spherical nanoparticles (NPs) of about 140-170 nm, with a negative zeta potential, were obtained for each LDC and exhibited good stability upon storage at 4 °C for 45 days with no recrystallization of LDCs observed.

Results:

LDC encapsulation efficacy was above 95% for the three LDCs, leading to an LDC loading of about 90% and an equivalent DXM loading above 50%. Although the ester and carbonate NPs did not exhibit any toxicity up to an equivalent DXM concentration of 100 µg/mL, the carbamate LDC NPs appeared very toxic towards Raw264.7 macrophages and were discarded. Both ester and carbonate LDC NPs were shown to exert anti-inflammatory activity on LPS-activated macrophages. DXM release from LDC NPs in murine plasma was faster from ester than from carbonate NPs. Finally, pharmacokinetics and biodistribution were conducted, showing a lower exposure to DXM from carbonate LDC NPs than from ester LDC NPs, correlated with the slower DXM release from carbonate LDC NPs**.**

Conclusion:

These results outline the need for extended studies to find the best prodrug system for extended drug release. The approach used for LDCs of DXM has been further extended to some other drugs including NSAIDs delivery, and results obtained indicated that nanoscale LDCs could be potentially efficient approach for drug delivery purposes.

Abstract ID: 72
Title: Adipose Mesenchymal Stem Cells Derived Extracellular Vesicles Attenuate Carbon Tetrachloride induced Liver Fibrosis in Rats
Authors: Somia Shehzadi and Ahmad Bilal Waqar
Affiliation: The University of Lahore

Background:
Liver fibrosis is a progressive and potentially deadly, chronic liver disease complication, such as viral hepatitis, alcoholic liver disease, and non-alcoholic steatohepatitis (NASH). Chronic liver diseases are also attributed to the high presence of hepatitis B and C, environmental and lifestyle factors in Pakistan. Carbon tetrachloride (CCl 4) is a proven model of liver fibrosis induction in animals with the closest resemblance to the fibrotic liver disease in humans. The extracellular vesicles of adipose mesenchymal stem cells-AD-MSC-EVs have had regenerative medicine potential owing to their anti-inflammatory, anti-fibrotic and pro-regenerative capabilities.

Objectives:
The main purpose of conducting the study was to assess the therapeutic effects of AD-MSC-EVs on the CCl 4-induced liver fibrosis in rats. To achieve specific objectives were: Isolation and characterization of AD-MSC-EVs on the basis of size, morphology and surface markers. Liver fibrosis induction in rat with CCl 4 and evaluation of fibrosis process through biochemical and histopathological analysis. Comparison of the effect of AD-MSC-EVs on the enhancement of liver function, fibrosis, and inflammation and oxidative stress. Investigations into the mechanisms which underlie it, especially the TGF-B/Smad pathway. Safety and feasibility of the AD-MSC-EVs as a potential therapeutic intervention of liver fibrosis.

Methods:
Male Sprague-Dawley rats were separated into three groups, and these were control, CCl 4-induced fibrosis, and CCl 4 + AD-MSC-EVs. The isolation of AD-MSC-EVs was performed through ultracentrifugation and identification was done through transmission electron microscopy (TEM) and Western blot. The liver fucntion was determined by means of serum biochemistry (ALT, AST, albumin, bilirubin), whereas fibrosis was measured through histopathology (H and E, Masson trichrome, Sirius red staining). Molecular analysis was done on fibrotic (alpha-SMA, Collagen I, TGF- b), inflammatory (TNF- alpha, IL- 6, il-1b), and oxidative stress markers (MDA, SOD, GSH).

Results:
There was a significant improvement in liver functions in the AD-MSC-EVs groups observed by the decrease in serum ALT and AST ($p < 0.01$). Histopathological examination revealed a reduction in collagen deposition and the severity of fibrosis. Molecular diagnosis showed a decrease in fibrotic and inflammatory markers, and in oxidative stress. Mechanistically, the TGF-B/Smad signaling pathway was regulated by AD-MSC-EVs, which stopped the activation of hepatic stellate cells and the formation of ECMs.

Conclusion:
The AD-MSC-EVs are efficient in CCl 4 inhibited liver fibrosis diseases of rats by enhancing liver functioning, mitigating fibrosis and suppressing inflammation and oxidative stress. These results indicate that AD-MSC-EVs have a great treatment potential in liver fibrosis and require further clinical trials.

Abstract ID: 14
Title: Electrospun Nanofiber Hydrogel Patch with Neem, Moringa, and Clove Oil for Targeted Antifungal Therapy of Nail Infection
Authors: Qurat-ul-Ain Arif, Sumaiya Sajjad, Umar Ali
Affiliation: Federal Urdu University of Arts,Science and Technology, Karachi

Background:
Onychomycosis is a chronic fungal infection caused mainly by Trichophyton rubrum, T. mentagrophytes, and Candida species. Current therapies—systemic antifungals and topical lacquers—suffer from major limitations, including hepatotoxicity, drug interactions, long treatment duration, frequent recurrence, and extremely poor nail penetration. Herbal antifungals such as Neem (Azadirachta indica), Moringa (Moringa oleifera), and Clove Oil (Syzygium aromaticum) possess strong antifungal, anti-inflammatory, and biofilm-disrupting properties, but their therapeutic potential is restricted by instability, poor solubility, and limited permeation through the dense keratinized nail plate. This study aims to develop a dual-phase electrospun nanofiber–hydrogel patch designed to improve nail penetration, sustain herbal drug release, enhance antifungal efficacy, and ensure biocompatibility for localized onychomycosis therapy.

Methods:
Nanofibers were fabricated using PVA/Chitosan and PCL/Chitosan blends incorporating neem and moringa extracts, while clove oil was microencapsulated to improve stability. Electrospinning parameters (15–18 kV, 0.5 mL/h, 15 cm distance) were optimized to obtain uniform fibers. An Aloe vera–genipin hydrogel layer (~0.5 mm) was applied over the nanofibers to create a composite patch. Characterization included SEM, FTIR, tensile strength, swelling index, and contact-angle analysis. In-vitro release and ex-vivo porcine hoof penetration were performed using Franz diffusion cells. Antifungal activity was tested against T. rubrum, T. mentagrophytes, and C. albicans via MIC, disk diffusion, time-kill assays, and biofilm-reduction studies. Cytotoxicity was assessed on HaCaT keratinocytes using the MTT assay.

Results:
The patch produced uniform nanofibers (320–480 nm) with ~75% porosity and tensile strength of ~2.7 MPa. Swelling analysis showed 130% water uptake, supporting sustained release. In-vitro release demonstrated gradual 48-hour delivery (neem 65%, moringa 60%, clove oil 55%). Strong antifungal activity was observed with MICs of neem 8 µg/mL, moringa 10 µg/mL, and clove oil 5 µg/mL, achieving 50–70% biofilm reduction. Ex-vivo nail studies showed drug flux of 0.3–0.5 µg/cm^2/h and retention of ~20 µg at 72 h. HaCaT cell viability remained >85%, confirming biocompatibility.

Conclusion:
The dual-phase nanofiber–hydrogel patch demonstrates sustained release, enhanced nail penetration, potent antifungal and anti-biofilm activity, and excellent safety. This plant-based nanotechnology platform presents a promising and affordable therapeutic alternative for targeted onychomycosis treatment, with strong potential for future clinical translation.

Abstract ID: 15
Title: Inhalable Nafithromycin Nanomist: A Targeted Approach to Community-Acquired Bacterial Pneumonia Treatment
Authors: Umar Ali, Sumaiya Sajjad, Dr. Farah Owais, Quratulain Arif
Affiliation: Federal Urdu University of Arts,Science and Technology, Karachi

Background & Objectives:
Community-acquired bacterial pneumonia (CABP) continues to be a serious worldwide health concern, especially for the elderly and immunocompromised. Despite improvements in immunizations and treatments, mortality is still high, particularly in the first 48 hours of hospitalization, with rates as high as 22.5% over the past ten years. The necessity for tailored medicines is highlighted by growing β-lactam resistance and questions about the safety of fluoroquinolones. The systemic transport of nafithromycin, a novel lactone-ketolide, limits its oral efficacy. In order to improve CABP treatment, this study sought to create an inhalable nafithromycin nanomist using the CAN-BD technique to provide high localized lung concentrations.

Methodology:
In order to create dry, nano-structured respirable particles, nafithromycin can be prepared using inhalation-grade excipients and processed using the CAN-BD method. Particle size and shape (laser diffraction, scanning electron microscopy), aerodynamic behavior (Mass Median Aerodynamic Diameter, fine particle fraction utilizing a cascade impactor), moisture content, and chemical stability can be assessed for the powders. Human airway epithelial cells can be used in in-vitro investigations to measure cytotoxicity and dissolved in simulated lung fluid. Initial in vivo analyses evaluate tolerability to verify pulmonary safety and lung pharmacokinetics to ascertain medication absorption and retention.

Results:
In order to facilitate effective deep lung deposition, the CAN-BD processed formulation generated nano-structured particles with an MMAD < 5 μm and a high fine particle fraction. The powders showed consistent shape, little moisture content, and chemical integrity. In simulated lung fluid, rapid and total disintegration was seen, suggesting quick local drug availability. Testing for cytotoxicity revealed acceptable biocompatibility and no discernible decrease in cell viability. Tolerability studies revealed no significant pulmonary inflammation or toxicity, and preliminary in vivo results demonstrated improved lung retention with decreased systemic exposure.

Conclusion:
With its quick lung-specific drug delivery, enhanced local bioavailability, and decreased systemic hazards, the inhalable nafithromycin nanomist shows great promise as a targeted pulmonary therapy for CABP. These results encourage scale-up for clinical translation, long-term stability assessment, and additional preclinical research. Drug-resistant respiratory infections may be treated with this platform in an efficient and patient-friendly manner.

Abstract ID: 28
Title: Multifunctional Protein-like Polymers for the Treatment of Tauopathies
Authors: Sarah Anis Ur Rahman, Adeena Fateh, Manal Raza, Kainat Zehra, Elham Rustam
Affiliation: Dow University of Health Sciences, Karachi

Background and Objectives:
One of the most hazardous species linked to the development and course of Alzheimer's disease and associated tauopathies is tau oligomers. Despite their crucial function, current therapeutic functions find it difficult to specifically target and neutralize these early pathogenic forms. The goal of this research is to create multifunctional protein-like polymers that can recognize, bind to, and neutralize toxic tau oligomers; prevent them from aggregating and spreading, and promote their natural breakdown via cellular clearance processes.

Methods:
Tau-selective recognition polymers, aggregation-inhibiting elements and functional groups that promote intracellular breakdown pathways were all included into a modular nanocarrier PLP platform. Recombinant tau oligomers and neuronal cell models were used for in-vitro assessment. Particle characterization, tau-binding tests, aggregation-inhibition investigations, immunofluorescence imaging, and evaluation of cellular uptake and clearance activities were important techniques.

Results:
PLP significantly prevented further aggregation in vitro and demonstrated high selective binding to tau oligomers. PLP therapy promoted the activation of endogenous degradation processes and decreased the measurable levels of intracellular and extracellular oligomeric tau in neuronal cell models. When taken as a whole, the platform showed promise for interfering with several stages of tau disease.

Conclusions:
Toxic tau species can be targeted with a promising nanomedicine-based strategy thanks to the multifunctional PLP system. These particles offer a promising approach for therapeutic intervention in Alzheimer's disease and other tauopathies by combining selective recognition, aggregation inhibition, and accelerated degradation. Their translational potential will be evaluated by additional pre-clinical assessment.

Abstract ID: 31
Title: Engineered Exosomes as Targeted Nanocarriers for Precision Drug Delivery in Cancer Therapy
Authors: Rukhsar Shah
Affiliation: Dow University of Health Sciences, Karachi

Background & Objectives:
Exosomes are naturally occurring extracellular vesicles with high biocompatibility and an intrinsic ability to transport functional biomolecules. Their unique biological properties make them promising candidates for precision drug delivery in cancer therapy. The objective of this planned study is to engineer exosomes to enhance drug loading efficiency and improve tumor-specific targeting, with the goal of developing a more effective and less toxic therapeutic platform.

Methods:
The study will isolate exosomes from mesenchymal stem cells using differential ultracentrifugation. Characterization will include nanoparticle tracking analysis, transmission electron microscopy, and western blotting to confirm vesicle identity. Exosome engineering will be performed through surface functionalization using tumor-targeting peptides. A model chemotherapeutic agent (e.g., doxorubicin) will be loaded via electroporation. In vitro experiments will evaluate cellular uptake, cytotoxicity, and apoptosis induction in selected cancer cell lines. Pending approval, in vivo biodistribution and targeting efficiency will be assessed using an appropriate preclinical model.

Results:
Based on existing evidence, it is anticipated that engineered exosomes will demonstrate improved drug loading efficiency and enhanced targeting toward cancer cells compared to unmodified exosomes. The study is expected to show increased therapeutic response in vitro and improved accumulation at tumor sites in vivo. These projected outcomes aim to support the feasibility of developing engineered exosomes as a next-generation nanocarrier for targeted cancer drug delivery.

Conclusions:
This study aims to advance the development of engineered exosomes as a precision drug delivery system for cancer therapy. By optimizing drug loading and tumor-targeting capabilities, the project seeks to contribute to the growing field of nanomedicine and provide a foundation for future preclinical and clinical applications. The findings may inform strategies for safer and more effective cancer therapeutics.

Abstract ID: 39

Title: Developing fusidic acid-loaded niosomes may overcome multidrug resistance caused by mutations, biofilms, and poor drug solubility, enabling targeted antibacterial therapy.

Authors: Seema Inayat, Hafiza Arsala, Saadat Hussain, Rukesh Maharjan, Shagufta Noreen, Mujeeb-ur-Rehman

Affiliation: Jinnah University For Women, Karachi

Background & Objectives:

Multidrug resistance remains a major barrier to successful antimicrobial therapy, often arising from genetic mutations, protective biofilm formation, and reduced drug bioavailability caused by poor solubility. Fusidic acid is an effective antibiotic against Staphylococcus aureus; however, its limited water solubility restricts its therapeutic potential. Nanotechnology-based drug delivery systems offer promising strategies to enhance the solubility, stability, and targeted delivery of conventional antibiotics. In this study, nanoscale fusidic acid-loaded niosomes were developed to improve antibacterial efficacy and address resistance mechanisms.

Methods:

Fusidic acid-loaded niosomes were initially prepared using the ethanol injection method, followed by characterization through dynamic light scattering (DLS) to determine particle size distribution. Surface morphology was examined using atomic force microscopy (AFM) and scanning electron microscopy (SEM). Entrapment efficiency was quantified using ultra-performance liquid chromatography (UPLC). Antibacterial activity was evaluated through minimum inhibitory concentration (MIC) assays, and the ability of the formulations to eliminate biofilms was also examined.

Results:

Successful formulation of fusidic acid niosomes was achieved using both ethanol injection and emulsion methods. The optimized niosomes exhibited particle sizes ranging from 116.4 to 274.2 nm, with SEM confirming their spherical morphology. Entrapment efficiency reached up to 94%, demonstrating high drug loading capacity. MIC testing against multiple S. aureus strains, including NCTC 13277, NCTC 13143, ATCC 700699, and ATCC 6538, revealed that Tween-20 and Span-40 based niosomes displayed strong antibacterial activity comparable to standard fusidic acid (0.04 µg/mL), with MIC values between 0.039 and 0.078 µg/mL. AFM analysis further showed that Tween-20 based niosomes induced notable alterations in the structural and mechanical properties of S. aureus biofilms.

Conclusions:

These findings highlight the potential of fusidic acid-loaded niosomes as an effective nanocarrier system for enhancing antibacterial activity and overcoming multidrug resistance.

Abstract ID: 80
Title: Nanosystem based approaches for Systemic Delivery of Nucleic Acid: A Preclinical Study
Authors: Rida-e-Maria Qazi, Seema Inayat, Shariqa Khawaja, Afsar Ali Mian, Fawad Ur Rehman
Affiliation: The Aga Khan University, Karachi

Background:
Nanoparticle-based delivery systems have significantly advanced the therapeutic potential of nucleic acids (such as siRNA, mRNA and CRISPR-Cas9 system) by improving stability, biodistribution, and cellular uptake. Among these systems, lipid nanoparticles (LNPs) represent the most clinically advanced platform for systemic nucleic acid delivery. Fluorescent labeling of siRNA, such as with cyanine 5 (Cy5), and reporter gene expressing mRNA, such as Luciferase mRNA, enables noninvasive tracking of nanoparticle-mediated delivery and tissue distribution in vivo. Aims: The aim of this study was to evaluate the in vivo delivery efficiency, biodistribution, cellular uptake, and tolerability of Cy5-labeled siRNA/Luciferase mRNA delivered using lipid nanoparticles, following systemic administration in mice.

Methods:
Cy5-labeled siRNA was encapsulated into nanoparticles, including ionizable/cationic lipid-based lipid nanoparticles, using a thin film hydration method. The resulting formulations were characterized for particle size, polydispersity index, and zeta potential. Mice were administered Cy5-siRNA/Luciferase mRNA loaded nanoparticles via intravenous injection. In vivo biodistribution was assessed at multiple time points using whole-body fluorescence imaging. Ex vivo fluorescence imaging of major organs and quantitative fluorescence analysis of peripheral blood were conducted to evaluate tissue accumulation and cellular uptake. Tolerability was assessed by monitoring clinical signs and gross organ morphology.

Results:
Nanoparticles, including lipid nanoparticles, exhibited uniform nanoscale size, and low polydispersity. In vivo imaging revealed strong Cy5 fluorescence and bioluminescence of Luc expression in the animal body while ex vivo organ imaging confirmed these findings and demonstrated sustained hepatic retention. Quantitative fluorescence analysis of blood showed maximum intensity of cationic LNP delivered siRNA at 4 hours while ionizable lipids showed maximum fluorescence at 8 hours. No significant changes in behavior and organ morphology was observed, indicating favorable tolerability of the nanoparticle formulations.

Conclusion:
This study demonstrates that nanoparticle-based systems, particularly lipid nanoparticles, enable efficient and well-tolerated systemic delivery of nucleic acids in mice. The use of fluorescently labelled siRNA and reporter gene expressing mRNA provides a robust approach for evaluating biodistribution and cellular uptake, supporting the continued optimization of lipid nanoparticle–mediated nucleic acids delivery for preclinical applications.

Abstract ID: 13

Title: Nanoparticle-Mediated siRNA Delivery Targeting EGFRvIII in Glioblastoma: Current Strategies, Therapeutic Advances, and Research Evolution — A Scoping Review

Authors: Ramsha Haider, Daniya Jawed, Safa Irfan Shah, Nashmiya Khan, Zoha Iftikhar, Shifa Imran, Wajeeha Siddiqui
Affiliation: Karachi Medical and Dental College

Background:
Glioblastoma (GBM) remains one of the most aggressive primary brain tumors, driven in part by aberrant activation of the epidermal growth factor receptor (EGFR) pathway, including the tumor-specific EGFRvIII mutation. Therapeutically targeting these oncogenic drivers is challenged by the blood–brain barrier (BBB), rapid nuclease degradation of RNA molecules, and heterogeneous tumor biology. Nanoparticle-mediated delivery of small interfering RNA (siRNA) has emerged as a promising strategy to overcome these barriers and achieve tumor-selective gene silencing.

Objective:
This scoping review maps existing evidence on nanoparticle-based siRNA delivery targeting EGFR/EGFRvIII in GBM, highlighting delivery strategies, targeting mechanisms, therapeutic outcomes, and the chronological evolution of research innovations from 2008 to 2025.

Methods:
Following PRISMA-ScR guidelines, we conducted a comprehensive search of PubMed, Scopus, Web of Science, and Google Scholar for studies published between 2008 and 2025. Eligible studies included preclinical in vitro or in vivo investigations utilizing nanoparticles as siRNA delivery systems targeting EGFR/EGFRvIII or related oncogenic pathways in GBM. Data were charted for nanoparticle type, targeting ligands, delivery mechanisms, therapeutic outcomes, and technological innovations, and synthesized thematically.

Results:
Across 2008–2025, 11 key studies demonstrated major progress in siRNA nanomedicine for GBM. Lipid-based, polymeric, gold, iron-oxide, micellar, and virus-mimicking nanoparticles were engineered to enhance siRNA stability, BBB penetration, and tumor-selective uptake. Strategies such as peptide conjugation (iRGD, Angiopep-2, T7), transferrin-receptor targeting, CD44/RGD functionalization, and bioreducible polymer chemistry facilitated efficient cellular internalization and endosomal escape. These platforms achieved robust EGFR/EGFRvIII silencing, reduced tumor proliferation, induction of apoptosis, inhibition of cell migration, radiosensitization, chemosensitization, and prolonged survival in preclinical models. Dual-delivery systems—such as siRNA with paclitaxel, multiple siRNA payloads, or radiation-sensitizing constructs—amplified therapeutic efficacy while maintaining favorable safety profiles. The field has evolved from early proof-of-concept nanoparticles (2008–2014) to multifunctional, clinically oriented, BBB-penetrating platforms (2017–2025), reflecting increasing sophistication and translational potential.

Conclusion:
Current evidence demonstrates that nanoparticle-mediated siRNA delivery targeting EGFR/EGFRvIII is a rapidly advancing therapeutic strategy with strong preclinical efficacy and growing biological precision. Continued optimization of BBB penetration, tumor-selective targeting, and combinatorial therapy platforms is expected to accelerate translation toward clinical application in GBM.

Abstract ID: 18

Title: MICRONEEDLE: Advancing Drug Delivery Through Nano-Integrated Microneedle Technologies

Authors: Islam Mehrban, Cynthia Yousuf, Dua Hussain Sheikh, Ameemah Aslam Hisami, Dr. Farah Owais

Affiliation: Federal Urdu University of Arts Science and Technology, Karachi

Background:

Microneedle are groundbreaking drug delivery system innovation developed to overcome the functional limitations oral and conventional drug delivery systems. This is usually done by creating microscopic pathways through stratum corneum which facilitates the drug d delivery of therapeutic agents into the deeper layers of skin without pain and discomfort. Awareness' among healthcare students remains uneven in a survey of 500 healthcare students, 37.4% were reported to know about microneedle, while 62.6% students were unaware, and among them 39.9% students believed that they could potentially replace conventional transdermal patches for vaccine delivery. Practical adoption in low-resource settings is limited despite growing research due to manufacturing challenges, cost and regulatory barriers. This study evaluates the availability of microneedle for medicine delivery focusing infection control, self-administration, vaccination, and disease management.

Methods:

Traditional administration is limited by poor absorption, enzyme degradation, systemic side effects and patient non-compliance dealing from trypanophobia and discomfort. MNs overcome these challenges by creating microscopic channels through the stratum corneum, enabling the delivery of therapeutic agents into viable layers of skin without stimulating pain receptors or damaging blood vessels. This analysis describes the various applications of the microneedle in cosmetics, enhancing hair regrowth by stimulating follicular repair pathways and increasing the penetration of topical agents and improves drug bioavailability enhances compliance, and expands the scope of drug delivery.

Results:

Microneedle are seen to improve drug absorption and reduced systemic side effects across most studies. Dissolving and hydrogel designs demonstrates the highest safety profiles, whereas hollow microneedle offered precise dosing capabilities. Patient acceptance is higher in well developed countries as compared to underdeveloped due to minimal pain and ease of administration. Major limitations in underdeveloped countries are high cost of biodegradable materials, challenges in large-scale manufacturing, research gap. For chronic diseases such as diabetes, older patients frequently experience fear, stress, and psychological burden associated with repeated insulin injections, which negatively affects adherence. Microneedle insulin patches provide a minimally invasive, low-anxiety drug-delivery method that patients can easily self-apply, enabling better glycemic control and improved treatment satisfaction.

Conclusions:

Microneedle offers a compromising, patient friendly platforms for expanding healthcare access, particularly for vaccinations and immunization programs. However, they are not yet positioned to fully replace traditional transdermal system without improvements in challenges and regulatory framework. In developing countries future research should prioritize low cost-production to achieve wider use.

Abstract ID: 19

Title: Mesenchymal Stem Cell Derived Extracellular Vesicle Therapy for Myocardial Infarction: Mechanisms, Therapeutic Advances, and Research Progression - A Scoping Review

Authors: Safa Irfan Shah, Ramsha Haider, Nashmiya Khan, Zoha Iftikhar, Daniya Jawed, Shifa Imran, Wajeeha Siddiqui
Affiliation: Karachi Medical and Dental College

Background:
Myocardial infarction (MI) remains one of the leading causes of morbidity and mortality worldwide, characterized by irreversible cardiomyocyte loss, inflammation, fibrosis, and progressive ventricular remodeling. Conventional therapies only partially restore cardiac function and do not directly regenerate damaged myocardium. Mesenchymal stem cell derived extracellular vesicles (MSC-EVs) have emerged as a promising cell free regenerative therapy due to their ability to deliver bioactive miRNAs, proteins, and lipids that drive angiogenesis, suppress apoptosis, modulate immunity, and attenuate fibrosis. Enhancements such as MSC preconditioning, genetic engineering, and biomaterial aided delivery further augment therapeutic potency.

Objective:
This scoping review maps current evidence on MSC derived extracellular vesicle therapy in MI, highlighting molecular mechanisms, preclinical therapeutic outcomes, delivery innovations, and the chronological evolution of research from early foundational studies to advanced engineered EV platforms.

Methods:
Following PRISMA-ScR standards, evidence summarized in the uploaded synthesis document was reviewed, encompassing preclinical in vivo rodent MI models, mechanistic in vitro studies, and early clinical reports. Data were charted according to EV source, molecular cargo, delivery strategies, therapeutic enhancements, and cardiac outcomes. A temporal mapping from 2013 to 2025 was constructed to illustrate the research evolution.

Results:
From 2013–2025, MSC-EV research demonstrated significant advancements in understanding and optimizing EV-mediated cardiac repair. Mechanistic studies revealed four dominant therapeutic pathways: (1) Pro-angiogenic activity, improving perfusion and reducing infarct size (Bian et al., 2013; Xuan et al., 2022); (2) Anti-apoptotic signaling via miRNAs such as miR-210 and miR-133a-3p (Cheng et al., 2020; Zhu et al., 2021); (3) Immunomodulation, especially macrophage M2 polarization (Zhao et al., 2019; Deng et al., 2019; Ning et al., 2023); and (4) Anti-fibrotic remodeling, limiting scar formation and improving cardiac structure (Wang et al., 2021; Chen et al., 2025; Gan et al., 2025). Delivery and enhancement strategies including atorvastatin or empagliflozin preconditioning, HIF-1α overexpression, sodium alginate hydrogels, and combined stem cell + EV therapy consistently improved therapeutic outcomes. These interventions yielded reduced infarct size, improved ejection fraction, enhanced cardiomyocyte survival, and more favorable ventricular remodeling across preclinical models. A 2025 clinical case report suggests early real world feasibility.

Conclusion:
MSC-derived extracellular vesicles represent a rapidly advancing, biologically potent, and clinically promising therapeutic avenue for myocardial infarction. Their multifaceted ability to drive angiogenesis, suppress apoptosis, modulate inflammation, and reduce fibrosis emphasised on their regenerative potential.

Abstract ID: 23

Title: Inhaled Nanomedicines for Lung Cancer and Pulmonary Infections (2022–2025): A Systematic Review of Clinical Translation, Deposition Efficiency, and Therapeutic Outcomes

Authors: Farah Owais, Sania Kausar, Sehrish, Qurat-ul-lain Arif
Affiliation: Federal Urdu University of Arts Science and Technology, Karachi

Background and Objective:
Pulmonary delivery of nanomedicines has emerged as a targeted and minimally toxic approach for lung cancer, tuberculosis (TB), and nontuberculous mycobacterial (NTM) infections. Rapid advances in inhalable nanotechnology since 2022—including lipid, polymeric, and hybrid nanocarriers—have led to a surge in clinical trials.

Methods:
This systematic review followed PRISMA guidelines. Searches were conducted in PubMed, Scopus, ClinicalTrials.gov, ChiCTR, and ICTRP from January 2022 to November 2025 using the terms: “inhaled OR pulmonary” AND “nano” AND (“lung cancer” OR “tuberculosis” OR “NTM”) AND (“clinical trial” OR “phase”)*. Inclusion criteria: human studies, Phase I–III trials, inhaled nanoformulations, and articles reporting deposition, pharmacokinetics, or clinical outcomes. Exclusion criteria: animal-only studies, non-inhaled nanomedicines, editorials, reviews without clinical data.A total of 171 records were identified across databases (142 PubMed studies and 29 registered trials). After removal of duplicates and screening for relevance, 142 studies met inclusion criteria. Clinical trial registries (ClinicalTrials.gov and WHO ICTRP) yielded 14 unique clinical trials investigating inhaled nanomedicine approaches for lung cancer, tuberculosis, or NTM.

Results:
Across 21 Phase I–III trials, lung deposition efficiency ranged 38–68% (peripheral/central ratio 1.4–2.9), with lung residence times of 72–168 h for sustained-release nanoformulations. In five Phase II lung cancer trials (n=366), inhaled nano-chemotherapeutics (liposomal cisplatin/carboplatin, docetaxel NPs, nano-paclitaxel) demonstrated a pooled ORR of 51.4% (95% CI 46–57%), and median PFS of 9.2–12.1 months, outperforming historical IV controls (ORR 15–30%). In pulmonary TB/NTM (six trials, n>850), inhaled nano-antibiotics achieved sputum culture conversion rates of 78–89% at 16 weeks, compared to 45–60% with oral therapy alone ($p < 0.0001$). Systemic exposure was reduced by 82–94%, and grade ≥3 pulmonary toxicity remained

Abstract ID: 34

Title: Nigella sativa Seed Extract and Zinc Oxide Nanoparticles: A Green Approach to Formulating Topical Wound Healing Ointment

Authors: Ayeza Abbasi, Hadia Hussain Khan, Aiman Abdul Rehman, Mehwin Mohtashim, Samra Nizam

Affiliation: Ziauddin University, Karachi

Background and Objective:

The growing incidence of antibiotic resistance has prompted the need for alternate strategies for wound healing that are safe, efficacious and sustainable. Herein, we document a topical wound-healing ointment wherein the drug was prepared from the extract of Nigella sativaseeds with biosynthesized zinc oxide nanoparticles (ZnO NPs) synthesized using an eco friendly plant-mediated approach of Aloe vera peel extract.

Methods:

The green fabrication of ZnO NPs was proved by naked eye characterization, FTIR, SEM–EDS analysis (scanning electron microscopy–Energy dispersive spectroscopy), UV–Vis spectrum and XRD studies illustratingthe formation of the nanoparticles, encapsulation of phytochemicals surrounding it, and crystalline wurtzite lattice. The semi-solid fraction mediated with thymoquinone and associated bio-actives documented for its anti-microbial and antioxidant potential.Antibacterial potentials were tested for five multidrug resistant clinical isolates which include Escherichia coli, Klebsiella pneumoniae, Staphylococcus aureus, Pseudomonas aeruginosa and Salmonella typhi.

Results:

Both ZnO NPs and N. sativa extract showed broad-spectrum inhibitory impacts, and the Gram-negative species were more sensitive to them. A primingeffect was seen by combining them simultaneously: the cooperative enhancement of their antibacterial action, still detectable at 1:8 dilution revealed that associations provided a more satisfactory inhibitory profile than those obtainable with single components.The optimized ointment formula was prepared by adding 12.5 mg N. sativa extract and 1.25 mg ZnO NPs to the modified simple ointment base, as determined from the MIC assay results.

Conclusion:

The combined antibacterial, antioxidant and wound healing supportive therapeutic interventions indicate a high potential to serve as an alternative topical application for infected or wounds that fail to heal while being environmentally friendly, low toxic and in line with green synthesis technology as well as the goals of sustainable development by reducing our dependency towards synthetic antimicrobials.

Abstract ID: 35
Title: Fabrication Methods and Applications of Carbon Nanofibers: Recent Advancements in Cancer Therapy
Authors: Zoya Amina, Daniya Nadeema, Kanza Ziaa, Munsif Ali Jatoib, Noor Kamila, Zoha Mahmood
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Background & Objective:
A nanofiber is regarded as a fibrous material with at least one dimension less than 100 nm. Nanofibers hold exceptional properties such as large specific surface (ratio of fibre surface to their volume), small and variable fibre diameter, high porosity, chemical stability, thermal stability and controllable morphology. In this research work, a systemic review of carbon nanofibers is elaborated in the perspective of their specified applications i.e., drug delivery, tissue engineering and cancer therapy.

Methodology:
The study design was established and constructed initially having an intense and detailed literature review by utilizing all available databases related to pharmaceutical sciences in general and drug delivery systems in particular. For this, using systematic literature review (SLR) approach, several highly cited and relevant research articles, proceedings and theses were studied

Results:
Primarily, the carbon nanofibers are discussed in detail with their fabrication methods i.e., growth through chemical vapor deposition; electrospinning; self-assembly; templated synthesis and phase segregation.The applications associated with nanofibers include biomedical and healthcare, environmental remediation, textiles and apparel, electronics, and energy. Concluding the role of nanofibers as anticancer agents in the diagnosis and treatment of various cancers.

Conclusion:
Hence, based upon the systematic literature review and research study, the forthcoming perspectives and recommendations are provided for future insights and applications of carbon nanofibers.

Abstract ID: 50
Title: Nanomedicine-Based Drug Delivery Systems in Precision Oncology: Current Advances and Translational Challenges
Authors: Alvena Noor
Affiliation: Ziauddin University, Karachi

Background & Objectives:
Conventional cancer therapies are often limited by poor selectivity, systemic toxicity, and multidrug resistance. Nanomedicine has emerged as a promising strategy to enhance targeted drug delivery, improve therapeutic efficacy, and reduce adverse effects. This paper aims to review recent advances in nanomedicine-based drug delivery systems in precision oncology, with a focus on their mechanisms, clinical relevance, and translational challenges.

Methods:
A structured literature review was conducted using PubMed, Scopus, and Web of Science databases. Peer-reviewed articles published between 2015 and 2025 were included. Studies focusing on nanoparticle-based drug delivery, tumor targeting mechanisms, clinical outcomes, and translational applications in cancer therapy were analyzed. Mechanisms of Tumor TargetingPassive Targeting Passive targeting relies on the EPR effect, allowing nanoparticles to accumulate in tumor tissues due to leaky vasculature. Active Targeting Active targeting involves ligand–receptor interactions, where nanoparticles are functionalized with antibodies, peptides, or small molecules targeting tumor-specific markers.

Results:
Nanocarriers such as liposomes, polymeric nanoparticles, dendrimers, and inorganic nanoparticles demonstrated improved drug bioavailability, enhanced tumor targeting via passive and active mechanisms, and reduced systemic toxicity. Several nanoformulations, including liposomal doxorubicin and nanoparticle albumin-bound paclitaxel, have shown significant clinical benefits. However, challenges related to scalability, toxicity assessment, regulatory approval, and interpatient variability remain major barriers to widespread clinical translation.
Translational and Regulatory Challenges
Despite promising preclinical data, clinical translation remains limited. Challenges include: Large-scale manufacturing and reproducibility Long-term toxicity and biodistribution concerns Regulatory complexity Tumor heterogeneity and patient-specific variability Addressing these barriers requires interdisciplinary collaboration among scientists, clinicians, and regulatory authorities.

Future Perspectives:
Advancements in biomarker-driven targeting, artificial intelligence-assisted nanoparticle design, and personalized nanomedicine are expected to further enhance the role of nanotechnology in precision oncology.

Conclusion:
Nanomedicine represents a transformative approach in precision oncology, offering targeted, effective, and personalized cancer therapies. Continued research and streamlined regulatory pathways are essential to translate laboratory innovations into routine clinical practice.

Abstract ID: 55
Title: Engineering Nanomedicine for Combination Cancer Immunotherapy
Authors: Dr. Madiha Saeed
Affiliation: COMSATS University Islamabad, Lahore Campus

Cancer immunotherapy is rapidly maturing towards extensive clinical use. However, the clinical translation of cancer immunotherapy-based approaches is hindered by poor tumor specificity, off-target toxicity, and inadequate bioavailability, which has invigorated a fervor of investigation into nanomedicine-based approaches. Nanomedicine-based cancer immunotherapy has emerged as a promising approach to improve the therapeutic potential. The recent advances in the synergistic cancer immunotherapy will be discussed, with a focus on tumor immune microenvironment-responsive highly sophisticated supramolecular prodrugs. How the combination of nanomedicine with photoimmunotherapy, radiation therapy, and chemotherapy can broaden the impact of cancer immunotherapy will be explored. The integration of nanomedicine with potentially curative therapeutic modalities may broaden the clinical performance of conventional approaches.

Abstract ID: 64
Title: Extracellular Vesicle–Derived microRNAs as Liquid Biopsy Tools for Cancer: A Translational Framework for Precision Public Health
Authors: Sami Ur Rehman
Affiliation: Virtual University of Pakistan

Background & Objectives:
Cancer-related mortality in low- and middle-income countries (LMICs) is disproportionately high, largely driven by delayed diagnosis and a lack of affordable molecular diagnostic infrastructure. Extracellular vesicles (EVs), including exosomes, have emerged as critical mediators of intercellular communication, carrying bioactive microRNAs (miRNAs) that reflect the molecular signature of their parent tumor cells. Unlike circulating free-miRNAs, EV-encapsulated miRNAs are protected from RNase-mediated degradation by a lipid bilayer, ensuring high stability in biofluids. This study aims to systematically synthesize evidence on the diagnostic utility of EV-derived miRNAs across major cancers and propose a scalable translational framework for integrating these biomarkers into resource-limited healthcare systems.

Methods:
A systematic literature review was conducted following PRISMA guidelines across PubMed, Scopus, and Google Scholar (2015–2025). The search strategy utilized Boolean operators combining terms such as "Extracellular Vesicles," "microRNA," "Liquid Biopsy," and "Cancer Diagnostics." Inclusion criteria were restricted to peer-reviewed clinical or translational studies reporting EV-associated miRNA profiling in human cohorts (blood, urine, or saliva). Data were extracted regarding EV isolation techniques (e.g., ultracentrifugation, polymer-based precipitation, microfluidics), miRNA quantification methods, and diagnostic sensitivity/specificity. A secondary thematic analysis was performed to identify barriers to clinical adoption in LMICs, forming the basis for the proposed "Precision Public Health" roadmap.

Results:
The review identified a core set of EV-derived miRNAs with high diagnostic reproducibility: miR-21 and miR-1246 for early-stage breast cancer; miR-155, miR-210, and miR-21 for non-small cell lung cancer (NSCLC); and miR-92a and miR-23a for colorectal malignancies. These biomarkers consistently demonstrated the ability to distinguish malignant cases from benign controls with AUC values ranging from 0.82 to 0.96. Furthermore, the study identified that EV-miRNA profiles often correlate with TNM staging and therapeutic resistance. To address the "bench-to-bedside" gap, a three-tiered translational framework was developed: (1) Standardization of low-cost EV enrichment protocols; (2) Leveraging existing COVID-19-era RT-qPCR infrastructure for miRNA quantification; and (3) Implementation of centralized digital platforms for data interpretation in regional diagnostic hubs.

Conclusions:
EV-derived miRNAs represent a robust, minimally invasive class of biomarkers that can revolutionize oncology in settings where tissue biopsies are impractical. By shifting the focus from high-cost sequencing to targeted EV-miRNA panels, precision oncology can be democratized. This framework provides a strategic pathway for public health authorities to transition global nanomedicine research into scalable, life-saving diagnostics.

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