Programme and Abstracts

International Conference on ReproNext: Emerging Horizons in Reproductive Health Research
&
42nd Annual Meeting of the Society for Reproductive Biology and Comparative Endocrinology (SRBCE -2025)

11-13 December, 2025

Organized by

ICMR - National Institute for Research in Reproductive and Child Health, Mumbai (ICMR-NIRRCH)

Convenor

Dr. Geetanjali Sachdeva

Director, ICMR-NIRRCH

Organizing Secretary

Dr. Srabani Mukherjee

Scientist G, ICMR-NIRRCH

Co-Organizing Secretary

Dr. Dipty Singh

Scientist E, ICMR-NIRRCH

Venue

Ravindra Natya Mandir, Sayani Road, Prabhadevi, Mumbai - 400025

ReproNext Emerging Horizons in Reproductive Health Research

PROGRAMME AND ABSTRACTS

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and

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ReproNext Emerging Horizons in Reproductive Health Research

सत्यमेव जयते

डॉ. राजीव बहल, एमडी, पीएचडी.
DR. RAJIV BAHL MD, PhD

सचिव, भारत सरकार
स्वास्थ्य अनुसंधान विभाग
स्वास्थ्य एवं परिवार कल्याण मंत्रालय एवं
महानिदेशक
भारतीय आयुर्विज्ञान अनुसंधान परिषद

Secretary, Government of India
Department of Health Research
Ministry of Health & Family Welfare
Director-General
Indian Council of Medical Research

Message

I am pleased to present this compilation of the abstracts of research observations to be presented at the ***'International Conference on ReproNext: Emerging Horizons in Reproductive Health Research'***, held in conjunction with the ***42nd Annual Meeting of the Society for Reproductive Biology and Comparative Endocrinology (SRBCE)*** at Mumbai from 11th-13th December, 2025. I hope that the gathering of distinguished scientists, clinicians, and young researchers will generate stimulating interactions and the dissemination of new knowledge relevant to health and disease.

Reproductive health is central to the overall well-being of societies, and the rapid advances in reproductive biology, endocrinology and translational medicine are opening new horizons for prevention, diagnosis and treatment of various disorders, diseases and conditions, that though may originate in reproductive organs but are likely to have implications on overall health. Conferences such as ReproNext provide a platform to researchers to share their cutting-edge discoveries, deliberate on challenges and chart pathways for future research that can directly benefit communities across India and the world.

I commend the organizers at ICMR-NIRRCH and SRBCE for curating a scientifically rich program that bridges fundamental science with clinical applications, with an overarching goal to foster dialogue across different disciplines. I hope that the ideas exchanged at the meeting will inspire innovative solutions, strengthen networks, and contribute to the development of effective solutions to address national and global priorities in reproductive health.

I extend my warm congratulations to all contributors and participants, and I look forward to the impact, this collective knowledge will have in shaping healthier generations.

Rajiv Bahl

Rajiv Bahl

V. Ramalingaswamy Bhawan, Ansari Nagar, New Delhi - 110 029
Tel.: 011-26588204, Website : www.icmr.gov.in, E-mail : secy-dg@icmr.gov.in

डॉ. गीतांजलि सचदेवा
पी एच डी, एफ एन एस सी
निदेशक
आई सी एम आर – राष्ट्रीय प्रजनन एवं
बाल स्वास्थ्य अनुसंधान संस्थान
स्वास्थ्य अनुसंधान विभाग, स्वास्थ्य और परिवार
कल्याण मंत्रालय, भारत सरकार

Dr. Geetanjali Sachdeva
PhD, FNASc
Director
ICMR-National Institute for Research in
Reproductive and Child Health
Department of Health Research, Ministry of Health
and Family Welfare, Government of India

11th December, 2025

Message

On behalf of the ICMR-National Institute for Research in Reproductive and Child Health (ICMR-NIRRCH), it is my privilege to welcome you to the International Conference on ReproNext: Emerging Horizons in Reproductive Health Research, and the 42nd Annual Meeting of the Society for Reproductive Biology and Comparative Endocrinology (SRBCE). Hosting this prestigious event in Mumbai is both an honour and a responsibility, as it underscores our commitment to advancing reproductive and child health research in India.

The conference theme "Emerging Horizons in Reproductive Health Research" aptly captures the dynamic landscape of reproductive biology today. From molecular insights into various physiological functions and dysfunctions to breakthroughs in translational medicine, the breadth of topics reflects the interdisciplinary nature of the field. By bringing together leading experts, early-career scientists, clinicians, and young students, ReproNext is likely to serve as a crucible for ideas that may culminate in innovations, inventions and mentorship.

At ICMR-NIRRCH, our mission has always been to translate scientific knowledge into tangible health outcomes. This conference exemplifies that mission by fostering dialogue that bridges laboratory research with clinical practice, and by highlighting the importance of collaborative networks in addressing pressing health challenges.

We gratefully acknowledge the generous financial support provided by the Society for the Study of Reproduction (SSR), USA; Department of Biotechnology (DBT); Department of Science and Technology (DST), and the Council of Scientific and Industrial Research (CSIR). Their contributions have been instrumental in enabling us to convene this gathering of inquisitive minds and to advance the frontiers of reproductive and endocrinology research.

I take this opportunity to sincerely acknowledge the very valuable support extended by Prof. (Dr.) Rajiv Bahl, Director-General, Indian Council of Medical Research; Prof. (Dr.) Sanghmitra Pati, Additional Director-General, Ms. Manisha Saxena, Senior Deputy Director-General (Admin) and Mr. Rajesh Jagdish, Deputy Director-General (Admin) towards organizing this meeting. I would like to put on record my appreciation for Dr. Srabani Mukherjee, Dr. Shailesh Pande and Dr. Dipty Singh for taking the mammoth responsibility of convening the meeting that entailed not only commitment, dedication but also communion with multiple stakeholders at various levels. I express my heartfelt gratitude to the organizing committees, invited speakers, participants and the president and all council members of the SRBCE. I hope that the proceedings of the meeting will serve as a record of scientific excellence and also as a navigator of future research in reproductive biology and endocrinology.

Geetanjali Sachdeva

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SOCIETY FOR REPRODUCTIVE BIOLOGY AND COMPARATIVE ENDOCRINOLOGY (Regd.TN S.No.279 0f 1987)

Secretariat: Department of Endocrinology, Dr ALM PG Institute of Basic Medical Sciences, University of Madras, Taramani Campus, Taramani- Velachery Link Road Chennai - 600 113,India, Phone: 91-44-24547040, Email: srbce1981@gmail.com; Web site: www.srbce.org

Conference message

It is heartening that the ICMR-National Institute for Research in Reproduction and Child Health (NIRRCH), Mumbai is organizing the 42st Annual Meeting of Society for Reproductive Biology and Comparative Endocrinology (SRBCE) and International Conference on ReproNext: Emerging Horizons in Reproductive Health Research; during 11th to 13th December 2025. SRBCE has been a vibrant scientific society that has nurtured hundreds of reproductive biologists and endocrinologists right from its inception in 1981. The society has successfully held its annual meetings for the last forty-four years and has provided a platform for the classical and advanced comparative endocrinologists to intensively interact and to work towards the global and national challenges in the area of reproductive biology. Most importantly, SRBCE has given utmost importance for the young presenters such as doctoral students and post-doctoral researchers.

This year, the ICMR-NIRRCH has taken up the endeavour of organizing the annual meeting of SRBCE with the theme of emerging horizons in reproductive health research. The scientific sessions are very interesting and a galaxy of renowned national and international researchers will be presenting their findings and deliberating on the contemporary issues of reproductive health and endocrinology. I take this opportunity to congratulate Dr. Srabani Mukherjee, Scientist G, ICMR-NIRCRH for taking up this responsibility and wishing her all the success. I also specially thank Dr. Geetanjali Sachdeva, Director, ICMR-NIRRCH for extending all the support to conduct this exciting conference. All the very best to each and every participant of this conference and hope that this event will be fulfilling in all aspects.

Dr Suresh Yenugu
President, SRBCE

SOCIETY FOR REPRODUCTIVE BIOLOGY AND COMPARATIVE ENDOCRINOLOGY (Regd.TN S.No.279 0f 1987)

Secretariat: Department of Endocrinology, Dr ALM PG Institute of Basic Medical Sciences, University of Madras, Taramani Campus, Taramani- Velachery Link Road Chennai - 600 113,India, Phone: 91-44-24547040, Email: srbce1981@gmail.com; Web site: www.srbce.org

Message for the Conference

The 42st Annual Meeting of Society for Reproductive Biology and Comparative Endocrinology (SRBCE) is being hosted by the National Institute for Research in Reproduction and Child Health (NIRRCH), Mumbai during 11th to 13th December 2025. Ever since its inception in 1981, the SRBCE has a unique distinction of hosting annual meetings without a break. The aim of these meetings is to provide a common platform for researchers in the field of Reproductive Biology and Comparative Endocrinology and to build a network for promoting collaborative research. The topics for this meeting encompass diverse aspects of advanced biological sciences. Though, technological advancements and online meetings have become a new norm; meetings in physical mode have a much greater impact to upscale scientific pursuits. Every year, SRBCE meetings are attended in large numbers by scientists and research students from India and abroad wherein; the organizers have maintained high standards of scientific discourses. As a Secretary of this reputed society, I thank all the speakers and attendees from India and abroad for consenting to deliver lectures at this conference. Also, I applaud the efforts of Dr Srabani Mukherjeet, Scientist G, ICMR-NIRRCH and Organizing Secretary, Dr Geetanjali Sachdeva, Director, ICMR-NIRRCH and the team in ensuring a global representation for the conference truly making it an international meeting and wish success for this event.

Dr Ranjitsinh Devkar
Secretary SRBCE

COMMITTEES

Convenor
Dr. Geetanjali Sachdeva
Director, ICMR-NIRRCH, Mumbai

Organizing Secretary
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Scientist G, ICMR-NIRRCH

Co-Organizing Secretary
Dr. Dipty Singh
Scientist E, ICMR-NIRRCH

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Ms. Dhanisha Mohapatra
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PROGRAMME SCHEDULE

International Conference on
ReproNext: Emerging Horizons in Reproductive Health Research

and

42nd Annual Meeting of the Society for Reproductive Biology and Comparative Endocrinology (SRBCE -2025)

11th-13th December, 2025

Venue: Ravindra Natya Mandir, Sayani Road, Prabhadevi, Mumbai-400025

Organized by
ICMR - National Institute for Research in Reproductive and Child Health, Mumbai

Day 1: Inaugural Session
11th December, 2025
Ravindra Natya Mandir

08:45-09:30	**Registration**	
09:30-09:40	**Invocation and Lighting of the Lamp**	
09:40-09:55	**Welcome of the Dignitaries**	
09:55-10:05	Welcome Address	**Dr. Geetanjali Sachdeva** Director, ICMR-NIRRCH
09:55-10:15	About the Conference	**Dr. Srabani Mukherjee** Organizing Secretary and Scientist G, ICMR-NIRRCH
10:15-10:20	About the Society	**Dr. Suresh Yenugu** President, SRBCE
10:20-10:25	Vote of Thanks	**Dr. Ranjitsinh Devkar** Secretary, SRBCE
10:25-11:00	**Chief Guest** **Keynote Address** Cervical Cancer: Cause, Care and Cure (5Cs)	**Padma Shri Dr. Neerja Bhatla** Professor Emeritus, Division of Gynecologic Oncology, NCI-AIIMS, Jhajjar **Chairpersons: Dr. Usha Saraiya, Dr. A. V. Ramachandran**
11:00-11:10	**Group Photo**	
11:10:-11:30	**High tea**	

<table>
<tr><th>Day 1</th><th colspan="3">11th December 2025
Ravindra Natya Mandir</th></tr>
<tr><td>11:30-
12:05</td><td colspan="3">Scientific Session - 1
Plenary Lecture (PL-1)
Chairpersons: Dr. Michael Aruldhas, Dr. Chander Puri
Gonadotropin secretion patterns and female reproductive aging
Dr. Rajendra Kumar, University of Colorado School of Medicine, USA</td></tr>
<tr><td>12:05-
12:25</td><td colspan="3">Invited Lecture (IL-1)
New insights into underlying causes of diabetic kidney disease
Dr. Suresh Mishra, University of Manitoba, Canada</td></tr>
<tr><td>12:25-
12:45</td><td colspan="3">Invited Lecture (IL-2)
Understanding the conundrum of preterm birth: Clues from multiomic investigations
Dr. Arindam Maitra, NIBMG, Kalyani, West Bengal</td></tr>
<tr><td>12:45-
13:05</td><td colspan="3">Invited Lecture (IL-3)
OXPHOS sustains and glycolysis accelerates sperm motility
Dr. Rajender Singh, CDRI, Lucknow</td></tr>
<tr><td>13:05-
13:30</td><td colspan="3">Award Lecture - Prof. Chandana Haldar Best Paper Award
Chairpersons: Dr. A. V. Ramachandran, Dr. Sanjiva Kholkute
Different dietary compositions alter pubertal onset in Wistar rats
Dr. Parth Pandya, Navrachana University, Vadodara</td></tr>
<tr><td>13:30-
14:30</td><td colspan="3">Lunch</td></tr>
<tr><td colspan="2">Ravindra Natya Mandir
Scientific Session - 2A
Chairpersons: Dr. Andreas Meinhardt, Dr. N. K. Lohiya, Dr. Rajender Singh</td><td colspan="2">Lok Kala Dalan 1 (3rd Floor)
Scientific Session - 2B
Chairpersons: Dr. Ashutosh Haldar, Dr. Susan Thomas</td></tr>
<tr><td rowspan="3">14:30-
16:00</td><td rowspan="3">Competitive Oral Presentations - Students
(OP-C-1 to OP-C-11)</td><td>14:30-
14:50</td><td>Invited Lecture (IL-4)
Modulating checkpoint pathways to preserve ovarian reserve and to impede the ovarian aging
Dr. H.B.D. Prasada Rao, NIAB, Hyderabad</td></tr>
<tr><td>14:50-
15:10</td><td>Invited Lecture (IL-5)
DNA break mapping in placental cells uncovers vulnerability of repetitive sequences in pregnancy complication
Dr. Vijay Pratap Singh, ICMR-NIRTH, Jabalpur</td></tr>
<tr><td>15:10-
15:30</td><td>Invited Lecture (IL-6)
Role of dietary quality in the regulation of gut and ovarian physiology in carp
Dr. Sourav Mukherjee, University of North Bengal, Siliguri</td></tr>
<tr><td>16:00-
16:30</td><td colspan="3">Tea Break</td></tr>
</table>

<table>
<tr><td colspan="2">Scientific Session - 3A:
Chairpersons:
Dr. Rupasri Ain, Dr. Bhakti Pathak</td><td colspan="2">Scientific Session - 3B:
Chairpersons:
Dr. Nafisa Balasinor, Dr. Indrasish Bhattacharya</td></tr>
<tr><td>16:30-
16:50</td><td>Invited Lecture (IL-7)
Intergenerational effects of prenatal folate and B12 imbalance: An epigenetic perspective
Dr. Jyotdeep Kaur, PGIMER, Chandigarh</td><td>16:30-
16:50</td><td>Invited Lecture (IL-11)
Disruptor of GSK3α: A non-hormonal contraceptive that targets maturing spermatozoa in epididymis
Dr. Souvik Dey, MAHE, Manipal</td></tr>
<tr><td>16:50-
17:10</td><td>Invited Lecture (IL-8)
Environment, epigenetics and embryogenesis: maternal-child health in the face of climate change
Dr. Srimonta Gayen, IISc Bangalore</td><td>16:50-
17:10</td><td>Invited Lecture (IL-12)
L-NAME induces spermiation failure and transgenerational sperm defects in Wistar rat
Dr. Nishi Kumari, ICMR-NIRRCH</td></tr>
<tr><td>17:10-
17:30</td><td>Invited Lecture (IL-9)
Inflammation-driven protein kinases signaling association with ovulation during endometriosis
Dr. Rajesh Kumar Jha, CDRI, Lucknow</td><td>17:10-
17:30</td><td>Invited Lecture (IL-13)
3D-organoids as an emerging in- vitro experimental model for studying prostate cancer associated-one metastasis: Interaction of cancer cells with pre-osteoblasts
Dr. Vani Venkatappa, IISc, Bangalore</td></tr>
<tr><td rowspan="2">17:30-
17:50</td><td rowspan="2">Invited Lecture (IL-10)
Maternal exposure to nonylphenol (Np) induces ovarian dysfunction and transgenerational developmental impairments in zebrafish (Danio Rerio)

Dr. Sudipta Maitra, Vishwa Bharti, WB</td><td>17:30-
17:40</td><td>Oral Presentation - Non-Competitive - 1
Integrative transcriptomic profiling reveals immune-metabolic hub genes and therapeutic targets in PCOS
Dr. Devaraj Sankarganesh, VIT, Vellore</td></tr>
<tr><td>17:40-
17:50</td><td>Oral Presentation - Non-Competitive - 2
Clinical, biochemical features and transcriptomic responses in PCOS subjects with metabolic syndrome
Dr. Nirupama Chatterjee, Artemis Hospitals, Gurgaon</td></tr>
<tr><td rowspan="3">17:50-
19:00</td><td rowspan="3">Exhibition Hall (1st Floor)
Scientific Session - 4

Poster Presentations
(PP-C-1 to PP-C-21)</td><td>17:50-
18:00</td><td>Oral Presentation - Non-Competitive - 3
Uterine alterations in PCOS and reproductive senescence: Melatonin in rescue
Dr. Shruti R. Hansda, BHU, Varanasi</td></tr>
<tr><td>18:00-
18:10</td><td>Oral Presentation - Non-Competitive - 4
Effects of Bisphenol S on reproductive health: Role of melatonin
Dr. Rakesh Verma, BHU, Varanasi</td></tr>
<tr><td>18:10-
19:00</td><td>Executive Committee meeting of SRBCE</td></tr>
<tr><td>19:00-
20:00</td><td colspan="3">Ravindra Natya Mandir
Cultural Programme</td></tr>
<tr><td>20:00-
21:00</td><td colspan="3">Gala Dinner</td></tr>
</table>

Day 2	12th December 2025 Mini Auditorium (3rd floor)		
Scientific Session - 5 **Chairpersons: Dr. Thimmappa Shivanandappa, Dr Rajendra Kumar**			
09:30-10:00	**Oration Lecture - *Prof. B. B. Kaliwal Gold Medal Oration*** Endocrine research in wildlife: Scope in conservation and management **Dr. Govindhaswamy Umapathy**, CSIR-CCMB, Hyderabad		
10:00-10:25	**Award Lecture - *Prof. S. K. Maitra Best Paper Award*** In-vitro analysis of *Solanum virginianum* L. extract and melatonin: Synergistic induction of apoptosis in Mda-Mb-231 breast cancer cells **Dr. Darshee Baxi**, Navrachana University, Vadodara		
10:25-10:55	**Plenary Lecture (PL-2)** Unveiling the immune shield – A dive into testicular and epididymal immunology **Dr. Andreas Meinhardt**, Justus-Liebig-University Giessen, Germany		
10:55-11:10	**Tea Break**		
Scientific Session - 6 **Chairpersons: Dr. Andreas Meinhardt, Dr. Ashutosh Halder**			
11:10-11:40	**Plenary Lecture (PL-3)** Early gestational diabetes – What is new? **Padma Shri Dr. V Mohan**, Dr. Mohan's Diabetes Specialities Centre, Chennai		
11:40-12:00	**Invited Lecture (IL-14)** Disorders of sexual differentiations **Dr. Anurag Lila**, KEM Hospital, Mumbai		
Mini Auditorium (3rd Floor) ***Scientific Session - 7A*** **Chairpersons:** **Dr. Mona Sharma, Dr. Deepak Modi**		**Lok Kala Dalan 1 (3rd Floor)** ***Scientific Session - 7B*** **Chairpersons:** **Dr. Ilangovan Ramachandran, Dr. Venkataraman P**	
12:00-12:20	**Invited Lecture (IL-15)** Growth hormone's role in diabetic kidney disease: A new role for an old hormone **Dr. Anil Kumar Pasupalati**, University of Hyderabad	12:00-12:20	**Invited Lecture (IL-19)** Microfluidic chip-based system for subtype-specific detection of circulating tumour cells in breast cancer **Dr. Satish Ramalingam**, SRM Institute of Science and Technology, Chennai
12:20-12:50	**Invited Lecture (IL-16)** Cross-species transcriptomic insights into the interplay between adipogenesis and angiogenesis **Dr. Suttur Malini**, University of Mysore	12:20-12:50	**Invited Lecture (IL-20)** HMGB1-RAGE axis and the dynamics of immune cells during endometrial repair in rat model of induce menstruation **Dr. Uddhav Chaudhari**, ICMR-NIRRCH
12:50-13:10	**Invited Lecture (IL-17)** Can early pregnancy microbiome–immune signatures predict the risk of adverse outcomes? **Dr. Vikrant Bhor**, ICMR-NIRRCH	12:50-13:10	**Invited Lecture (IL-21)** High-fat diet activates pro-inflammatory response in the mouse prostate **Dr. Natarajan Bhaskaran**, Saveetha Medical College

<table>
<tr><td>13:10-
13:30</td><td>Invited Lecture (IL-18)
Seasonal breeding as the “Nature’s Contraceptive”? The story from songbirds
Dr. Vinod Kumar, KGMU, Lucknow</td><td>13:10-
13:30</td><td>Invited Lecture (IL-22)
Dysregulated seminal miRNAs reveal aberrant functional pathways in unexplained male infertility
Dr. Gagandeep Kaur Gahlay, Guru Nanak Dev University, Amritsar</td></tr>
<tr><td>13:30-
14:30</td><td colspan="3">Lunch</td></tr>
<tr><td>14:30-
16:10</td><td colspan="3">Mini Auditorium (3rd Floor)
Scientific Session - 8

Chairpersons: Dr. Rajendra Kumar, Dr. Smita Mahale, Dr. Arnab Banerjee
Competitive Oral Presentations - Students (OP-C-12 to OP-C-22)</td></tr>
<tr><td>16:10-
16:25</td><td colspan="3">Tea Break</td></tr>
<tr><td rowspan="4">16:25-
17:30</td><td rowspan="4">Exhibition Hall (1st Floor)
Scientific Session - 9

Poster Presentations
(PP-C-22 to PP-C-43)</td><td>16:25-
16:40</td><td>BD FACSDiscover S8: Image enabled spectral cell sorter
Swapnil C Walke, BD BioSciences</td></tr>
<tr><td>16.40-
16.50</td><td>Reprogenomics: Sequencing & CMA in clinical care
Dr. Vinay Varghese, Thermo Fisher Scientific</td></tr>
<tr><td>16.50-
17.00</td><td>Key technological advances in Digital PCR making it a promising tool for genetic testing
Dr. Rana Pratap Singh, QIAGEN India Pvt</td></tr>
<tr><td>17.00-
17.10</td><td>Spectral Flow Cytometry: A new lens for understanding complex disease
Dr. Abhishek Chowdhury, Premas Life Sciences</td></tr>
<tr><td>17:30-
19:00</td><td colspan="3">Mini Auditorium (3rd Floor)

Prof. A. V. Ramachandran Best Research Paper Award for paper published in JER

Scroll of Honour

Lifetime Achievement Award

General Body Meeting of SRBCE
Conducted by Dr. Ranjitsinh Devkar, Secretary SRBCE</td></tr>
<tr><td>19:00-
20:00</td><td colspan="3">Dinner</td></tr>
</table>

Day 3	**13th December 2025** **Mini Auditorium (3rd Floor)** ***Scientific Session - 10*** **Chairpersons: Dr. Taru Sharma, Dr. Geetanjali Sachdeva**
09:30-10:00	**Plenary Lecture (PL-4)** Mammalian egg activation: Exploring Ca^{2+} oscillations and the influence of other divalent ions **Dr. Rafael Fissore**, University of Massachusetts, Amherst, USA
10:00-10:20	**Invited Lecture (IL-23)** Molecular underpinnings governing genetic complexity of the prostate cancer and pathways to targeted therapy **Dr. Bushra Ateeq**, Indian Institute of Technology Kanpur
10:20-10:40	**Invited Lecture (IL-24)** A case for maternal screening and intervention to prevent congenital CMV infection (cCMV) **Dr. Vainav Patel**, ICMR-NIRRCH, Mumbai
10:40-10:55	**Tea Break**
Scientific Session - 11 **Chairpersons: Dr. Vikas Dighe, Dr. Rajalakshmi Manikkam, Dr Shailesh Pande**	
10:55-11:15	**Invited Lecture (IL-25)** Fertility preservation in oncological patients: Challenges and opportunities **Dr. Satish Adiga**, MAHE, Manipal
11:15-11:25	**Competitive Oral Presentation - Young Scientist - 1** Molecular insights into zebrafish ovulation: Perspectives on redox homeostasis, inflammatory mediators, and mitochondrial bioenergetics **Dr. Soumyajyoti Ghosh**, Visva-Bharati University, Santiniketan, West Bengal
11:25-11:35	**Competitive Oral Presentation - Young Scientist - 2** Molecular insights into Ad4bp/Sf-1 mediated regulation of steroidogenesis in catfish **Dr. Sonika Kar**, University of Hyderabad
11:35-11:45	**Competitive Oral Presentation - Young Scientist - 3** Reduced insulin/IGF-1 signaling extends reproductive span through somatic gonadal collagen-mediated maintenance of oocyte quality in *Caenorhabditis elegans* **Dr.Neha Kaushik**, AIIMS, New Delhi
11:45-11:55	**Competitive Oral Presentation - Young Scientist - 4** Maternal plasma Human Cytomegalovirus (HCMV) DNA positively correlates with viral shedding and altered microbiome composition in breast milk **Gauri Bhonde**, ICMR-NIRRCH, Mumbai
11:55-12:05	**Competitive Oral Presentation - Young Scientist - 5** Regulatory functions of circular RNAs in prostate cancer metastasis **Dr. Bodhana Dhole**, AIIMS, New Delhi
12:05-13:30	***Scientific Session - 12*** **Panel Discussion:** Reproduction in a changing world: A one health view of effect of environment on fertility

	Moderator: Dr. Anushree Patil, ICMR-NIRRCH, Mumbai **Panelists:** **Dr. Priyank Kothari**, Andrologist, ICMR-NIRRCH **Dr. Vandana Bansal**, Foetal Medicine Expert, Surya Hospitals **Dr. Vyankatesh Shivane**, Metabolic Expert, KEM Hospital **Dr. Panchali Moitra**, Nutrition Expert, SNDT Women's University **Dr. Ravindra Zende**, Veterinary Public Health Expert, Mumbai Veterinary College **Dr. Harish Phuleria**, Environmental Science Expert, IIT Bombay **Dr. Surya S. Durbha**, Agriculture Science Expert, Mississippi State University
13:30-14:30	**Lunch**
14:30-15:45	***Valedictory Ceremony*** **Chief Guest: Dr. Manoj Kumar Dhar**, Director, AcSIR

Mini Auditorium (3rd Floor)

Valedictory Ceremony of ReproNext 2025
13th December 2025

14:30-15:00	Welcome and Address of Chief Guest **Dr. Manoj Kumar Dhar**, Director, AcSIR
15:10-15:25	Glimpses of the Conference **Dr. Dipty Singh**
15:25-15:40	Certificate distribution
15:40-15:45	Vote of Thanks **Dr. Srabani Mukherjee**
15:45-15:55	Group Photo
15.55 onwards	**High Tea**

ABSTRACTS

KEYNOTE ADDRESS

KA-1	**Padma Shri Dr. Neerja Bhatla** *Professor Emeritus, Department of Preventive Oncology, National Cancer Institute Jhajjar, AIIMS Delhi* **Cervical Cancer: Cause, Care and Cure (5Cs)**

KA-1

CERVICAL CANCER: CAUSE, CARE AND CURE (5CS)

Neerja Bhatla

Professor Emeritus, Department of Preventive Oncology, National Cancer Institute Jhajjar, Former Head, Department of Obstetrics & Gynaecology, AIIMS Delhi

Email: neerja.bhatla07@gmail.com

Cervical cancer is one of the few cancers that has a viral etiology. Persistent infection with high-risk genotypes of the human papillomavirus (HPV) was identified as the cause of cervical cancer. This breakthrough discovery led to Professor Harald zur Hausen being awarded the Nobel Prize in 2008. His discovery paved the way for the development of HPV vaccines and nucleic acid-based HPV tests. India has also developed an indigenous HPV vaccine and diagnostic tests. These arsenals have equipped our country with effective countermeasures against cervical cancer.

In 2020, the World Health Organization launched the global strategy based on a three-pillar approach:

i. HPV vaccination of 90% of girls before the age of 15 years

ii. Screening of 70% of women with an HPV test by 35 years and again by 45 years

iii. Treatment and care of 90% of women detected to have a preinvasive or invasive lesion.

Modelling studies indicate that if these targets are in place by 2030, India can achieve the elimination target of 4 per 100,000 person-years by 2062. It is an exciting challenge because it is doable. We have the basic tools: a national cancer screening policy, an indigenous HPV vaccine, point-of-care HPV tests, portable colposcopes, and treatment devices. Research from India paved the way for fewer dose schedules, thereby improving affordability and availability. Same-day treatment is now a reality. Women who desire future fertility now have options of conservative management to retain their reproductive potential if detected early.

We need to string the available tools together now, enable linkages and referral systems, and take advantage of the digital revolution to establish screening and vaccine registries to be linked to the cancer registries. We shall also explore public-private partnerships and corporate social responsibility to fill in the gaps and close the loop. We also need to augment our cancer care services, training of cadres in screening and treatment at primary, secondary, and tertiary levels, provision of requisite equipment, and attention to quality control.

At the same time, we must continue our efforts to develop new and better methods. We shall develop effective models of care for screening and care through research, for applicability in public health care settings. Development of affordable HPV tests needs to be scaled up. We need more innovations, development of various types of predictive and prognostic biomarkers, etc. Therapeutic vaccines will be a game-changer. These vaccines are presently under development to treat HPV infection and to treat cervical intraepithelial neoplasia. Improvements in prophylactic vaccines, expanding the target groups, and improved access will expedite attaining the elimination goal. Together, we can ensure that cervical cancer is a scourge of the past.

PLENARY LECTURES

PL-1	**Dr. T. Rajendra Kumar, PhD** *Professor & Edgar L., Patricia M. Makowski and Family Endowed Chair Division of Reproductive Sciences Department of Obstetrics & Gynecology CU Anschutz School of Medicine Aurora, Colorado, USA* **Gonadotropin Secretion Patterns and Female Reproductive Aging**
PL-2	**Dr. Rafael A. Fissore** *Department of Veterinary and Animal Sciences, University of Massachusetts, Amherst, Massachusetts, USA* **Mammalian Egg Activation: Exploring Ca^{2+} Oscillations and the Influence of Other Divalent Ions**
PL-3	**Padma Shri Dr. V. Mohan, M.D.** *Dr. Mohan's Diabetes Specialities Centre & Madras Diabetes Research Foundation, Chennai* **Early Gestational Diabetes – What is New?**
PL-4	**Dr. Andreas Meinhardt** *Justus-Liebig-University Giessen, Germany* **Unveiling THE Immune Shield – A Dive into Testicular and Epididymal Immunology**

GONADOTROPIN SECRETION PATTERNS AND FEMALE REPRODUCTIVE AGING

T. Rajendra Kumar

Professor & Edgar L., Patricia M. Makowski and Family Endowed Chair Division of Reproductive Sciences Department of Obstetrics & Gynecology CU Anschutz School of Medicine Aurora, Colorado, USA

Email: raj.kumar@cuanschutz.edu

The gonadotropic hormones, luteinizing hormone (LH) and follicle-stimulating hormone (FSH) are heterodimers, synthesized in gonadotropes and regulated by gonadotropin-releasing hormone. Yet, they have evolved distinct modes of cellular secretion. LH is secreted as pulses from the regulated release pathway while FSH is constitutively secreted. Why this hormone-specific pattern has evolved and whether the target organ, the ovary, senses specific hormone release pattern as a distinct signal input is not understood. We have engineered mice in which the intracellular trafficking and secretion pattern of FSH is genetically re-routed. Our in-vivo approach combined with multi-omics analyses allowed us to investigate the ovarian responses to altered pattern of FSH signalling and the resulting dramatic ovarian phenotypes including enhanced ovulations and extended female reproductive lifespan. The ability to modify the secretory fate of proteins in vivo has pathophysiological significance and could explain the etiology of several hormone hyperstimulation and resistance syndromes. Our studies provide a molecular basis for the evolution of gonadotropin secretion patterns and explain the origin of estrus cycles in mammals using trackable mouse genetic models.

PL-2

MAMMALIAN EGG ACTIVATION: EXPLORING CA^{2+} OSCILLATIONS AND THE INFLUENCE OF OTHER DIVALENT IONS

Rafael A. Fissore

Department of Veterinary and Animal Sciences, University of Massachusetts, Amherst, Massachusetts, USA

Email: rfissore@vasci.umass.edu

Calcium oscillations are the driving signal for egg activation and the initiation of embryonic development in mammals. The sperm factor PLCζ1 is the primary trigger of these oscillations. In mice, however, males lacking PLCζ1 remain subfertile, and our findings show that another PLC can function as a backup, providing limited fertility. Importantly, these PLCs act in an interdependent manner to ensure the timely onset of fertilization and developmental success. Beyond calcium, two other divalent cations, zinc (Zn^{2+}) and magnesium (Mg^{2+}), play indispensable roles in meiosis and early development. We identified TRP channels, specifically TRPV3 and TRPM7, as key regulators of Zn^{2+} and Mg^{2+} homeostasis during these stages. However, the mechanisms controlling the functional activity of these TRP channels and the cellular targets of these ions remain largely unknown. In this talk, I will highlight the critical steps regulated by these PLCs and TRP channels, discuss unresolved questions, and point to promising directions for future research.

EARLY GESTATIONAL DIABETES – WHAT IS NEW?

V. Mohan

Chairman & Chief of Diabetology, Dr. Mohan's Diabetes Specialities Centre & Madras Diabetes Research Foundation, Chennai, India

Email: drmohans@diabetes.ind.in

Gestational diabetes (GDM) refers to glucose intolerance first detected during pregnancy. Traditionally, GDM was diagnosed between 24 – 28 weeks of pregnancy. More recently, GDM is being diagnosed in the first trimester of pregnancy and this is referred to as 'Early GDM' and the traditional GDM is called 'Late GDM'.

The prevalence of Early GDM varies widely due to different criteria being used, however it is highest in S. Asians and Indians. Women with Early GDM present with worse metabolic profile than Late GDM. The pathophysiology of Early GDM appears to be due to both worse beta-cell dysfunction as well as greater insulin resistance compared to late GDM. Increased occurrence of adverse pregnancy outcomes, higher incidence of postpartum dysglycemia and a greater need for insulin use have been reported in women with Early GDM. The Treatment Of BOoking Gestational diabetes Mellitus (ToBOGM) study, which is a randomised control trial published in New England Journal of Medicine (NEJM), showed the benefits of early treatment in women with Early GDM in terms of better neonatal outcomes. It is important to screen all pregnant women in India before the 14th week of pregnancy to pick up early GDM. This can help to improve the outcomes both in the mother and the child.

PL-4

UNVEILING THE IMMUNE SHIELD – A DIVE INTO TESTICULAR AND EPIDIDYMAL IMMUNOLOGY

Andreas Meinhardt

Justus-Liebig-University Giessen, Germany

Email: andreas.meinhardt@anatomie.med.uni-giessen.de

Macrophages are sentinel cells that play an essential role in tissue homeostasis, tissue repair, and inflammation resolution, as well as being a key player in the normal innate immune response of almost all organs. Testicular and epididymal macrophages represent the most abundant immune cell type in the respective organs. These cells are involved in regulating the delicate balance between inciting an inflammatory response against invading pathogens and supporting the normal homeostasis of the organ. To gain insight into the heterogeneity of macrophages in the adult mouse testis and epididymis single cell RNA seq analysis showed distinct subclusters of macrophages that are thought to occupy specific niches. Infections of the human testis and epididymis are usually caused by bacterial infections, which lead to an infiltration of leukocytes and subsequent damage to both organs. Our studies suggest that infiltrating monocyte-derived macrophages play an important role in promoting tissue damage and blocking their entry can attenuate the progression of diseases. In the testis, macrophage seems to be responsible for controlling excessive immune reactions, helping to restore initial tissue damage four weeks after infection with uropathogenic E. coli. The epididymis is different as along the anatomical axis of the epididymis, an immunological gradient with strong inflammatory amplitude in the cauda, and low inflammatory amplitude in the initial segment is observed. Combined scRNA-seq with immunofluorescence analysis point to locally restricted macrophage-driven regulatory circuits regulating the different immunoreactions in the testis and epididymal regions.

AWARD LECTURES

AL-1	***Prof. B. B. Kaliwal Gold Medal Oration*** **Govindhaswamy Umapathy** *Laboratory for the Conservation of Endangered Species (LaCONES), CSIR-CCMB* **Endocrine Research in Wildlife: Scope in Conservation and Management**
AL-2	***Prof. S. K. Maitra Best Paper Award*** **Darshee Baxi** *Navrachana University, Vadodara* **In-Vitro Analysis of *Solanum virginianum* L. Extract and Melatonin: Synergistic Induction Of Apoptosis In Mda-Mb-231 Breast Cancer Cells**
AL-3	***Dr. Chandana Haldar Best Paper Award*** **Parth Pandya** *Navrachana University, Vadodara* **Different Dietary Compositions alter Pubertal Onset in Wistar Rats**

Prof. B. B. Kaliwal Gold Medal Oration

ENDOCRINE RESEARCH IN WILDLIFE: SCOPE IN CONSERVATION AND MANAGEMENT

Govindhaswamy Umapathy

Laboratory for the Conservation of Endangered Species (LaCONES)

CSIR- Centre for Cellular and Molecular Biology (CCMB)

Email: guma.ccmb@csir.res.in

The number of critically endangered species has significantly increased in recent years, necessitating urgent protection to prevent their extinction in the wild. Conservation efforts should include both in situ (on-site) and ex situ (off-site) management of populations. Conservation Physiology is an emerging discipline that focuses on how endocrine studies can aid in wildlife conservation. It draws on a wide range of existing research areas, including theoretical, diagnostic, and management studies, to enhance the survival and reproduction of threatened animals. Accurate information about a species' reproductive biology and health is essential for effective management of animals in both captivity and the wild. Using non-invasive hormone analysis on faecal, hair, saliva, or feather samples, we can monitor levels of estrogen, progestogen, glucocorticoids, and androgens. This analysis allows us to assess the reproductive status, health, and stress levels of wild animals over both short and long-term periods. Our research programs on hormone monitoring in wild animals, utilising non-invasive samples, have provided valuable insights that help wildlife managers better conserve endangered species in both captivity and the wild. In this talk, I will discuss the various procedures and methods involved in non-invasive hormone analysis, as well as recent research findings in this area.

Prof. S. K. Maitra Best Paper Award

IN-VITRO ANALYSIS OF SOLANUM VIRGINIANUM L. EXTRACT AND MELATONIN: SYNERGISTIC INDUCTION OF APOPTOSIS IN MDA-MB-231 BREAST CANCER CELLS

Darshee Baxi

Associate Dean and Program Chair, Associate Professor, Department of Biomedical and Life Sciences, School of Science, Navrachana University, Vadodara

Email: darsheeb@nuv.ac.in; darsheebaxi@hotmail.com

Solanum virginianum (Sv) herb finds widespread usage in various medical systems, notably it is a component of the traditional herbal formulation "Dashamula". Melatonin is widely acknowledged as a chemical and a cell defender owing to its anti-oxidative and immunomodulatory properties. Despite extensive references in traditional medicine, systematic evaluations of *Solanum virginianum* anticancer properties are scarce. Combination therapy has emerged as a promising strategy for tackling drug-resistant cancers, inhibiting tumour progression, and enhancing therapeutic efficacy. This study aims to investigate the anticancer effects of *Solanum virginianum* plant extract and melatonin, individually and in combination, on breast cancer cells. The anticancer potential of individual treatments, as well as combinations of *Solanum virginianum* leaf extract and melatonin, was evaluated using cell migration inhibition, clonogenic assay, DNA fragmentation assay, nuclear morphology study, flow cytometry (FACS) analysis, and gene expression investigation. The methanolic leaf extracts of *Solanum virginianum* demonstrated significant anti-proliferative effects on MDA-MB-231 breast cancer cells. Furthermore, the combination of *Solanum virginianum* methanolic leaf extract and melatonin exhibited anti-migratory and anti-tumorigenic properties. This was further confirmed by gene expression studies related to both intrinsic and extrinsic apoptotic pathways. Notably, In combination groups, there was an increased expression of apoptotic genes (CASP3 ($p < 0.001$), CASP8 ($p < 0.05$), CASP9 ($p < 0.05$), BAX ($p < 0.01$) and anti-inflammatory genes (IL4, IL10), along with a decreased expression of the anti-apoptotic gene (BCL2) and metastatic genes (MMP2, MMP9). Overall, present study is the first comprehensive investigation into the potential of *Solanum virginianum* as an anticancer agent in conjunction with melatonin, highlighting the promising application of a combination approach for breast cancer treatment.

Dr. Chandana Haldar Best Paper Award

DIFFERENT DIETARY COMPOSITIONS ALTER PUBERTAL ONSET IN WISTAR RATS

Harsh Shah, Nehareeka Dan, Ankita Salunke, A. V. Ramachandran, and **Parth Pandya**

TREE Lab, Division of Biomedical and Life Sciences, School of Science, Navrachana University, Vadodara

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Puberty is a crucial developmental phase influenced by neuroendocrine signals involving various neuropeptides and hormones. The impact of various diet combinations on the initiation of puberty in female rats was studied, along with the crosstalk between hypothalamic neuropeptides that might influence it. Weaned female Wistar rats were segregated into five groups and given different diets for a duration of 21 d. The diet included high-fat, high-carbohydrate, high-protein, cafeteria diet and standard chow. Throughout the 21-day period, body weight, vaginal opening and blood samples were recorded. Post sacrifice parameters like hormonal analysis, gene expression, protein expression and Immunolocalization were performed. The High Fat Diet (HFD), High carbohydrate Diet (HCD), and Cafeteria diet (CafD) groups exhibited early vaginal opening, increased body weight, elevated somatic indices, and Follicle-stimulating Hormone (FSH), Luteinizing Hormone (LH), and estradiol levels were elevated in regards to the control group. Gene expression analysis showed upregulation of Kiss1, Kiss1r, Pomc, Lep, Lepr, and Gnrh, while Npy, Agrp, and Mkrn3 were downregulated. Protein expression studies confirmed the increased levels of KISS1, KISS1R, LEPR, and POMC, particularly in the HFD and HCD groups. Histone acetylation analysis revealed higher global acetylation in the hypothalamus of HFD and HCD groups. This study highlights the significant role of dietary composition in modulating pubertal onset through the neuroendocrine pathways. These findings suggest that high-fat and high-carbohydrate diets expedite puberty by altering the expression of key hypothalamic neuropeptides and hormones. This underscores the importance of nutrition in reproductive development and its broad implications for adolescent health.

INVITED LECTURES

IL-1	**Suresh Mishra** *University of Manitoba, Canada* **New Insights into Underlying Causes of Diabetic Kidney Disease**
IL-2	**Arindam Maitra** *BRIC-NIBMG, Kalyani, West Bengal* **Understanding the Conundrum of Preterm Birth: Clues from Multiomic Investigations**
IL-3	**Rajender Singh** *CSIR-CDRI, Lucknow* **OXPHOS Sustains and Glycolysis Accelerates Sperm Motility**
IL-4	**Natarajan Bhaskaran** *Saveetha Medical College, Chennai* **High-Fat Diet Activates Pro-Inflammatory Response in the Mouse Prostate**
IL-5	**H. B. D. Prasada Rao** *BRIC-NIAB, Hyderabad* **Modulating Checkpoint Pathways to Preserve Ovarian Reserve and to Impede Ovarian Aging**
IL-6	**Sourav Mukherjee** *University of North Bengal, Siliguri, Darjeeling* **Role of Dietary Quality in the Regulation of Gut and Ovarian Physiology in Carp**
IL-7	**Vijay Pratap Singh** *ICMR-NIRTH, Jabalpur* **DNA Break Mapping in Placental Cells Uncovers Vulnerability of Cgg Repetitive Sequences in Pregnancy Complication**
IL-8	**Sameer Gupta** *Institute of Science, BHU, Varanasi* **Developmental Exposure to Phthalates Adversely affects Metabolic Outcomes in Male Offspring**

IL-9	**Jyotdeep Kaur** *PGIMER, Chandigarh* **Intergenerational Effects of Prenatal Folate and B12 Imbalance: An Epigenetic Perspective**
IL-10	**Srimonta Gayen** *Indian Institute of Science, Bangalore* **Environment, Epigenetics and Embryogenesis: Maternal-Child Health in the Face of Climate Change**
IL-1	**Rajesh Kumar Jha** *CSIR-CDRI, Lucknow* **Inflammation-Driven Protein Kinases Signaling Association with Ovulation During Endometriosis**
IL-12	**Vainav Patel** *ICMR-NIRRCH, Mumbai* **A Case for Maternal Screening and Intervention to Prevent Congenital CMV Infection (cCMV)**
IL-13	**Souvik Dey** *Manipal Academy of Higher Education, Manipal* **Disruptor of Gsk3α: A Non-Hormonal Contraceptive that Targets Maturing Spermatozoa in Epididymis**
IL-14	**Nishi Kumari** *ICMR-NIRRCH, Mumbai* **L-Name Induces Spermiation Failure and Transgenerational Sperm Defects in Wistar Rat**
IL-15	**Vani Venkatappa** *Indian Institute of Science, Bangalore* **3D-Organoids as an emerging in- vitro experimental model for studying prostate cancer associated-bone metastasis: Interaction of cancer cells with pre-osteoblasts**
IL-17	**Anil K Pasupulati** *University of Hyderabad* **Growth Hormone's Role in Diabetic Kidney Disease: A New Role for an Old Hormone**

IL-18	**Suttur Malini** *University of Mysore* **Cross-Species Transcriptomic Insights into the Interplay between Adipogenesis and Angiogenesis**
IL-19	**Vikrant Bhor** *ICMR-NIRRCH, Mumbai* **Can Early Pregnancy Microbiome–Immune Signatures Predict the Risk of Adverse Outcomes?**
IL-20	**Vinod Kumar** *King George's Medical University, Lucknow* **Seasonal Breeding as the "Nature's Contraceptive"? The Story from Songbirds**
IL-21	**Satish Ramalingam** *SRM Institute of Science and Technology, Chennai* **Microfluidic Chip-Based System for Subtype-Specific Detection of Circulating Tumour Cells in Breast Cancer**
IL-22	**Raghav Kumar Mishra** *Institute of Science, BHU, Varanasi* **Chronic Unpredictable Stress Induces Male Sexual Dysfunction: A Complex Interplay of Neurotransmitters and Erection Pathway**
IL-23	**Uddhav Chaudhari** *ICMR-NIRRCH, Mumbai* **HMGB1-RAGE Axis and the Dynamics of Immune Cells during Endometrial Repair in Rat Model of induce Menstruation**
IL-24	**Amaresh Kumar Singh** *Institute of Science, BHU, Varanasi* **Investigating the Etiology of Osteoarthritis and the Impact of Nutraceuticals on the Onset and Progression of Osteoarthritis**
IL-25	**Sudipta Maitra** *Visva-Bharati University, Santiniketan, West Bengal* **Maternal Exposure to Nonylphenol (Np) Induces Ovarian Dysfunction and Transgenerational Developmental Impairments in Zebrafish (*Danio Rerio*)**

IL-26	**Bushra Ateeq** *Indian Institute of Technology, Kanpur* **Molecular Underpinnings Governing Genetic Complexity of the Prostate Cancer and Pathways to Targeted Therapy**
IL-27	**Satish Kumar Adiga** Kasturba Medical College, Manipal **Fertility Preservation in Oncological Patients: Challenges and Opportunities**

NEW INSIGHTS INTO UNDERLYING CAUSES OF DIABETIC KIDNEY DISEASE

Suresh Mishra

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Chronic kidney disease (CKD) remains among the top causes of morbidity and mortality in the world, affecting around 11% of the population globally. The number of people receiving renal replacement therapy worldwide exceeds 2.5 million and is expected to double to 5.4 million by 2030. Currently, there is no cure for this disease and the treatment primarily focuses on disease management. Over a period of time, patients with CKD starts to lose their kidney function and may progress to end-stage kidney disease (ESKD) when the kidneys are no longer able to work at a level needed for day-to-day life. The economic burden of CKD and ESKD is staggering worldwide. Diabetes remains a significant (~ 40%) contributor to CKD in adults. However, our knowledge of the underlying molecular mechanisms involved in new-onset diabetic kidney disease (DKD) and its progression to ESKD remains limited. Prohibitin-1 (PHB1) is an evolutionarily conserved pleiotropic protein that primarily resides in the mitochondria. Transgenic mouse models of PHB1 have revealed its role in biological sex-related differences in adipose and immune functions. However, the mechanisms involved remain largely unclear. Moreover, it is not known whether PHB1 plays a role in sex-related differences in other cell and tissue types. To explore this at the systemic level, we focused on two key conserved post-translational modification sites in PHB1 (i.e., the Cys69 and Tyr114 residues, which we have identified before) and developed Phb1C69A and Phb1Y114F knock-in mouse models (together referred to as the Phb1-Ki mice) using state-of-the-art CRISPR-Cas9 technology. Consistent with previous findings from the PHB1 and mutant-PHB1Y114F transgenic mice, both Phb1-Ki mouse models displayed altered sex-related differences in adipose and immune phenotypes, signifying the importance of the Cys69- and Tyr114-linked functions in the sexually dimorphic features of PHB1. Interestingly, during their phenotypic characterization, a sex-related difference in their kidney sizes were apparent. Further analysis revealed structural abnormalities in PTECs mitochondria and in glomerular podocytes in the Phb1-Ki mice compared with the wild-type mice. When challenged with experimentally-induced T1D and T2D, the Phb1-Ki mice displayed increased susceptibility to DKD, indicating the role of PHB1 in kidney biology and an effect of its dysregulation in the development of DKD. This prompted us to examine PHB1 levels in kidney tissues from the diabetes-linked mouse models of DKD. A significant decrease in their PHB1 levels was apparent indicating a potential link between them. Similarly, a decrease in PHB1 levels was found in human renal cells exposed to high glucose levels (mimicking diabetes-related hyperglycemia) indicating a direct effect of high glucose on PHB1 levels in them. The Phb1-Ki mouse models have created an opportunity to advance our understanding of PHB1's role in renal mitochondrial biology, including in sex differences, and of its dysregulation in new-onset and progressive DKD. Specially, the models provide insight into the role of PHB1's Cys69- and Tyr114-linked function in these processes, which are virtually unknown in current literature.

IL-2

UNDERSTANDING THE CONUNDRUM OF PRETERM BIRTH: CLUES FROM MULTIOMIC INVESTIGATIONS

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Preterm birth (PTB), being the leading cause of neonatal and infant mortality, imposes significant burden on the public health infrastructure. Incidence of preterm birth is the highest in South Asia and India accounts for 23.4% of global preterm births, being the largest contributor. However, no genomic studies on PTB have been reported yet either from India or from other South Asian countries.

We conducted a GWAS and a longitudinal EWAS in the GARBH-Ini (interdisciplinary Group for Advanced research on BirtH outcomes – DBT India Initiative), a prospective pregnancy cohort, to study multidimensional correlates of spontaneous PTB (sPTB) in India. We found SNPs and epigenomic alterations in preterm delivering women which might regulate specific biological pathways important in pregnancy. We also undertook single cell transcriptomic investigations of placentae from preterm and term deliveries and found involvement of specific cell types and cell states. Our findings provide novel insights which implicate molecular mechanisms involving placental perfusion and impaired response to hypoxia, dysregulation of immune responses as well as differential expression of specific alternate spliced transcripts in specific placental cell types in preterm delivery.

These findings are being used for developing predictive biomarkers of preterm delivery to triage women at risk, as well as formulate novel methods of intervention. Such efforts can help in reducing the occurrence of preterm birth and contribute to the enhancement of healthy births.

IL-3

OXPHOS SUSTAINS AND GLYCOLYSIS ACCELERATES SPERM MOTILITY

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Female-controlled non-hormonal contraception is dependent on the identification of new targets that can be safely targeted in the female reproductive tract. Sperm traverse a distance of about 1000 times in comparison to their own length and this journey is highly energy intensive. Therefore, targeting sperm energetics is an attractive choice for achieving non-hormonal contraception. While several knockout mouse experiments have suggested critical roles of glycolytic enzymes in fertility, species-specific differences in energy dependence of sperm necessitate the evaluation of human sperm energy pathways for contraception. In the present study, we evaluated the significance of glycolysis in human sperm motility and fertility by energy substrate depletion and specific inhibition of glycolysis and OXPHOS. Energy substrate depletion experiments revealed that access to internal glucose or other energy intracellular energy substrates was sufficient to maintain sperm motility for hours, but exogenous glucose (i.e., glycolysis) was a critical requirement for sperm hyperactivation. Similarly, inhibition of glucose access under normal physiological conditions using 2-DG showed almost complete loss of sperm hyperactivation, further suggesting the requirement of glucose for sperm hyperactivation. We interrogated the significance of OXPHOS using specific inhibitors (oligomycin, rotenone and antimycin A or FCCP), suggesting a significant contribution of OXPHOS to sperm motility and hyperactivation, but OXPHOS inhibition was unable to reduce hyperactivation drastically under physiological conditions. Multiple lines of experiments suggested a critical requirement of glycolysis for sperm hyperactivation and a supportive role of OXPHOS in sperm hyperactivation. Accordingly, inhibition of glycolysis but not that of OXPHOS showed significant reduction in sperm hyperactivation under physiological conditions, suggesting that glycolytic inhibition can be a promising approach to achieving non-hormonal contraception. "3D-Organoids as an emerging in-vitro experimental model for studying prostate cancer associated-bone metastasis: Interaction of cancer cells with pre-osteoblasts".

HIGH-FAT DIET ACTIVATES PRO-INFLAMMATORY RESPONSE IN THE MOUSE PROSTATE

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High-Fat Diet (HFD) has emerged as an important risk factor not only for obesity and diabetes but also for urological disorders. Recent research provides ample evidence that HFD is a putative cause for prostatic diseases including prostate cancer. The mechanisms whereby these diseases develop in the prostate have not been fully elucidated. In this study we discuss signaling pathways that intricately involved in HFD-induced prostate disease. HFD induces oxidative stress and inflammation in the prostate gland, and these adverse influences transform it from a normal to a diseased state. HFD also causes a significant increase in the levels of pro-inflammatory cytokines and gene products through activation of two important signaling pathways: the Signal Transducer and Activator of Transcription (STAT)-3 and Nuclear Factor-kappa B (NF-kB). Our studies demonstrate that HFD accelerates the generation of reactive oxygen species by driving the NADPH oxidase system, exacerbating oxidative stress in the prostate. Both these pathways function as transcription factors required for regulating genes involved in proliferation, survival, angiogenesis, invasion and inflammation. The crosstalk between these two pathways enhances their regulatory function.

C57BL/6 mice were either fed with regular diet (RD) or HFD for 4 and 8 weeks. Plasma cytokine levels were determined by multiplex analysis. Western blotting was performed to determine the expression of NF-kB, Stat-3, Akt, PDK1, PKCe, and their phosphorylated forms along with pathologic evaluation of the prostate. Immunoprecipitation and electrophoretic mobility shift assay (EMSA) were conducted to study the association between Stat-3 and NF-kB.

C57BL/6 mice fed with HFD showed a significant increase in the plasma levels of IL-1ß, IL-6, IL-17, and TNFa after 4 and 8 weeks of feeding, compared with RD controls. HFD feeding elevated the intraprostatic expression of IL-6 and caused activation of PKCe and Akt, the upstream kinase regulating Stat-3 and NF-kB. Nuclear extracts from the prostates of mice fed with HFD exhibited constitutively activated levels of Stat-3 and NF-kB/p65.

IL-5

MODULATING CHECKPOINT PATHWAYS TO PRESERVE OVARIAN RESERVE AND TO IMPEDE OVARIAN AGING

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The ovarian reserve, a finite pool of primordial oocytes established before birth, serves as the foundation of female fertility and reproductive longevity in both humans and livestock. Its progressive depletion drives ovarian aging and infertility, while also limiting breeding efficiency in high-value animal species. Beyond natural decline, external stressors such as chemotherapy and radiation pose severe threats to oocyte survival, leading to premature ovarian failure and irreversible infertility in young cancer survivors. At the heart of oocyte quality surveillance lies the p63-dependent DNA damage checkpoint, where the TAp63α isoform functions as a molecular sentinel, eliminating damaged oocytes to safeguard genomic fidelity across generations. However, excessive or prolonged activation of this pathway can trigger unwarranted oocyte loss, even in the absence of severe genotoxic insult. In this talk, I will present our recent work dissecting the molecular circuitry of p63 activation and its upstream regulators, CHK2, CK1, and associated cofactors, and how subtle modulation of these components can recalibrate the oocyte stress response. By fine-tuning this checkpoint pathway, we can protect oocyte viability without compromising genome integrity. This strategy holds promise not only for extending reproductive lifespan and fertility preservation in cancer survivors but also for enhancing reproductive efficiency in livestock, bridging fundamental biology with translational and agricultural impact.

IL-6

ROLE OF DIETARY QUALITY IN THE REGULATION OF GUT AND OVARIAN PHYSIOLOGY IN CARP

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The physiology of an organism is maintained by obtaining energy from the digestion and metabolism of food in the digestive tract. When the food is available, at what frequency does food become available per day, whether the ad libitum quantity of food has been received by the digestive tract, and, more importantly, what about the quality of the food entered within the gut? All these questions are very important from the point of view of proper nutrition and a healthy lifestyle. Complex food needs various enzymes for its proper digestion. Metabolism and energy production from the food material generate free radicals, which are neutralized by internal antioxidants. Proper nutrition, efficient digestive physiology, effective management of oxidative stress, and reproductive health are crucial for sustaining physiological homeostasis. Moreover, somatic growth, reproduction, and overall health are essential for revenue generation since fish hold significant economic value. This presentation has revealed two crucial insights in the field of nutrition and reproduction: (1) digestion and stress management in the gut, and (2) reproductive physiology, focusing on the nutritional aspect of fish feeds.

In the past few years, our lab has investigated age-related alterations in gut histo-architecture, gastro-somatic index (GaSI), feeding intensity, and gut melatonin levels from the fingerling to adult stages of carp, Catla catla. However, in this context, the functions of gut melatonin, which depend on the availability of food, the timing of food supply, the frequency of feeds/day, the quality of food, and the growth stages of carp, still need to be clarified. Our recent studies demonstrated a significant relation among the gut tissue architecture, gut melatonin levels and feeding characteristics in relation to gut and ovarian physiology. For example, the development of the gut, particularly by the measurement of gut tissue architecture, demonstrated an inverse correlation with gut melatonin content. However, gut melatonin may play a significant role in protein and microbial digestion in the mature gut by reducing gut oxidative stress. Although, the mechanism still needs to be investigated. But nonetheless, the alteration of the dietary protein and tryptophan percentage of fish feed, which caused an increase in gut melatonin levels, exhibited a significant impact on the growth performance, stress management, and digestive physiology of fish. More interestingly, when gut melatonin was increased by dietary modulation, a parallel increase in serum melatonin and ovarian melatonin was observed. Under such a melatonin stimulatory diet plan, at least the juvenile carp to date has shown pro-gonadal effects by increasing Stage-I oocytes abundance and by reducing oxidative stress in the developing ovary. This opens avenues for future research on the role of feed-induced gut melatonin in fish nutrition and reproduction.

IL-7

DNA BREAK MAPPING IN PLACENTAL CELLS UNCOVERS VULNERABILITY OF CGG REPETITIVE SEQUENCES IN PREGNANCY COMPLICATION

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During placental development, trophoblast cells increase their ploidy by going through many rounds of replications either by endocycle or syncytium formation. During this process, they accumulate DNA damage, likely due to replication error from the dampening of the ATR (ATM and RAD3-related) DNA damage response pathway, and as gestation progresses, there is a gradual physiological increase in DNA damage. To investigate the physiological role of DNA damage, we mapped damage sites using □H2A.X chromatin immuno precipitation (ChIP) in mouse parietal trophoblast giant cells (P-TGCs) at 9.5dpc stage of pregnancy and from human term placental cells. We were able to identify 51 reproducible DNA damage sites in P-TGCs. Since repetitive DNA sequences are more prone to DNA damage, we also analysed several repetitive sequences. Our finding suggest that placental cells show more breaks in CGG repeats as compared to other repeats. We hypothesize that CGG repeats accumulate DNA damage during pregnancy and may be more prone to breaks in various pathophysiological conditions such as fetal growth restriction. Using invitro human trophoblast stem cell culture model and blood samples from different stages of human pregnancies, we showed that flanking sequences corresponding to CGG repeats can be detected as cell free DNA (cfDNA) and the amount of these repeats in cfDNA increases in the case of fetal growth restriction. We propose that detection of these repeats in mother's bloods as cfDNA can be used as non-invasive prenatal marker for early placental stress and pregnancy complication.

IL-8

DEVELOPMENTAL EXPOSURE TO PHTHALATES ADVERSELY AFFECTS METABOLIC OUTCOMES IN MALE OFFSPRING

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Global rise in obesity to epidemic levels indicates that it is a far more complex issue than a simple imbalance between calorie intake and expenditure. One emerging explanation is the chemical toxin hypothesis, which posits that obesity may be a consequence of exposure to industrial and consumer-use chemicals. These synthetic environmental pollutants can disrupt normal endocrine signaling, thereby interfering with hormonal regulation of metabolism and promoting the development of obesity. Phthalates, a group of such chemicals, are widespread contaminants to which humans are routinely exposed via inhalation, ingestion, injection, or skin contact. While their reproductive toxicity has been extensively studied, much less is known about their role in metabolic dysfunction. This study investigates the impact of perinatal exposure to phthalates on adipose tissue development and metabolic regulation in later life, using the C57BL/6 mouse model. Pregnant mice were exposed to a mixture of low and high molecular weight phthalates. The F1 offspring were analyzed at various developmental stages. Although no significant changes in plasma glucose or lipid profiles were detected up to 8 weeks of age, by 16 weeks, notable metabolic alterations emerged. These included increased body weight, larger adipocyte size, greater fat accumulation, elevated food intake, impaired insulin sensitivity, and altered adipose tissue function in phthalate-exposed progeny. The findings indicate that phthalate exposure during critical developmental windows can have lasting, transgenerational effects, predisposing offspring to metabolic disorders. This supports the hypothesis that environmental chemical exposure in early life may contribute to the growing prevalence of obesity in the population.

INTERGENERATIONAL EFFECTS OF PRENATAL FOLATE AND B12 IMBALANCE: AN EPIGENETIC PERSPECTIVE

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Mandatory supplementation of folic acid and a lack of guidelines to supplement vitamin B12 during pregnancy might create an imbalance of vitamin B12, and folic acid is likely to predispose offspring to poor growth and development. We aimed to investigate the impact of maternal folic acid and vitamin B12 imbalances during pregnancy on newborn outcomes. Blood and placental samples were collected from pregnant women to estimate RBC folate, serum vitamin B12, and homocysteine levels. Newborn anthropometric parameters were recorded. Based on the levels of folate and vitamin B12, pregnant women were stratified into different groups. All the anthropometric parameters of the newborn and placental weight were reduced in the groups of women with an altered ratio of the two vitamins. Furthermore, in a separate study using an animal model, the transgenerational effects of dietary manipulations were examined through epigenetic alterations. C57BL/6 mice (F0) were given different dietary combinations of folic acid and low vitamin B12, and the animals were mated. F1 pups were either continued on the same diet (sustained group) or shifted to a normal diet (transient group) for 6-8 weeks. Results revealed that a dietary imbalance of folic acid and vitamin B12 altered global methylation, as well as gene-specific methylation and histone methylation, in placental tissues. Hence, exposure to an imbalanced diet low in vitamin B12 and folic acid can lead to alterations in the establishment of epigenetic marks, which cannot be restored even by shifting the postnatal diet to a balanced one. The study forms the basis of keeping balance in the levels of folate and vitamin B12 during gestation in pregnant women.

IL-10

ENVIRONMENT, EPIGENETICS AND EMBRYOGENESIS: MATERNAL-CHILD HEALTH IN THE FACE OF CLIMATE CHANGE

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Recent studies highlight the critical role of epigenetic regulation in early embryogenesis and fetal development. However, the epigenetic machinery is highly sensitive to environmental factors, making it vulnerable to stress-induced perturbations that can lead to developmental defects. In recent years, many regions—including India—have experienced increasingly severe heat waves combined with high humidity during prolonged summers. Emerging evidence suggests that maternal heat exposure during pregnancy is associated with adverse outcomes such as preeclampsia, low birth weight, fetal growth restriction, stillbirth, and pregnancy loss. Despite these alarming trends, the molecular mechanisms underlying susceptibility to heat humidity stress remain poorly understood, and no protective strategies currently exist. In this talk, I will present findings from a mouse model of gestational heat-humidity stress, demonstrating how such environmental conditions influence the epigenome and alter fetal placental gene expression leading to embryonic defect and lethality.

Note: I used Copilot to make minimal improvements to the text and correct grammatical errors

IL-11

INFLAMMATION-DRIVEN PROTEIN KINASES SIGNALING ASSOCIATION WITH OVULATION DURING ENDOMETRIOSIS

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Ovarian folliculogenesis is affected by endometriosis, which is a benign endocrine disorder in 10% reproductive-age women. Endocrine-disrupting agents can also contribute to it. Endometriosis is characterized by hyperestrogen and progesterone resistance at the endocrine level. It is symptomatically represented by pelvic pain and inflammatory responses. The person will have endometrial tissue ectopically, as discharge like the menstrual fluid, which gives rise to inflammation, pelvic pain, infertility, etc. The inflammatory chemokine MCP-1 has been shown to increase in both systemic and local circulation, which promotes the development of endometriosis. Hence, we explored whether the systemic circulatory cytokines exert an effect on endometriotic tissue through various protein kinases, integrin-linked kinases (ILK), MAPK, etc., and alter the endometriotic cell behavior. We utilized human endometriotic cells, human tissue, and a mouse model of endometriosis. We found increased inflammation in response to MCP-1, which seems to act through ILK. MCP-1 promoted the aggregation, colonization, and adhesion of human endometriotic cells. The anti-inflammatory response was also downregulated in systemic and local environments. ILK and MAPK are established signaling cascades, and we found EKR3 associated with endometriosis and affected by the ovulatory event during endometriosis. At the preclinical level, ILK modulation decreased the lesion development and local estradiol production, increased the anti-inflammatory response, and suppressed the inflammation. In conclusion, we report that ILK is the target of MCP-1, and targeting ILK ameliorates endometriosis at the preclinical level.

(Life Sciences 2024 and Molecular Human Reproduction 2025).

IL-12

A CASE FOR MATERNAL SCREENING AND INTERVENTION TO PREVENT CONGENITAL CMV INFECTION (cCMV)

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As the largest world-wide cause of congenital infections HCMV presents a significant challenge to maternal and child health in India that bears amongst the largest burdens of cCMV, no national screening program and yet, ironically, the highest seroprevalence in women of reproductive age. Our efforts delineating immune correlates of protection against congenital infection with associated adverse pregnancy outcomes together with an integrated maternal screening approach will be presented to advocate for maternal screening and intervention to prevent cCMV in India.

IL-13

DISRUPTOR OF GSK3A: A NON-HORMONAL CONTRACEPTIVE THAT TARGETS MATURING SPERMATOZOA IN EPIDIDYMIS

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This study examined whether selective inhibition of glycogen synthase kinase 3 alpha (GSK3α) using the paralog-specific inhibitor BRD0705 could serve as a reversible and non-hormonal male contraceptive by impairing sperm maturation and motility. GSK3α, unlike its paralog GSK3β, plays a critical role in sperm function, as shown in Gsk3a knockout mice that are infertile due to impaired sperm maturation. Male contraceptive options are currently limited, making paralog-selective inhibitors a promising strategy largely free from side effects. In this study, C57BL/6 male mice were treated with BRD0705 (20 mg/kg i.p. for one week). Fertility testing was performed by mating treated and control males with females, while sperm motility, morphology, ATP levels, and biochemical markers were assessed. Treatment led to complete infertility in male mice, with no litters from the females immediately mated after the treatment period. This was associated with reduced sperm motility, midpiece abnormalities, decreased ATP levels, and reduced expression of Hexokinase 1, indicating impaired glycolysis. Western blotting confirmed reduced Tyr279 phosphorylation of GSK3α, demonstrating effective targeting. Fertility was fully restored within eight weeks of stopping treatment, with treated mice siring nearly as many pups as controls, and sperm parameters returned to normal. These effects resembled the phenotypes of Gsk3a knockout mice, confirming the specificity of GSK3α inhibition. These results show that BRD0705 induces temporary, reversible infertility without adverse effects. The findings not only highlight GSK3α as a novel male contraceptive target but also provide new insights into sperm energy metabolism, with implications for both contraception and male infertility therapies.

IL-14

L-NAME INDUCES SPERMIATION FAILURE AND TRANSGENERATIONAL SPERM DEFECTS IN WISTAR RAT

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Spermatogenesis in rats is a complex, multi-stage process culminating in the release of mature spermatids from Sertoli cells (SCs) into the seminiferous tubular lumen via spermiation. This final step is critically dependent on the integrity of Sertoli-spermatid junctions, particularly ectoplasmic specializations (ES) and tubulobulbar complexes (TBCs). In our study, male Wistar rats were administered NG-nitro-L-arginine methyl ester (L-NAME), a nitric oxide synthase (NOS) inhibitor, at 10 and 20 mg/kg body weight for three weeks. Persistent hypertension, reduced sperm count, abnormal sperm morphology, and impaired spermatogenesis were observed even two months' post L-NAME treatment. Histological analysis revealed an increased number of lagging step 19 spermatids in stage VIII and retained spermatids in stages IX–XI, indicating spermiation failure. Additionally, elevated intra-testicular testosterone levels were noted. Notably, reduced sperm counts were also observed in F1 and F2 male progeny, suggesting transgenerational effects. These findings highlight the critical, though indirect, role of the NO-pathway in maintaining Sertoli-germ cell junction integrity and facilitating successful spermiation. Our results underscore the vulnerability of the spermiation process to NO deficiency and its potential long-term impact on male fertility across generations.

IL-15

3D-ORGANOIDS AS AN EMERGING IN-VITRO EXPERIMENTAL MODEL FOR STUDYING PROSTATE CANCER ASSOCIATED-BONE METASTASIS: INTERACTION OF CANCER CELLS WITH PRE-OSTEOBLASTS

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Prostate cancer (PCa) is the second most commonly diagnosed malignancy in men and the fifth leading cause of cancer –related mortality worldwide. Bone metastases are the primary causes of mortaility and has been considered a significant determinant of both patient survival and quality of life. PCa-bone metastasis (PCa-BM) presents significant clinical challenges, majorly due to the formation of mostly osteoblastic & rarely osteolytic/mixed lesions. However, the factors contributing to the incidence of bone metastasis is challenging in vivo, due to lack of PCa-BM models and clinical heterogeneity of human PCa. Hence, there is an urgent need for establishment of the reliable pre-clinical PCa-BM models to study the progression and severity of this disease. Therefore, we aim to establish a PCa-BM models to study the interaction of PCa cells and pre-osteoblasts. We successfully established the pre-clinical models, both in vitro- 3D-PCa organoids and in-vivo models: intra-cardiac and intra-tibial BM mouse models to study PCa-BM. We showed (1) these PCa organoids exhibited osteomimicry and osteoblastic phenotype. (2) the PCa proliferation, extent of metastasis are closely associated with the interaction and crosstalk of the factors derived from cancer cells and bone cells in bone microenvironment. Hence, PCa organoids models are a powerful pre-clinical in vitro tools, in addition to in vivo models for in-depth exploration of the PCa-BM. Mostimportantly, 3D-PCa-Organoid model could be used as a promising approach prior to in-vivo studies, for addressing clinical challenges, testing anti-cancer drug sensitivity-screening, the development of potential biomarkers and novel targeted therapeutic strategies against PCa-BM.

IL-17

GROWTH HORMONE'S ROLE IN DIABETIC KIDNEY DISEASE: A NEW ROLE FOR AN OLD HORMONE

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Diabetes is a significant risk factor for the development of diabetic kidney disease, and about 30% of people with type I diabetes mellitus will eventually develop end-stage kidney disease. Growth hormone (GH) and its mediator insulin-like growth factor (IGF-I) are crucial for kidney development and function in healthy conditions. However, elevated circulatory levels of GH in type I diabetes mellitus disrupt homeostasis and cause changes in the kidney's structure and function, such as hypertrophy, glomerulosclerosis, and proteinuria. Glomerular podocytes are specialized cells in the nephron, and they practically represent the kidney's filtration function. Podocytes are terminally differentiated cells, and podocyte injury or loss causes significant damage to the glomerulus, manifested by varying degrees of proteinuria. Recent studies have identified that podocytes express GH receptors and are key targets of GH action, particularly in settings with type 1 diabetes mellitus. Our lab (www.nephlab.com) demonstrated that GH evokes the reactivation of the embryologically active Notch signaling in adult podocytes. Aberrant activation of embryologically active Notch signaling in quiescent adult podocytes ensures cell-cycle reentry, but results in aberrations in cytokinesis and consequent mitosis-associated cell death.

IL-18

CROSS-SPECIES TRANSCRIPTOMIC INSIGHTS INTO THE INTERPLAY BETWEEN ADIPOGENESIS AND ANGIOGENESIS

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Obesity is characterized not only by excessive adipose tissue expansion but also by concurrent vascular remodeling, yet the molecular coordination between adipogenesis and angiogenesis remains incompletely understood. Herein, we integrated bulk RNA-seq datasets capturing the differentiation of human mesenchymal stem cells (GSE136230), murine 3T3-L1 preadipocytes (GSE129957), and mouse embryonic fibroblasts (GSE152750) into adipocytes to investigate conserved transcriptional programs linking these processes. Data preprocessing involved quality control with FastQC, trimming with Trimmomatic, and transcript abundance estimation using Salmon, followed by differential expression analysis in DESeq2. Functional enrichment with Cluster Profiler and protein–protein interaction network construction via Metascape identified biological processes enriched in angiogenesis across both preadipocyte and mature adipocyte stages. We observed that key pro-angiogenic genes (e.g., VEGFD, ANGPT1/2, IGF1/2, PDGFD, MMP family) were upregulated from early differentiation stages, while select anti-angiogenic factors (e.g., IL12, IFNA, COL4A2, TIMP2) were consistently downregulated. PCA and heatmap analyses revealed distinct temporal gene expression patterns with strong clustering by differentiation stage and conservation between human and mouse models. Protein–protein interaction mapping highlighted the hub nodes potentially regulating vascular remodelling during adipocyte maturation. According to our research, adipocytes have molecular markers that are consistent across species and actively support angiogenesis throughout development. In addition to identifying potential regulators that could guide treatment approaches for obesity and metabolic diseases, this integrated transcriptome approach offers mechanistic insight into the vascularization of adipose tissue. These findings provide opportunities for targeted modification of the adipose tissue vasculature in disease contexts and demonstrate the usefulness of cross-species omics study in deciphering intricate tissue relationships.

IL-19

CAN EARLY PREGNANCY MICROBIOME–IMMUNE SIGNATURES PREDICT THE RISK OF ADVERSE OUTCOMES?

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Healthy pregnancy requires synchronized adaptations in the maternal gut microbiome and immune system. Early disruptions to this balance may increase the risk of adverse pregnancy outcomes (APOs).

Healthy primigravida women attending the antenatal clinic at N. Wadia Maternity Hospital, Mumbai, were recruited in the first trimester and followed longitudinally through the third trimester. Paired stool and blood samples were obtained from the recruited participants following informed consent. Gut microbiome composition was characterized using 16S rRNA sequencing (Oxford Nanopore Technology). Innate and adaptive immune cell subsets were quantified by multi-parametric flow cytometry. Microbiota–immune associations were assessed via correlation analyses. Functional assays evaluated the ability of stool-derived metabolites to modulate THP-1 cell differentiation in vitro.

Healthy pregnancies demonstrated trimester-specific microbiome remodelling accompanied by balanced immune regulation and coordinated microbiota–immune interactions. A small subset of these women later experienced APOs. Analysis of their first-trimester samples revealed early gut microbial dysbiosis, altered monocyte/NK/Treg frequencies, and disrupted microbiota–immune correlations. Fecal supernatants containing gut derived metabolites from these pregnant women showed altered immune cell differentiation potential in THP-1 assays, indicating disrupted functional immune modulation.

This longitudinal analysis establishes a baseline understanding of dynamic microbiome–immune adaptations during healthy pregnancy. Preliminary findings from a small subset of healthy women who developed APOs suggest that early gut microbiome–immune perturbations may serve as risk indicators for future adverse outcomes. Larger studies are warranted to validate these trends and support development of early screening strategies for maternal–fetal health.

IL-20

SEASONAL BREEDING AS THE "NATURE'S CONTRACEPTIVE"? THE STORY FROM SONGBIRDS

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Seasonal breeding is described as naturally occurring mechanisms that lead to a recurring reversible pattern of fertility and infertility in adult animals. In fact, the ability of regulatory system underlying reproductive processes is turned 'on' and 'off' at precise times year after year. This confers distinct selective advantage, by enabling species to reproduce during the time of the year when the conditions in surrounding environment favor the survival of offspring borne. Over the years, our laboratory has been interested in identifying putative mechanisms underlying seasonal variations in reproductive functions using both non-migratory and migratory songbirds as the experimental systems. In this lecture, I propose to discuss briefly the interface of environment-clock-hypothalamic gene switches involved in the transition between non-breeding and breeding periods in seasonally breeding songbirds, with particular reference to autumn breeder spotted munia (*Lonchura punctulata*) and migratory buntings (*Emberiza* sp.).

IL-21

MICROFLUIDIC CHIP-BASED SYSTEM FOR SUBTYPE-SPECIFIC DETECTION OF CIRCULATING TUMOUR CELLS IN BREAST CANCER

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Breast Cancer – a heterogenous disease is the reason for cancer-related deaths in women worldwide. Monitoring of the disease and coming up with new treatment and early detection strategies remains a challenge in the field of oncology. Circulating Tumour Cells (CTCs), shed from primary tumour into the blood circulation is an upcoming field of interest that offers a non-invasive alternative to tissue biopsies for disease diagnosis and prognosis. CTCs serve as a promising biomarker for early detection, disease monitoring, and therapeutic response.

The challenge involved with CTC is the isolation and capture owing to the rarity. We hypothesised that integrating nanoparticle – assisted capture with microfluidic technology can enhance CTC isolation efficiency.

Our study aims to develop a microfluidic lab-on-chip based platform integrated with nanoparticle assisted magnetic capture of CTCs from breast cancer patients and the subsequent subtype classification with the help of conjugated carbon dots.

Objectives are: To synthesise and characterise chitosan-folic acid-iron oxide nanoparticles for the magnetic capture of CTCs. To design a three layered PMMA microfluidic chip for isolation of the CTCs. To develop aptamer/antibody conjugated carbon dots for the subtype classification of breast cancer and to finally validate the developed chip, nanoparticle and carbon dots using cell line capture and uptake studies followed by real time validation.

Chitosan – Folic acid – Iron Oxide (CFI) nanoparticles were synthesised by co-precipitation method which uses TPP as a cross linker via EDC-NHS chemistry for the functionalisation of folic acid. A three-layered PMMA microfluidic chip was designed to magnetically capture the CFI bound CTCs. Luminescent carbon dots were synthesised and conjugated with aptamers using EDC/NHS chemistry and was employed for the subtype classification of breast cancer.

Chitosan-Folic Acid-Iron Oxide nanoparticles were successfully synthesised and characterised and the efficiency of CTC capture was assessed using breast cancer cell lines. Similarly, the carbon dots have been synthesised and characterised and has been conjugated with specific antibodies for the classification of the breast cancer subtypes. The chip designed has been validated for the flow and capture using breast cancer cell line spiking studies.

Cancer, Circulating tumour cells, Microfluidic, Nanotechnology, Breast Cancer

IL-22

CHRONIC UNPREDICTABLE STRESS INDUCES MALE SEXUAL DYSFUNCTION: A COMPLEX INTERPLAY OF NEUROTRANSMITTERS AND ERECTION PATHWAY

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Sexual arousal and potency are key determinants of male sexual health, a significant concern affecting an estimated 322 million men worldwide with some form of sexual dysfunction, much of which is related to daily life stress. The relationship between psychological stress and male sexual dysfunction is complex and multifactorial, reflecting the dynamic nature of stress. This study aimed to investigate the effects of chronic unpredictable stress (CUS) on the molecular mechanisms governing erection physiology and its association with neurotransmitters involved in sexual potency and behavior. Adult male Parkes mice were randomly divided into control and CUS-exposed groups, with the latter exposed to CUS for 35 days. Behavioral analysis showed that CUS exposure induces anhedonia and depressive behavior. CUS negatively affected neurotransmitters dopamine and glutamic acid, leading to altered pro-sexual behavior demonstrated by reduced non-contact erections and disrupted sexual behavior, culminating in decreased frequency of mount, intromission, and ejaculation. It also prolonged sexual exhaustion by increasing the latencies of these behaviors. Additionally, CUS elevated corticosterone levels while decreasing circulating gonadotropins and testosterone. CUS induced endothelial dysfunction, as evidenced by altered penile histomorphology with a decreased smooth muscle/collagen ratio. Exposure to CUS adversely affected NO availability for penile erection, by decreasing the neurotransmitter Ach and other erection facilitatory markers such as p-Akt, nNOS, eNOS, and cGMP, while increasing the inhibitory marker PDE5α. In conclusion, CUS disrupts erectile function and sexual behavior via neurotransmitter dysregulation and Akt/NO-cGMP/PDE5α signaling pathways.

IL-23

HMGB1-RAGE AXIS AND THE DYNAMICS OF IMMUNE CELLS DURING ENDOMETRIAL REPAIR IN RAT MODEL OF INDUCE MENSTRUATION

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The human endometrium undergoes cyclical breakdown and repair during menstruation, indicating a regulated balance between inflammation and tissue regeneration. This process is majorly coordinated by immune cells such as neutrophils, macrophages, and uterine natural killer (uNK) cells, which mediate tissue shedding, debris clearance, and repair. High mobility group box 1 (HMGB1), released from stressed or necrotic cells, signals through the receptor for advanced glycation end products (RAGE) to modulate immune responses via regulating leukocyte activity and cytokine release in various tissues. The current study was undertaken to explore whether HMGB1 plays a role in endometrial repair. Toward this, a rat model of induced menstruation (RMIM) was developed and characterized. The model demonstrated complete endometrial breakdown at 24h and complete endometrial repair at 48h, post-progesterone withdrawal. Immunofluorescence was performed to evaluate the relative presence of HMGB1, RAGE,kappa-B (NF-κB), vascular endothelial growth factor (VEGF) staining, and recruitment of macrophages in the endometrium. An increase in HMGB1 and RAGE expression in the endometrium and secretion of HMGB1 in uterine fluid was observed, at 8 h post-progesterone withdrawal (T8). Increased expression of NF-κB peaked at T24 with increased frequency of immune cells neutrophils, microphages and uNK cell. Inhibiting the HMGB1/RAGE axis at T0 and T24 delayed the endometrial repair at T48 post progesterone withdrwal. Moreover, inhibiting the axis at T24 completely impeded endometrial re-epithelialization in at T48. HMGB1/RAGE axis inhibition reduced the staining intensities of NF-κB and VEGF and reduced recruitment of immune cells in endometrium. This study provides initial evidence that extracellular HMGB1 released by degrading endometrium plays a role in endometrial repair via RAGE by regulated recruitment of immune cells.

IL-24

INVESTIGATING THE ETIOLOGY OF OSTEOARTHRITIS AND THE IMPACT OF NUTRACEUTICALS ON THE ONSET AND PROGRESSION OF OSTEOARTHRITIS

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Osteoarthritis (OA) is the most prevalent diseases associated with ageing, resulting in joint stiffness, pain and socioeconomical burden. The pathophysiological features of OA include inflammation, articular cartilage degradation, subchondral bone remodelling and osteophyte formation. During the progression of the disease, the cellular and molecular changes follows developmental process of bone formation i.e. endochondral ossification, where the bone morphogenetic proteins (BMPs) and synovial inflammation are thought to participate in a central role. Therefore, our main aim is to investigate the hierarchy and the interplay between these two intriguing molecular events during OA. As an extension of the study, we formulated a nutraceutical including four potential Ayurvedic herbs (named nutraceutical) and supplemented it to papain induced OA mice model to counter the pathological changes during the disease. To observe the role of BMP signalling and macrophage mediated inflammation we performed Anterior Cruciate Ligament transection (ACLT) in C57/BL6 mice line and used the conditional gain-of-function mouse line in which BMP signalling can be activated. We also used clodronate laden liposomes to deplete macrophages from the intra-articular joint to examine the impact of macrophage mediated inflammation upon BMP signalling activation. We examined the expression of endochondral ossification markers (pSMAD1/5/8, Ihh, ColX) and inflammatory markers (NF-κβ, IL-1β, iNOS) following ectopic BMP activation as well as post scavenging synovial macrophages. Our results showed ACLT induced mechanical insult activated the BMP signalling in the articular cartilage and significantly increased the expression of pSMAD1/5/8, Ihh, ColX, MMP13 and pro-inflammatory indicators which was reverted following depletion of synovial macrophages. Moreover, our data revealed that the expression of the endochondral ossification indicators were regulated by the polarization state (M1 or M2) of synovial macrophages. Interestingly, the supplementation of nutraceutical significantly reduced the expression of inflammatory mediators and matrix proteases in the articular cartilage and halted the progression of OA. Our findings suggested that ectopic BMP signalling and inflammatory mediators alone can induce the osteoarthritic like changes but synergistically accelerate the progression of OA.

MATERNAL EXPOSURE TO NONYLPHENOL (NP) INDUCES OVARIAN DYSFUNCTION AND TRANSGENERATIONAL DEVELOPMENTAL IMPAIRMENTS IN ZEBRAFISH (*DANIO RERIO*)

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Nonylphenol (NP), a pervasive endocrine-disrupting chemical, is environmentally persistent and bioaccumulative, raising significant concerns regarding reproductive toxicity and transgenerational health risks. Here, we investigated the mechanistic effects of maternal NP exposure (50 and 100 µg/L; 28 days) on ovarian function, fecundity, and developmental programming in zebrafish (*Danio rerio*), emphasizing F1 offspring. NP accumulated preferentially in ovaries, inducing oxidative stress, MAPK (ERK1/2, JNK, p38) activation, AP-1 induction, and pro-inflammatory cytokine upregulation, triggering ovarian inflammation and apoptosis through Bax/Bcl2 imbalance, Caspase-3 activation, and PARP1 cleavage. Disruption of estrogen receptor (ER) signalling (enhanced ERα, suppressed ERβ), gonadotropin receptor transcripts, and steroidogenic markers (StAR, P450 aromatase, igf3) revealed compromised ovarian microenvironment, consistent with impaired hCG-induced oocyte maturation and reduced fecundity. In unexposed offspring, maternal NP exposure led to delayed embryogenesis, increased malformations (yolk sac and pericardial edema, axial curvature, swim bladder defects), reduced survival, diminished hatching rates, and bradycardia. Molecular analyses in larvae demonstrated exacerbated oxidative stress, suppressed immune response (reduced macrophages/neutrophils population, downregulated TLR4/NF-κB pathway), apoptotic activation (p53, Caspases-3/8/9), and epigenetic alterations in transcript abundance of DNA methylation and histone modification enzymes, suggesting persistent transcriptional reprogramming. Furthermore, NP disrupted energy metabolism by impairing yolk lipid mobilization (mttp1, apoa1a, scarb1), attenuating fatty acid β-oxidation (acox1, cpt1), while downregulating PPARα, PGC-1α, SIRT1, and phosphorylated AMPKα. Mitochondrial dysfunction, reflected in reduced ETC subunits (complex I-V) and decreased membrane potential, underscored energy deficits in F1 larvae. Collectively, our results, demonstrating maternal health as a cornerstone of offspring development, reveal how endocrine disruptors pose long-lasting generational risks. Acknowledgement: Financial assistance from Anusandhan National Research Foundation (ANRF)-sponsored research project (Grant No. CRG/2023/002389) to SM is gratefully acknowledged.

IL-26

MOLECULAR UNDERPINNINGS GOVERNING GENETIC COMPLEXITY OF THE PROSTATE CANCER AND PATHWAYS TO TARGETED THERAPY

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Molecular heterogeneity in prostate cancer (PCa) drives highly variable clinical outcomes and remains a formidable obstacle in effective disease management. Identifying robust molecular signatures to classify distinct cancer subtypes and unravel their underlying mechanisms is therefore pivotal for advancing precision medicine and designing more effective therapeutic strategies. Approximately 50% of primary PCa harbor gene fusions involving members of the ETS transcription factor family, while another key subclass (~10-15%) exhibits higher levels of SPINK1, and associates with aggressive disease. In my talk, I will discuss the role of miRNAs in the post-transcriptional regulation of SPINK1, and how epigenetic drugs or synthetic mimics could restore the expression of these miRNAs, resulting in reduced SPINK1-mediated tumorigenesis. For locally advanced and metastatic PCa patients, androgen deprivation therapy (ADT) remains the mainstay of treatment. However, these patients eventually progress to the castration-resistant stage, and a subset often develops ADT-induced neuroendocrine PCa. I will provide insight into the Androgen Receptor (AR)-SPINK1 axis and its connection with neuroendocrine prostate cancer. I will also discuss the oncogenic role of Distal-less homeobox-1 (DLX1), present in ~60% of PCa patients, and the underlying mechanism involving ERG/AR as its key transcriptional regulators. I will also discuss how bromodomain and extra-terminal (BET) protein inhibitors and/or anti-androgen drugs could disrupt the ERG/AR-mediated DLX1 transcription, and lead to reduced DLX1 expression and downstream oncogenic effects. Overall, my talk will focus on how our findings led to the identification of actionable genetic alterations or molecular pathways that paved the way for evidence-based therapeutic interventions.

IL-27

FERTILITY PRESERVATION IN ONCOLOGICAL PATIENTS: CHALLENGES AND OPPORTUNITIES

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Advances in cancer therapy have significantly improved survival, however gonadotoxic treatments continue to threaten the reproductive potential of children and young adults. Fertility preservation (FP) has thus emerged as a critical component of comprehensive cancer care, though its implementation in developing countries remains limited. Our studies highlight both biological and systemic challenges in oncofertility practice and identify opportunities for progress. Investigations on immature testicular tissue (ITT) from prepubertal boys revealed that spermatogonial quantity, a determinant of fertility restoration potential, varies across malignancies but remains generally preserved prior to cryopreservation, supporting the feasibility of paediatric male FP programs. Complementary experimental work using mouse and human models demonstrated that ultraprofound-hypothermic storage preserves ITT viability and endocrine function, offering translational insights into optimizing cryopreservation protocols. Surveys among oncologists, gynaecologists, and primary care physicians across India underscored limited awareness and knowledge gaps in FP referral pathways, compounded by financial and infrastructural constraints. Global surveys through the Oncofertility Consortium further highlighted similar barriers across low-resource settings but also showcased innovative local adaptations and growing professional networks. Collectively, these findings underscore the urgent need for multidisciplinary collaboration, policy inclusion, and education to ensure equitable access to FP services. Emerging biotechnologies, dynamic culture systems, and global engagement platforms present unique opportunities to translate laboratory advances into real-world fertility preservation solutions for cancer patients.

ORAL PRESENTATIONS: COMPETITIVE

OP-C-1	**Ahmed Saleel** *Central University of Kerala* **Evaluation of Sex Specific transcriptomic Profile During Gonadal Development in the Asian Seabass (Lates calcarifer , Bloch 1790)**
OP-C-2	**Achyutham Hotha** *Karnatak University, Dharwad* **Role of GABA in Stress-Induced Suppression of Reproduction in Female *Poecilia Sphenops***
OP-C-3	**Anushruti Singh** *ICMR-NIRRCH, Mumbai* **Impact of Maternal Methyl Donor Deficiency During Gestation on fertility and Germ Cells' DNA Methylation in Mouse Offspring**
OP-C-4	**Piyali Mondal** *BRIC-NIMB, Kalyani West Bengal* **Gene expression programs and alternative splicing in placental cell states reveal novel underpinnings of preterm birth**
OP-C-5	**Jyoti Batgire** *ICMR-NIRRCH, Mumbai* **Gut microbiome profiles associated with HCMV in pregnancies complicated by BOH**
OP-C-6	**Harsha Chandrashekhar Palav** *ICMR-NIRRCH, Mumbai* **Early maternal immune signatures of HCMV infection and its association with pregnancy outcomes and cCMV: a prospective study**
OP-C-7	**Hetvi Shah** *Navrachana University, Vadodara, Gujarat* **Kisspeptin-10–Induced KISS1 Activation Orchestrates Comprehensive Anti-Tumor, Anti-Metastatic, and Pro-Apoptotic Reprogramming in Triple-Negative Breast Cancer Cells**
OP-C-8	**Meghali Borkotoky** *ICMR-NIRRCH, Mumbai* **Proteomic Analysis of 2D versus 3D Grown Ovarian Cancer Cells : Insights into Ferroptosis Regulation**

OP-C-9	**Kshitija Rahate** *ICMR-NIRRCH, Mumbai* **Multi-omics profiling reveals metabolic rewiring as central to interactions between human vaginal cells and Candida albicans**
OP-C-10	**Pratik Devadiga** *ICMR-NIRRCH, Mumbai* **Predictive functional profiling reveals gut microbial metabolic shifts linked to immune activation in HIV infection**
OP-C-11	**Anupama Hoory** *Visva Bharati (A Central University)* **Altered Myokine Profile during Progression of Obesity-Mediated Type 2 Diabetes in Male Mice**
OP-C-12	**Sambuddha Banerjee** *Visva-Bharati University, Santiniketan, West Bengal* **The Zebrafish Ovulatory Brain: Mechanistic Insights into the Neuroendocrine and Metabolic Cascades Governing Reproduction**
OP-C-13	**Debankur Pal** *CSIR-IICB, Kolkata* **OSR1 Orchestrates and Limits Cardiac Fibrosis and Hypertrophy**
OP-C-14	**Akanksha Pranoty** *University of Hyderabad* **Elucidating the Role of FGF2 in Fish Testicular Steroidogenesis**
OP-C-15	**Rhydham Karnik** *Dr. Vikram Sarabhai Institute of Cell and Molecular Biology, Vadodara* **Bmal1 regulates monocyte trafficking in zeitgeber disrupted C57BL/6J mice**
OP-C-16	**Sujata Mishra** *University of Hyderabad* **Transgenerational reproductive toxicity of the histone deacetylase inhibitors, valproic acid and sodium butyrate in the male rats**
OP-C-17	**Aalaap Naigaonkar** *ICMR-NIRRCH, Mumbai* **From Follicle to Oocyte: Understanding the Impact of Energy Disruption in Women with PCOS**
OP-C-18	**Snehal Bhingardeve** *ICMR-NIRRCH, Mumbai* **Altered m6A-miRNA Interplay contributes to Epitranscriptomic Remodelling in PCOS Pathophysiology**

OP-C-19	**Agalya M** *University of Madras* **Cholecalciferol ameliorates the oxidative stress and renal dysfunction in PCOS rat model**
OP-C-20	**Rithika Rajendran** *ICMR-NIRRCH, Mumbai* **Effect of Extracellular High Mobility Group Box 1 in the Uterine Microenvironment During Embryo Implantation**
OP-C-21	**Itti Munshi** *ICMR-NIRRCH, Mumbai* **Why Lesions Last: Stromal DNA Repair Reprogramming in Endometriosis**
OP-C-22	**Anirudh Tiwari** *ICMR-NIRRCH, Mumbai* **Evaluation of the preventive effect of nano-curcumin, alpha-linolenic acid and their combination supplementation on a rat model of pre-eclampsia**

OP-C-1

EVALUATION OF SEX SPECIFIC TRANSCRIPTOMIC PROFILE DURING GONADAL DEVELOPMENT IN THE ASIAN SEABASS (LATES CALCARIFER, BLOCH, 1790)

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Asian Seabass (Lates calcarifer, family: centropomidae) is a catadromous, euryhaline marine teleost exhibiting protandrous hermaphroditism i.e. maturing initially (< 2 years) as males and subsequently (3–4 years) transforming into females. Despite the reported morphological and physiological changes, the molecular network(s) driving such sex reversal remain largely unknown limiting the farm productivity. This study aims to investigate the molecular mechanisms underlying the sex determination and differentiation in this species. The histological architecture and expression patterns of critical genetic markers were evaluated across successive developmental stages [30 DPH, 90 DPH, 180 DPH, 250 DPH, 15 ± 3 MPH, mature males (2–3 years), and females (4–5 years)] of gonads. The expression profile of multiple genes involving- i) bi-potential gonadal development (Wt1, Gata4); ii) sex determination (Amh, Dmrt1, Sox3, Foxl 2); iii) steroidogenesis (Cyp19a1a, Cyp11β, Sf1, Hsd11β); iv) germ cell maturation (Foxl 3, Piwil1, Zp2, Vasa, Sycp31, Spet7); v) Wnt Signalling (Ctnnb, Ck2a) and vi) retinoic acid signalling (Stra6) were evaluated. Our data revealed that the transcripts of sex determining (Amh, Dmrt1, Sox3, Wt1) and steroidogenic (Cyp11β, Hsd11β) genes were elevated in maturing and adult testes, whereas mRNAs of germ cell maturation (Vasa, Sycp31, Zp2, Piwil1), RA signalling (Stra6) and Wnt Signalling (Ck2a) genes were enriched in adult ovaries. We here propose Amh to be a prominent master regulator of male sex differentiation in this species. Subsequent studies will involve next generation sequencing of these developing gonads followed by experimental demonstration of steroid induced sex inversion.

OP-C-2

ROLE OF GABA IN STRESS-INDUCED SUPPRESSION OF REPRODUCTION IN FEMALE *POECILIA SPHENOPS*

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The relationship between stress and reproduction in fish is mediated through complex neuroendocrine pathways, including activation of the hypothalamic–pituitary–interrenal (HPI) axis. However, the role of gamma-aminobutyric acid (GABA) in reproductive stress response remains poorly understood in teleosts. In this study, we examined the involvement of GABA in stress and reproduction in female black molly *Poecilia sphenops* by employing GABAA and GABAB receptor-specific antagonists under normal and stress conditions. Chronic exposure to intermittent aquacultural stressors for 29 days significantly elevated brain GABA content as well as whole-body and head-kidney cortisol levels, concomitant with significant reduction in the ovarian-somatic index, numbers of follicles at both previtellogenic (stages I-III) and vitellogenic (stages IV and V) stages, most of the embryonic developmental stages and overall fecundity levels compared with experimental controls. Blockade of GABAA receptors with high-dose gabazine enhanced these reproductive indices in both normal and stressed fish. In contrast, although GABAB receptor antagonism by CGP-35348 hydrate stimulated stage I-V follicular development, their numbers in stress+ CGP-35348 hydrate-treated fish remained comparable to those in the stress-only group. Collectively, these findings indicate that GABA participates in the regulation of ovarian activity during stress, and that GABAA receptor blockade by gabazine effectively restores the stress-induced suppression of reproduction in viviparous teleosts. These findings provide valuable insights for improving breeding success in ornamental fisheries. Acknowledgement: This work is supported by Council of Scientific and Industrial Research (CSIR), New Delhi (File No. 09/0101(12338)/2021-EMR-I).

OP-C-3

IMPACT OF MATERNAL METHYL DONOR DEFICIENCY DURING GESTATION ON FERTILITY AND GERM CELLS' DNA METHYLATION IN MOUSE OFFSPRING

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The Developmental Origins of Health and Disease (DOHaD) framework links early-life nutritional perturbations to adult metabolic disorders; however, the epigenetic origins of reproductive dysfunction remain less understood. This study examined the effects of gestational methyl donor deficiency (vitamin B12, folate, and methionine) on reproductive development and fertility of F1 male mice. Pregnant OCT4-GFP mice received either a control chow diet (CCD) or a 40% methyl donor–deficient diet (MDD) from gestational day (GD) 5 to 20, with offspring subsequently maintained on CCD. Reproductive development was evaluated at GD18, postnatal day (PND) 22, and PND60 using histopathology and histomorphometry. Whole-genome bisulfite sequencing (WGBS) was performed on primordial germ cells (PND0) to identify differentially methylated cytosines (DMCs) and genes (DMGs). F1 males from MDD dams exhibited cryptorchidism, increased postnatal mortality, and progressive testicular degeneration from GD18 to PND60. Quantitative analyses revealed depletion of spermatogonia, spermatocytes, and Sertoli cells, alongside seminiferous tubule atrophy, germ cell sloughing, and TUNEL-positive apoptosis. Adult males of MDD group showed markedly reduced sperm count, motility, copulation index and fertility index as compared to CCD, indicating subfertility. Methylome profiling of primordial germ cells identified 18,835 DMCs and 331 DMGs, enriched for pathways regulating spermatogenesis, meiosis, and fertility. These findings demonstrate that gestational methyl donor deficiency profoundly disrupts male reproductive development through reprogramming of germline methylation. The results emphasizing that maternal one-carbon nutrient imbalance may lead to epigenetic alterations predisposing offspring to reproductive disorders.

OP-C-4

GENE EXPRESSION PROGRAMS AND ALTERNATIVE SPLICING IN PLACENTAL CELL STATES REVEAL NOVEL UNDERPINNINGS OF PRETERM BIRTH

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The placenta is composed of diverse maternal and fetal cell types and is the most important organ in pregnancy. We investigated the cell type and cell state diversity as well as gene expression programs and alternative splicing, by performing single-cell RNA sequencing of 8 and 7 placental samples from preterm and term deliveries, respectively, of women recruited in the GARBH-Ini cohort. We identified major cell types and cell states. Syncytialization was significantly down-regulated in syncytiotrophoblasts in preterm placentae, suggesting poor nutrient transfer from mother to fetus. Our results also suggested altered response to hypoxia might lead to up-regulated EMT in trophoblasts, enhanced angiogenesis in arterial endothelial cells, and spiral artery remodeling in vascular smooth muscle cells, which might ultimately affect placental bed formation. At the maternal–fetal interface, both maternal and fetal macrophages showed reduced antigen presentation, suggesting impaired immune response to infections. Additionally, dysregulation of cytotoxicity in NK cell states also suggested persistence of infection. We deployed long read single cell sequencing to elucidate, for the first time, cell state specific alternative splicing in the placenta, which provided novel information on differential expression of alternate spliced transcripts in preterm deliveries. Together, our findings highlight poor nutrient transfer, hypoxia driven EMT activation, spiral artery remodeling and immune suppression as central mechanisms in preterm birth and provide a comprehensive single-cell framework for understanding placental dysfunction and identifying future therapeutic targets

OP-C-5

GUT MICROBIOME PROFILES ASSOCIATED WITH HCMV IN PREGNANCIES COMPLICATED BY BOH

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Pregnancy-related immune modulation may increase susceptibility to human cytomegalovirus (HCMV), the leading cause of congenital infections associated with adverse pregnancy and neonatal outcomes. The gut microbiome plays a vital role in immune regulation, but its interaction with HCMV infection in pregnant women with a bad obstetric history (BOH) remains unclear. We investigated this relationship, hypothesizing that immune dysregulation linked to BOH might influence viral infections through microbiome alterations, or conversely, that microbiome composition could affect HCMV infection and immune balance during pregnancy.

Pregnant women were grouped as BOH HCMV-positive (n=10), BOH HCMV-negative (n=10), control HCMV-positive (n=10), control HCMV-negative (n=10), and non-pregnant (n=10). Gut microbiota profiles were determined using 16S rRNA sequencing (Oxford Nanopore Technology). Stool supernatants were analyzed for short-chain fatty acids (SCFAs) and their capacity to modulate Th1/Th2 responses in HuT78 cells.

BOH HCMV-negative women showed higher levels of Ruminococcus and Dorea (pro-inflammatory) and reduced Faecalibacterium (anti-inflammatory), along with a lower Th2 response. HCMV-positive women exhibited increased Enterococcus, Bacillus, Lactobacillus, and Clostridium. Despite the presence of beneficial taxa, fecal supernatants from HCMV-positive women reduced Th2 responses, likely due to elevated valerate and Enterococcus. BOH women with HCMV infection showed a combined microbial signature of BOH- and CMV-associated taxa.

BOH is associated with a pro-inflammatory microbiome, potentially resulting from prior immune insults. HCMV infection intensifies this imbalance, which may increase the risk of adverse pregnancy outcomes. Understanding these interactions may guide microbiome-based strategies to support healthier pregnancies.

OP-C-6

EARLY MATERNAL IMMUNE SIGNATURES OF HCMV INFECTION AND ITS ASSOCIATION WITH PREGNANCY OUTCOMES AND CCMV: A PROSPECTIVE STUDY

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Human Cytomegalovirus infection is common with high seroprevalence in Indian women of reproductive age >90%. India also bears highest burden of congenital HCMV infection (cCMV)>1.5%. Studies addressing role of maternal systemic and virus-specific cellular immune responses are limited. We delineated maternal immune signatures associated with cCMV and adverse pregnancy outcomes (APO). Pregnant women;83, with;(45) and without BOH;(38) were recruited and followed longitudinally. Non-pregnant women;19 were recruited cross-sectionally. Multiparametric flow cytometry was employed to evaluate early maternal cellular immunity within: Healthy, Pregnancy loss;(PL), congenital HCMV infection with symptoms;(cCMV+SB), cCMV, symptomatic birth;(SB) and normal pregnancy outcome;(NPO) groups. TBNK cells, Monocytes and their subsets were enumerated along with activation and PD1 expression within different subsets of T-cells. HCMV-specific T-cell response against HCMV-(pp65, IE1, gB) were analyzed using ICCS assay. Degranulation;(CD107a), chemokine;(MIP-1β), and Th1 cytokine production;(IFN-γ, TNF-α, and IL-2) was assessed within different T-cell subsets. PL and cCMV+SB the APO groups, were enriched for recent HCMV infection. PL showed higher levels of B-cells. Both groups showed increased activation but low PD-1 expression, indicative of immune dysfunction, along with high IL-2 production against pp65 and gB. HCMV-specific cytolytic responses were higher in these groups. cCMV group was enriched for HCMV reactivation and NPO group for past exposure. The cCMV group exhibited lower counts of lymphocytes, and monocytes, along with lack of cytolytic responses, Th1 cytokine production, and increased MIP-1β production along with increased activation and PD-1 expression, reflecting upon modulated immune response. Our findings highlight potential maternal immune signatures associated with cCMV and APO.

OP-C-7

KISSPEPTIN-10–INDUCED KISS1 ACTIVATION ORCHESTRATES COMPREHENSIVE ANTI-TUMOR, ANTI-METASTATIC, AND PRO-APOPTOTIC REPROGRAMMING IN TRIPLE-NEGATIVE BREAST CANCER CELLS

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Triple-negative breast cancer (TNBC), defined by the lack of ER, PR, and HER2 receptors, continues to be one of the most therapeutically challenging breast cancer subtypes. In this study, we evaluated the effects of exogenously administered Kisspeptin-10 (Kp-10) on MDA-MB-231 and MDA-MB-468 TNBC cell lines using a combination of in vitro approaches and computational analyses. Kp-10 exposure significantly reduced cell viability and migratory potential while inducing a dose-dependent increase in KISS1 transcripts, suggesting a self-reinforcing regulatory mechanism. Key transcription factors, including GATA2, CDX2, and FLI1 were upregulated, whereas ZEB1 expression declined, indicating a shift toward a less invasive transcriptional state. EMT-related changes supported this transition, with enhanced E-cadherin and β-catenin levels and decreased N-cadherin, Vimentin, and CD44 expression. Apoptotic pathways were activated, demonstrated by elevated CASP3, CASP8, CASP9, and BAX expression alongside reduced BCL2, reflecting involvement of both intrinsic and extrinsic mechanisms. Metabolomic profiling further revealed alterations associated with apoptosis, oxidative stress balance, and anti-angiogenic activity. In silico findings showed diminished KISS1 expression in metastatic TNBC tissues and indicated that higher GATA2 or CASP9 expression correlates with improved patient survival outcomes. Overall, these results demonstrate that Kp-10 enhances KISS1 levels and drives broad anti-tumor effects through transcriptional modulation, EMT inhibition, activation of apoptotic signalling, and metabolic reprogramming, reinforcing its therapeutic potential in TNBC.

OP-C-8

PROTEOMIC ANALYSIS OF 2D VERSUS 3D GROWN OVARIAN CANCER CELLS: INSIGHTS INTO FERROPTOSIS REGULATION

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Extracellular vesicles (EVs) are crucial mediators of intercellular communication in cancer profoundly impacting cancer cell survival, invasion, immune suppression and drug resistance. Given their release into body fluids, EV cargo represents a potential source of cancer biomarkers and due to their tumor promoting activity; they are also considered as potential therapeutic targets. To identify cargo vital for cancer progression, we undertook comparative proteomic profiling of EVs derived from ovarian cancer cells (OVCAR4) grown under 2D and 3D conditions. EVs were characterized by Nano-particle tracking analyzer and by immunoblotting for presence of classical EV markers. They were subjected to label free quantification by LC MS/MS. 3D-derived EVs were found to be enriched in proteins related to iron regulatory pathways. 14 out of 20 proteins in the 3D-EV cargo were involved in the suppression of ferroptosis- an iron-dependent, regulated cell death. Among these, two proteins were validated by Western blotting in three ovarian cancer cell lines. Sensitivity of these three cell lines to ferroptosis inducing agent was compared in 2D versus 3D culture condition and interestingly, all 3 cell lines showed resistance to ferroptosis under 3D condition. Further, proteins associated with the ferroptosis pathway were also upregulated in EVs isolated from ascites in the syngeneic mouse model of ovarian cancer. Consistent presence of ferroptosis regulating proteins across in vitro and in vivo models underscores their importance in ovarian cancer progression.

OP-C-9

MULTI-OMICS PROFILING REVEALS METABOLIC REWIRING AS CENTRAL TO INTERACTIONS BETWEEN HUMAN VAGINAL CELLS AND CANDIDA ALBICANS

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Candida albicans is a commensal fungus commonly present on mucosal surfaces of its human host. Under conditions of host stress, it transforms into a pathogenic state, causing localized or systemic infections. Symptomatic infections caused by Candida species in the reproductive tract of women are called vulvovaginal candidiasis (VVC). The treatment options for the management of VVC are limited to a few antifungal drugs, and several Candida species have been increasingly developing resistance to them. A better understanding of the host-pathogen interactions would pave the way to the identification of novel antifungal targets. We used an in vitro model of VVC wherein C. albicans was co-cultured with the human vaginal epithelial cell line A-431 in conditions mimicking the host microenvironment. Untargeted multi-omics techniques were employed to gain a global overview of their interactions and help connect the host and pathogen at different biological levels. Integrating the transcriptomics, proteomics and metabolomics profiles revealed significant perturbations in several metabolic pathways of both the host cell line as well as C. albicans. A possible competition for shared nutrients such as vitamins, amino acids and fatty acids was observed, suggesting their potential as novel antifungal targets.

OP-C-10

PREDICTIVE FUNCTIONAL PROFILING REVEALS GUT MICROBIAL METABOLIC SHIFTS LINKED TO IMMUNE ACTIVATION IN HIV INFECTION

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Sexually transmitted diseases like HIV extend beyond localised infection to cause systemic immune activation. The gut microbiome is a non-genetic host factor which is linked to HIV associated inflammation and mucosal barrier dysfunction. However, the functional metabolic consequences of these microbial changes remain poorly understood. In view of this, a cross-sectional analysis of the gut microbiome was carried out on antiretroviral therapy naïve HIV+ve (n=43) and HIV-ve (n=35) individuals. Microbiome data analysis of V3-V4 region of 16S rRNA was performed using QIIME2 pipeline and predictive microbial functional abundance was identified by PICRUSt2 software. Differential pathway enrichment was identified (FDR < 0.05) and correlated with short chain fatty acid levels and immune activation markers. HIV+ve samples showed enrichment of heme (log2FC=1.72) and menaquinone biosynthesis (log2FC=1.6), TCA/glyoxylate cycles (log2FC=2), and LPS/peptidoglycan biosynthesis (log2FC=1.92) pathways indicating elevated microbial energy metabolism and endotoxin potential. These correlated positively with increasing plasma sCD14 (r=0.5), viral load (r=0.4) and negatively with decreasing fecal butyrate (r=-0.23), succinate (r=-0.23) and CD4 count (r=-0.37). HIV-ve samples had higher formaldehyde assimilation (log2FC=-1.09) and glutamate degradation pathways (log2FC=-3.31) linked to detoxification which positively correlated with fecal butyrate (r=0.43), succinate (r=0.2) and CD4 count (r=0.34) and negatively with sCD14 (r=-0.44) and viral load (r=-0.51). Our finding suggests that HIV associated immune dysregulation reprogrammes functional pathways of gut microbiome which may in turn contribute to the systemic and mucosal immune activation. Targeting the taxa which contributes to these dysregulated functional pathways could represent novel strategy to restore immune homeostasis in HIV infection.

OP-C-11

ALTERED MYOKINE PROFILE DURING PROGRESSION OF OBESITY-MEDIATED TYPE 2 DIABETES IN MALE MICE

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The prevalence of obesity is considered as a global public health challenge. It is characterized by the imbalance between energy consumption and its expenditure, ectopic lipid accumulation in adipose tissue and peripheral organs and the increased risk of type 2 diabetes. Skeletal muscle acts as an important target tissue of insulin. It is responsible for the clearance of 80% of circulatory insulin-stimulated glucose uptake. Insulin resistance firstly developed in skeletal muscle. However, the role of skeletal muscle malfunction during obesity and its contribution in obesity-induced type 2 diabetes is not properly understood. Against this background, the objective of this work is to evaluate the secretory pattern of myokines along with the impairment of insulin sensitivity in normal condition and during the onset and progression of obesity-mediated type 2 diabetes. Experiments were performed on male mice, randomized into standard diet (SD) or control group and high-fat diet (HFD) group. For both groups, duration of feeding continued for 1 month, 4 months and 8 months. It was observed that in comparison to SD group, body weight and serum profile of metabolic parameters significantly increased in obese mice with HFD feeding. Structural changes of skeletal muscle tissue were observed by histological analyses and scanning electron microscopy with progression of obesity; supported by oil red O staining. In obese condition, alterations in the expression of myokines *viz.* myostatin, irisin and others and impaired insulin signalling were observed. The findings showed changes in the secretory pattern of myokines during the progression of obesity-induced insulin resistance.

OP-C-12

THE ZEBRAFISH OVULATORY BRAIN: MECHANISTIC INSIGHTS INTO THE NEUROENDOCRINE AND METABOLIC CASCADES GOVERNING REPRODUCTION

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The vertebrate ovulatory response is a coordinated concert of neuroendocrine, ovarian and environmental signals. While the role of hypothalamic neuropeptides during ovulation is well characterised in mammals, their involvement in the ovulatory response of zebrafish (Danio rerio), which breeds in laboratory conditions under controlled photo-thermal regimen, remains elusive. Our results demonstrate that, congruent with increased germinal vesicle breakdown at 0700h (pre-ovulatory period) and ovulation at 0830h (peri-ovulatory period), the expression of KISS1, KISS2, KISS1R and gnrh3, fshb and lhb transcripts in zebrafish brain undergo a sharp increase at 0700h. Concurrent upregulation of brain cyp11a1, cyp17, hsd3b and StAR, P450 Aromatase, and ERα proteins at 0700h hints at enhanced steroidogenic potential and estrogenic influence on the neuroendocrine axis. Additionally, enhanced IGF1 and p-IGF1Rβ (Tyr1135/1136) expression, along with increased p-ERK1/2 (Thr202/Tyr204) and p-AKT (Thr308 and Ser473) levels, indicate a probable KISS2-IGF1 crosstalk during morning hours. Mechanistically, reduced SIRT1 expression and altered histone modifications align well with elevated kiss2 transcription. Further, increased p-AMPKα (Thr172), GLUT1 and Citrate synthase, along with reduced PDHK1 expression, corroborate well with upregulated mitochondrial transcription factors (TFAM, NRF1), complex subunits (NDUFS4, SDHA, UQRCRC2, COXIV, ATP5α1), and increased ATP and ROS levels at 0700h, potentially indicating metabolic reprogramming in the brain during morning hours prior to ovulation. Collectively, these results elucidate for the first time that zebrafish ovulation involves a coordinated neuroendocrine-metabolic cascade, integrating KISS2-GnRH3-LH signalling, steroid feedback, IGF1-mediated pathways, and enhanced neuronal energy metabolism, underpinning the ovulatory response in zebrafish bred in captivity.

OP-C-13

OSR1 ORCHESTRATES AND LIMITS CARDIAC FIBROSIS AND HYPERTROPHY

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During physiological stress, increased oxygen demand prompts cardiac enlargement to maintain cardiac-output. However, sustained stress disrupts cardiomyocyte–extracellular matrix interactions, driving maladaptive remodelling, fibrosis, and eventual heart failure. OSR1, a cardiogenic zinc-finger transcription factor, is well-studied in fetal heart development, yet its role in adult cardiac hypertrophy remains largely unexplored. In rats, subcutaneous isoproterenol administration elevated the cardiosomatic index, upregulated pro-hypertrophic and fibrotic markers, and induced OSR1 overexpression. Global mRNA sequencing of hypertrophied hearts identified numerous differentially expressed genes (DEGs); in vivo validation confirmed 16 unreported genes implicated in key biological processes. These were further examined in hypertrophied neonatal rat ventricular cardiomyocytes (NRVMs), where eight genes mirrored hypertrophic responses under hypoxic stress, suggesting hypoxia-driven dysregulation. In silico promoter analysis revealed 26 DEGs as potential OSR1 transcriptional targets. Similarly, isoproterenol-treated mice exhibited increased OSR1, fibrosis, and hypertrophy, which were attenuated by administration of OSR1-shRNAs in lentiviruses. Nuclear-localized OSR1 interacted with 39 transcription factors in hypertrophied hearts, identified via Immunoprecipitation–Mass Spectrometry. Interestingly, OSR1 expression was higher in neonatal cardiac fibroblasts than in NRVMs, indicating a role of cellular communication in cardiac hypertrophy. OSR1-silenced cardiac fibroblasts showed reduced fibrotic activity, supporting OSR1's pro-fibrotic role in pathological hypertrophy. Our future aim is to investigate the mechanisms of EV-mediated OSR1-TFs interaction in the control of pathological hypertrophy and fibrosis. [This work was supported by grants from Department of Biotechnology, BT/PR50045/CMD/150/86/2023]

OP-C-14

ELUCIDATING THE ROLE OF FGF2 IN FISH TESTICULAR STEROIDOGENESIS

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Fibroblast growth factor (FGF) super family is conserved among vertebrates and are known to regulate several biological processes including reproduction. Studies pertaining to the role of these correlates in gonads is mostly restricted to FGF2 and FGF9 in mammals and a few reports in fish. For present work, two fish species, the common carp, Cyprinus carpio and the Asian catfish, Clarias batrachus/magur were chosen which breeds biannually and annually, respectively. Transcripts of fgf2 were sub-cloned and quantitative PCR analysis confirmed high expression of fgf2 in gonad and during spermiation phase in both the species. Similar expression of fgf2 in two species displaying differential seasonal reproductive phase pattern indicate an active role in testis. Further, it was also shown that gonadotropin and androgens can induce fgf2 expression. To comprehend the role of fgf2 in fish testicular function, tissue localization and transient gene silencing of fgf2 was performed. In testis, spermatocytes and interstitial cell layer showed immunoreactivity. Antisense fgf2 siRNA-PEI treatment in vivo, in both the species, declined transcript and protein levels of fgf2 on day1, hence, day1 was chosen to analyse the impact of transient gene silencing of fgf2. Upon fgf2-siRNA treatment, in both species, expression of several steroidogenesis related transcription factors and growth factors were altered. Serum testosterone and 11-ketotestosterone declined post fgf2-siRNA treatment. All these results indicated that fgf2 plays pivotal role in regulating testicular steroidogenesis by interacting with other steroidogenesis related factors/genes and is probably involved in androgen production either directly or indirectly.

OP-C-15

BMAL1 REGULATES MONOCYTE TRAFFICKING IN ZEITGEBER DISRUPTED C57BL/6J MICE

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Circulating monocytes are critical to innate immune system as the first responders to inflammation. Monocyte are known to migrate from the site of storage to inflamed peripheral tissues. However, dynamics of monocytes trafficking under conditions of altered clock conditions are not well characterised. Herein we have shown that monocyte recruitment at the sight of injury is regulated by the circadian clock gene Bmal1. We have observed that photoperiod induced circadian desynchrony affects the oscillating pattern of Bmal1, Clock, Per, Cry1 and Rev-erb□ in circulating monocytes. Simultaneously affecting the trafficking of monocytes as evidenced by flow cytometric analysis. It was observed that chronodisruption augments release of CD11b+ Ly6C+ Monocytes into circulation as evidenced by their higher abundance in blood and spleen. A timepoint based assessment of CD11b+Ly6C+ showed two peaks at ZT6 and ZT18 as compared to a single peak at ZT18 in control. Further, high number of CD11b+Ly6C+CD86+M2 monocytes in circulation implies a shift towards an anti-inflammatory phenotype. Monocytes from blood and bone marrow of chronodisrupted mice were further adoptively transferred to Control and CCL4 treated mice to decipher inflammo-tactic behavior. Flow cytometric analysis of Blood, Bone marrow, liver and spleen corroborates with our hypothesis wherein CD monocytes showed a 3 fold increased abundance in liver as compared to control. Taken together, an altered Bmal1 function affects piloting of monocytes that culminates in disrupted trafficking.

OP-C-16

TRANSGENERATIONAL REPRODUCTIVE TOXICITY OF THE HISTONE DEACETYLASE INHIBITORS, VALPROIC ACID AND SODIUM BUTYRATE IN MALE RATS

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Histone deacetylases (HDACs), are perturbed in a number of human diseases. Sodium valproate (VPA), a HDAC inhibitor (HDACi), has been a choice for the treatment of epilepsy. Sodium Butyrate (SB) is also a short chain fatty acid similar in structure and function to VPA is also used for treating neurological disorders. In this study we evaluated the transgenerational toxicity of VPA and SB. Adult male Wistar rats were treated daily for two months with VPA and SB at doses equivalent to that is prescribed for treatment in neuronal disorders. By natural mating, pups of F1 and F2 generations were obtained and reproductive parameters analyzed. Fertility rate, litter size, gender ratio and litter weight was significantly compromised in the male rats exposed to VPA, whereas such changes were not evident in SB treated male rats. Histopathological analyses indicated severe damage to the anatomical architecture in the epididymis of VPA treated rats. Perturbations in the levels of reproductive hormones (estradiol and testosterone) were observed in VPA treated rats, but not in SB treated rats. Surprisingly, the changes observed in the reproductive function of F0 rats were also compromised in the F1 and F2 generations. Such transgenerational changes were not observed in SB-treated rats. Administration of VPA at doses equivalent to that used in clinical set up may cause severe alterations in male reproductive physiology. In our study, SB did not cause any reproductive toxicity and can be a proposed as safer alternative to the VPA for the treatment of neurological disorders.

OP-C-17

FROM FOLLICLE TO OOCYTE: UNDERSTANDING THE IMPACT OF ENERGY DISRUPTION IN WOMEN WITH PCOS

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Oocyte quality is significantly influenced by cross-talk between oocytes and granulosa cells (GCs). GCs act as metabolic drivers of the oocyte by metabolizing glucose and supplying the oocyte with essential energy substrates and intermediates. PCOS is common endocrine disorder affecting women of reproductive age, characterized by elevated androgens, altered gonadotropin levels, insulin resistance, anovulation, and sub-optimal oocyte quality. We aim to investigate glucose metabolism dynamics in oocyte microenvironment in PCOS women and healthy controls undergoing IVF, by utilizing follicular fluid (FF) and GCs. Basal and stimulated glucose uptake was studied by flow cytometry and was found to be lower in GCs from PCOS women. Basal GLUT4 transcript and protein expression; and translocation were significantly lower in GCs from PCOS women. In PCOS follicle, glycolysis was downregulated as indicated by decreased transcript levels of rate-limiting enzymes, reduced pyruvate levels and lower real-time glycolysis. Alternatively, pentose phosphate pathway, polyol pathway and glycation were increased in GCs from PCOS women. GCs from PCOS women showed increased oxidative stress and higher expression and nuclear translocation of NRF2. Our work reveals that women with PCOS exhibit fundamental metabolic defects within follicular compartment, potentially impacting oocyte quality. By examining the energy dynamics present in the follicle, we can gain critical insights into pathways that may contribute to disrupted folliculogenesis in PCOS and essential metabolites that could be incorporated into oocyte and embryo culture media in the IVF settings. The cues from our data may help identify potential markers indicative of oocyte and embryo quality.

OP-C-18

ALTERED M6A-MIRNA INTERPLAY CONTRIBUTES TO EPITRANSCRIPTOMIC REMODELLING IN PCOS PATHOPHYSIOLOGY

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Polycystic ovary syndrome (PCOS) is an endocrine disorder characterised by hyperandrogenism, amenorrhoea, and ovulatory dysfunction. Although PCOS represents the leading cause of infertility, its underlying aetiology remains elusive. Among the various genetic and epigenetic factors implicated in its pathogenesis, alterations in DNA methylation and microRNA expression have emerged as key regulators of ovarian function. More recently, biochemical modifications of eukaryotic mRNA have been recognised as epitranscriptomic mechanisms that influence mRNA expression. In particular, the N6-methyladenosine (m6A) modification, which is crucial for mRNA splicing, degradation, and stabilisation, is involved in ovarian development, homeostasis, and pathological processes. However, the implications of m6A alterations in the PCOS ovary remain insufficiently explored. In this study, we investigated global m6A levels and the relative transcript expression profiles of m6A-regulatory genes in granulosa cells from women with PCOS. Our findings reveal elevated global levels of m6A methylation compared with age and BMI-matched controls, along with upregulation of m6A writer enzyme transcripts, including METTL3, METTL14, WTAP, and VIRMA. Consistently, we observed increased expression of reader transcripts (YTHDF1, YTHDF3, and YTHDC1) and downregulation of eraser transcripts (FTO and ALKBH5). Additionally, the miR-607, which targets FTO, was upregulated in PCOS. Notably, the global m6A level and expression profiles of METTL3, METTL14, FTO, YTHDF1, and miRNAs demonstrated interdependence and showed significant correlations with the elevated free and bioavailable androgens characteristic of PCOS follicles. Together, these findings provide new insights into the dysregulation of the m6A machinery in PCOS.

OP-C-19

CHOLECALCIFEROL AMELIORATES THE OXIDATIVE STRESS AND RENAL DYSFUNCTION IN PCOS RAT MODEL

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Polycystic ovarian syndrome (PCOS) is the most common endocrine disorder that affects women of reproductive age. Renal dysfunction in PCOS and therapeutic benefits of cholecalciferol (vitamin D3) are largely underexplored. Therefore, we examined the impact of PCOS on renal redox-antioxidant system and renoprotective potential of vitamin D3 in rats.

PCOS was induced by subcutaneous injection of testosterone propionate (TP) (10 mg/kg b.wt.) in 21-25 days old female Wistar rats for 35 days. Rats exhibiting persistent diestrous cycles were divided into 1) Control group: Corn oil alone, 2) PCOS group: TP, 3) PCOS + Vitamin D3 group: Cholecalciferol (1000 IU/kg b.wt.) orally daily for 30 days along with TP. After treatment period, rats in diestrous phase were euthanized. Serum and kidney tissues were analyzed for various parameters. Statistical analysis was performed by one-way ANOVA followed by SNK test.

There was a significant increase in kidney weight in PCOS rats, which was restored to control level following vitamin D3 supplementation. Serum urea and creatinine were elevated in PCOS group, while vitamin D3 treatment reduced these levels. Redox-antioxidant system analysis revealed increased hydrogen peroxide levels and lipid peroxidation, accompanied by reduction in non-enzymatic (vitamin C, reduced glutathione) and enzymatic (catalase, GPx) antioxidants in kidneys. Interestingly, vitamin D3 restored these parameters to near control levels.

Our study demonstrated for the first time that vitamin D3 supplementation alleviated the oxidative stress and renal dysfunction in testosterone-induced PCOS rats by modulating the redox homeostasis, highlighting its potential therapeutic implications for PCOS and associated renal dysfunction.

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OP-C-20

EFFECT OF EXTRACELLULAR HIGH MOBILITY GROUP BOX 1 IN THE UTERINE MICROENVIRONMENT DURING EMBRYO IMPLANTATION

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The breaching of the endometrium by the embryo and subsequent remodelling during implantation is akin to wound-healing response that requires a controlled inflammatory environment. However, a switch to an anti-inflammatory profile is crucial for a successful pregnancy. Also, the endometrial immune cells adopt more tolerogenic phenotypes to prevent embryo rejection. Our group had previously demonstrated that excess of the alarmin High Mobility Group Box 1 (HMGB1) in the uterine cavity led to implantation failure in rodents. The objective of this study was to determine whether HMGB1-mediated implantation failure was due to an altered immune profile in the uterus. Wistar rats received intrauterine administration of recombinant HMGB1 (rHMGB1) alone or with its inhibitor glycyrrhizin on day 3 post-coitum(p.c.). Control animals were administered saline. The animals were sacrificed on day 5p.c. Part of the uterus was used for paraffin-block preparation for histology and immunofluorescence. The uterus was cut open and scraped for the preparation of single-cell suspension for flow cytometric analyses of the immune cells. Uterine fluid was collected for cytokine array. Co-administration of glycyrrhizin with equal dose (0.8μg/15μL) of rHMGB1 mitigated the adverse effects of HMGB1 on implantation. This was also associated with improved decidualization reaction in the uteri that received glycyrrhizin+rHMGB1 treatment, as observed using immunofluorescence for Prolactin($p=0.0004$) and IGFBP1 ($p=0.0038$). Uterine (u)NK (DBA-lectin+) cells($p=0.0002$) and Tregs($p=0.0224$) frequencies were altered due to excess HMGB1, whereas glycyrrhizin+rHMGB1 treatment restored their numbers. rHMGB1-treatment also led to a reduction in the frequency of M2-macrophages activated during tissue remodelling (CD86+MHCIIlow), which were significantly higher in the horns that received glycyrrhizin+rHMGB1($p=0.0021$). Our investigations revealed that excess HMGB1 altered the immune profile, leading to implantation failure. These effects were abrogated by glycyrrhizin.

OP-C-21

WHY LESIONS LAST: STROMAL DNA REPAIR REPROGRAMMING IN ENDOMETRIOSIS

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Endometriosis—ectopic growth of endometrium-like tissue—affects 10% of reproductive-age women and is characterized by adhesive, invasive, and proliferative lesions across ovarian, peritoneal, and rectovaginal sites. Both eutopic (EUE) and ectopic (ECE) endometrium endure sustained replicative, oxidative, and inflammatory stress driven by high local estrogen, iron overload, immune dysregulation, and genotoxic exposures, yet the survival of endometrial cells in the ectopic niches remains enigmatic. Emerging genomic studies reveal epithelial-restricted cancer-driver mutations with relatively fewer stromal alterations, suggesting a protective advantage in the stromal cells of endometriotic lesions. So, we asked whether DNA damage signalling (DDR) is modulated in ectopic stromal cells. We profiled DDR sensors (γH2AX and MBD4) and transducers (ATM and pATM) in the stromal cells of matched EUE and ECE. Oxidative, genotoxic, and hyperestrogenic stimuli upregulated MBD4 in endometrial stromal cell line (T HESC), an effect blunted by ERα blockade. MBD4 silencing impaired damage recognition, reducing ATM phosphorylation ($p<0.05$) and γH2AX recruitment ($p<0.01$), at heightened DNA damage conditions, indicating that MBD4 helps launch stromal DDR. These findings reconcile reports of high damage signals in the endometriotic environment with stromal resilience and supports a model in which stromal DDR remodeling underpins lesion persistence in a hostile microenvironment. These insights motivate targeted, stromal-focused interventions.

OP-C-22

EVALUATION OF THE PREVENTIVE EFFECT OF NANO-CURCUMIN, ALPHA-LINOLENIC ACID AND THEIR COMBINATION SUPPLEMENTATION ON A RAT MODEL OF PRE-ECLAMPSIA

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Preeclampsia (PE) is a complex hypertensive disorder that occurs during pregnancy, characterized by maternal endothelial dysfunction, renal impairment, and systemic inflammation. It is associated with abnormal placentation, impaired uteroplacental blood flow, increased oxidative stress, and dysregulated immune signaling, leading to fetal growth restriction and long-term developmental effects. In India, PE affects around 8% of pregnancies, resulting in approximately 70,000 maternal and 500,000 neonatal deaths each year. In our study, we explored a nutritional supplement approach using Nano-curcumin (NC) and alpha-linolenic acid (ALA) to mitigate the adverse effects of PE on mothers and their offspring using an LPS+L-NAME induced PE rat model. We divided thirty Wistar rats into five groups: a Vehicle control group (0.05% CMC), a disease control group (PE) treated with LPS (20-80 µg/kg body weight via intraperitoneal injection) and L-NAME (300 mg in drinking water ad libitum), and three treatment groups (PE+NC at 400 mg/kg body weight, PE+ALA at 150 mg/kg body weight, and PE+NC+ALA with NC at 150 mg/kg body weight and ALA at 400 mg/kg body weight). We administered NC and ALA supplements to the rats by oral gavage for 14 days prior to conception, after which preeclampsia was induced from gestational day 12 onwards. Throughout the study, we monitored blood pressure, urinary creatinine and albumin levels, and assessed immune and hematological markers of PE, along with blood clinical chemistry and lipid profiles in the pregnant rats. After delivery, we observed the pups until they reached sexual maturation, recording their growth and developmental milestones. We also evaluated the learning abilities and cognitive functions of the offspring using a maze test. Our findings indicated that dietary supplementation with NC, ALA, or a combination of both during pregnancy significantly improved PE-related symptoms, including hypertension, proteinuria, and fetal mortality. Additionally, the offspring demonstrated normal growth and maturation parameters, pubertal differentiation, and cognitive performance as evaluated by the maze test. Analysis of lipid and fatty acid profiles showed that supplementing with ALA, alongside NC, enhanced the overall fatty acid composition.

ORAL PRESENTATIONS: NON-COMPETITIVE

OP-NC-1	**Devaraj Sankarganesh** *Vellore Institute of Technology, Vellore* **Integrative Transcriptomic Profiling Reveals Immune-Metabolic Hub Genes and Therapeutic Targets in Polycystic Ovary Syndrome**
OP-NC-2	**Nirupama Chatterjee** *Artemis, Hospitals, Gurgaon* **Clinical, Biochemical Features and Transcriptomic Responses in Polycystic Ovarian Syndrome Subjects with Metabolic Syndrome**
OP-NC-3	**Shruti R Hansda** *Institute of Science, BHU, Varanasi* **Uterine alterations in PCOS and reproductive senescence: Melatonin in rescue**
OP-NC-4	**Rakesh Verma** *Institute of Science, BHU, Varanasi* **Effects of Bisphenol S on Reproductive Health: Role of Melatonin**

OP-NC-1

INTEGRATIVE TRANSCRIPTOMIC PROFILING REVEALS IMMUNE-METABOLIC HUB GENES AND THERAPEUTIC TARGETS IN POLYCYSTIC OVARY SYNDROME

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Polycystic Ovary Syndrome (PCOS) is a multifactorial endocrine and metabolic disorder affecting women of reproductive age, characterized by hormonal imbalance, insulin resistance, and dysregulated lipid metabolism. Numerous transcriptomic studies have explored the molecular basis of PCOS; however, integrating findings across datasets remains challenging due to variations in study design and data quality. To obtain a more consistent molecular profile, we systematically selected and analyzed publicly available RNA-Seq datasets related to PCOS. After removing duplicates, superseries, and non-transcriptomic entries, three unique datasets were included: GSE193123, GSE138518, and GSE173160. RNA-Seq analysis was performed using HISAT2 for alignment, Samtools for file conversion and sorting, and featureCounts for quantification. Differential expression analysis identified 259 genes that were significantly upregulated or downregulated in PCOS samples compared to controls. Genes associated with lipogenesis, lipolysis, and steroidogenesis were subjected to enrichment analysis using ClusterProfiler, revealing pathways related to lipid metabolism, immune signaling, and inflammation. A STRING protein–protein interaction (PPI) network was constructed, followed by MCODE clustering and CytoHubba ranking, identifying six hub genes: BTK, CYBB, SELL, IL1B, SYK, and TLR2. Molecular docking with propionic acid (PA) and metformin (MT) showed consistently stronger binding affinities for metformin across all hub proteins, suggesting its greater modulatory potential. Overall, this integrative RNA-Seq analysis highlights key immune-metabolic regulatory genes and provides valuable insights into the molecular mechanisms and potential therapeutic targets for PCOS.

OP-NC-2

CLINICAL, BIOCHEMICAL FEATURES AND TRANSCRIPTOMIC RESPONSES IN POLYCYSTIC OVARIAN SYNDROME SUBJECTS WITH METABOLIC SYNDROME

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Polycystic Ovarian Syndrome (PCOS), a heterogenous hormonal and metabolic disorder affects around 20% women of reproductive age. Environmental and lifestyle factors are implicated in the onset. A subset of PCOS patients exhibit Metabolic Syndrome (MS). The study aimed to see the major biochemical, clinical features and transcriptomic signatures in these subjects.

Seventy-nine PCOS subjects were recruited during the period of January 2024- September 2025. After informed consent, clinical history was obtained and blood samples were collected on the second or third day of menstrual cycle for testing Luteinizing Hormone (LH), Follicle Stimulating Hormone (FSH), Anti-mullerian Hormone (AMH), Free testosterone, Hb1ac, fasting and post prandial (PP) insulin. Transcriptomic response in Peripheral Blood Mononuclear Cells was seen in 4 PCOS and 3 controls using 150 Paired End Sequencing on NextSeq 2000 (Illumina).

Of the 79 subjects, 42 subjects having one or more of the following features: BMI>25kg/m2, fasting insulin > 25 µIU, PP insulin > 55 µIU or Hb1ac >5.7 had MS symptoms. Fasting and PP insulin levels were significantly higher in these subjects. Significant negative correlation with age($p=0.01$) and positive correlation with BMI ($p=0.0001$), smoking ($p=0.049$) and sedentariness (0.06) were observed in PCOS patients with MS. Genes related to immune response, DNA Damage Response and metabolic pathways were differentially regulated.

PCOS with MS is more likely in younger females with unhealthy lifestyle like smoking and sedentariness. Deregulation of pathways like insulin signaling and high DNA damage was observed.

OP-NC-3

UTERINE ALTERATIONS IN PCOS AND REPRODUCTIVE SENESCENCE: MELATONIN IN RESCUE

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Polycystic ovarian syndrome (PCOS) is a reproductive, endocrine and metabolic disorder and it affects 5-15% of women of the reproductive age worldwide. For the better understanding of PCOS, different animal models ranging from rodents to non-human primates have been used. The ovary has been the centre of research in PCOS studies, however, less is known about the mechanism of its effect on uterine function. Moreover, there are several uterine disturbances in the PCOS like increased inflammation and hyperplasia which if unregulated, may lead to uterine adenocarcinoma. PCOS women often have reduced uterine receptivity and are susceptible to spontaneous abortions and resort to assisted reproductive technology use. Melatonin administration can restore uterine functions modulating cellular dynamicity, metabolic status, decreased oxidative and inflammatory load in PCOS. Ageing is the decline in the functioning of various tissues/organs along with the escalation in age-related anomalies. Ovarian ageing is a complex and multifactorial phenomenon. Reactive oxygen species (ROS) play an important role in in the ovulatory process. Reproductive senescence affects both ovary and the uterus. With reproductive senescence there is an increase in the oxidative stress in the ovaries which might lead to decreased follicular count. Melatonin is known for its anti-ageing properties and an anti-oxidant can prove to improve the ovarian parameters uterine parameters can might be helpful in delaying reproductive senescence.

OP-NC-4

EFFECTS OF BISPHENOL S ON REPRODUCTIVE HEALTH: ROLE OF MELATONIN

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Since the last few decades, the prevalence of subfertility/infertility related issues has risen markedly, distressing millions of people. The rapid development of industries has undeniably upgraded our everyday life but has simultaneously escalated ecological exposure to plethoras of hazardous substances which alters body's finely tuned hormonal balance and is thus regarded as endocrine disruptors. Bisphenol-S (BPS), earlier believed to be a safe substitute of bisphenol A (BPA) is utilized in making polyethersulfone plastics (PES), epoxy resins, food packaging, dye developer in thermal receipts, and other daily use products. BPS has been detected in environmental matrices as well as human samples including serum, urine, cord blood, placenta, follicular fluid and breastmilk indicating its systemic persistency for the tissues/organs and is thus exposing us at various stages of life from pre-conception to ageing. Melatonin, a pleotropic indoleamine hormone secreted from the pineal gland as well as extra-pineal sites has multifaceted biological roles including anti-apoptotic, anti-inflammatory, immunomodulatory and a potent antioxidant. We investigated the molecular basis of deleterious effects of BPS exposure on reproductive health and protective actions of melatonin supplementation on restoration of fertility.

YOUNG SCIENTISTS PRESENTATIONS

YS-1	**Soumyajyoti Ghosh** *Visva-Bharati University, Santiniketan, West Bengal* **Molecular Insights into Zebrafish Ovulation: Perspectives on Redox Homeostasis, Inflammatory Mediators, and Mitochondrial Bioenergetics**
YS-2	**Sonika Kar** *University of Hyderabad* **Molecular Insights into Ad4bp/Sf-1 Mediated Regulation of Steroidogenesis in Catfish**
YS-3	**Neha Kaushik** *AIIMS, New Delhi* **Reduced Insulin/IGF-1 Signaling Extends Reproductive Span through Somatic Gonadal Collagen-Mediated Maintenance of Oocyte Quality in *Caenorhabditis elegans***
YS-4	**Gauri Bhonde** *ICMR-NIRRCH, Mumbai* **Maternal Plasma Human Cytomegalovirus (HCMV) DNA Positively Correlates with Viral Shedding and Altered Microbiome Composition in Breast Milk**
YS-5	**Bodhana Dhole** *AIIMS, New Delhi* **Regulatory Functions of Circular RNAs in Prostate Cancer Metastasis**

MOLECULAR INSIGHTS INTO ZEBRAFISH OVULATION: PERSPECTIVES ON REDOX HOMEOSTASIS, INFLAMMATORY MEDIATORS, AND MITOCHONDRIAL BIOENERGETICS

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A pre-requisite for successful fertilization and embryonic development, ovulation is an intricate physiological phenomenon that involves the liberation of mature, metaphase II-arrested oocytes from their surrounding follicle layer into the ovarian cavity. The present study investigates the dynamic changes in the follicular microenvironment preceding ovulation in zebrafish, focusing on redox balance, inflammation, and mitochondrial bioenergetics during the natural ovulation process triggered by morning illumination. Our findings reveal that increased expression of key ovulatory mediators (lhcgr, star, 20β-hsd, pgr, pla2g4aa, ptgs2a/b, ptgesl, ptger4b) is accompanied by nuclear translocation of PGR, elevated free radicals, reduced antioxidant activity, heightened inflammatory mediators, and matrix metalloproteinase activity, leading to follicular membrane dissolution and fertilization membrane formation. Mechanistically, an elevated ovulatory response in hCG-treated FG follicles in vitro involves accumulation of superoxides, altered antioxidant machinery, upregulation of inflammatory mediators, pgr, and ovulation-associated genes in a manner sensitive to PKA- and MAPK3/1-mediated signalling. Intriguingly, as oocyte maturation progresses, a metabolic shift from glycolysis to oxidative phosphorylation occurs to meet the demands for ATP and support meiotic cell cycle progression. Congruent with a sharp increase in beta-oxidation related markers (CPT1A, ACOX1, ACAD9, PPARα), a remarkable increment in ATP and citrate synthase levels, along with altered mitochondrial membrane polarization (J-aggregate formation) in pre-ovulatory follicles, indicates heightened mitochondrial activity before ovulation. Strikingly, an upregulated expression of mitochondrial DNA copy number regulators (TFAM, NDI, COX I/II), respiratory chain subunits (Complex I-V) and transcription factors (PGC1α/β, ERRα, NRF-1, HSP60) correlate well with modulated mitochondrial fission (DRP-1, FIS-1)/fusion (OPA-1, MFN-1/2) mediators and energy sensors (SIRT1, AMPKα, PGC1α/β) before spawning, underscoring a critical role of mitochondrial energy homeostasis in zebrafish oocytes before ovulation.

MOLECULAR INSIGHTS INTO AD4BP/SF-1 MEDIATED REGULATION OF STEROIDOGENESIS IN CATFISH

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Steroidogenic enzyme genes play a pivotal role in vertebrate reproduction by regulating androgen biosynthesis, yet their transcriptional regulation remains less explored. This study elucidates the molecular regulation of 11β-hydroxylase (cyp11b1), a key enzyme in androgen production, in the Asian catfish, *Clarias batrachus*. The 5′ upstream region of cyp11b1 was cloned and analysed to identify potential transcription factor binding sites. Promoter activity was characterized using progressive deletion constructs and luciferase reporter assays, followed by site-directed mutagenesis and chromatin immunoprecipitation to confirm transcription factor binding. Results demonstrated that Ad4bp/Sf-1 and Foxp1 directly bind to the cyp11b1 promoter and regulate its transcription. Expression analysis revealed, ad4bp/sf-1 is highly expressed during the spawning phase and is upregulated by gonadotropin induction (hCG) and exogenous androgen treatment (methyl testosterone and 11-ketotestosterone). Ad4bp/Sf-1 was primarily localized in the interstitial layer/ Leydig cells, and also in spermatogonia and spermatocytes through immunohistochemistry. Transient gene silencing of ad4bp/sf-1 via siRNA treatment significantly reduced cyp11b1 expression, altered the expression of other steroidogenic enzyme genes and associated transcription factors, and lowered serum androgen levels, confirming its central role in testicular steroidogenesis. Collectively, these findings identify Ad4bp/Sf-1 as a crucial regulator of cyp11b1 transcription in catfish. This study provides novel insights into the transcriptional mechanisms governing androgen biosynthesis in teleosts and establishes a base for future investigations on the molecular regulation of fish reproduction.

REDUCED INSULIN/IGF-1 SIGNALING EXTENDS REPRODUCTIVE SPAN THROUGH SOMATIC GONADAL COLLAGEN-MEDIATED MAINTENANCE OF OOCYTE QUALITY IN *CAENORHABDITIS ELEGANS*

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The insulin/IGF-1 signaling (IIS) pathway is a key regulator of reproductive longevity, influencing germline development and oocyte quality. However, how IIS maintains the somatic gonad-critical for supporting oocyte maturation remains unclear. This study investigates the impact of reduced IIS on somatic gonadal integrity during reproductive aging in *C. elegans*. Distal gonads from wild-type and reduced IIS model animals were dissected at four defined time points and subjected to bulk RNA sequencing in triple biological replicates. Whole-worm samples were also sequenced at two time points for comparison. IIS-responsive genes were stratified into gonadal versus non-gonadal expression, and integrated with existing germline and gonad-specific transcriptomic datasets to assess spatial and temporal expression patterns. Functional analysis via systemic and gonad-specific RNA interference (RNAi) identified several collagen genes upregulated under reduced IIS. Knockdown of these collagens accelerated reproductive decline, compromised oocyte morphology, and led to deterioration of somatic gonadal structure-effects dependent on the IIS effector FOXO/DAF-16. Notably, these phenotypes occurred without a marked reduction in total reproductive output, indicating a role in structural maintenance rather than fertility per se. Differential interference contrast microscopy and qPCR confirmed temporal collagen regulation and its transcriptional control by IIS-regulated factors. Comparative analyses revealed significant overlap between gonadal and whole-worm expression, with distinct age-dependent patterns. Our findings identify somatic gonadal collagens as novel IIS-regulated effectors essential for maintaining gonadal integrity and reproductive function with age. This work provides mechanistic insight into reproductive aging and highlights potential molecular targets to mitigate age-related reproductive decline.

MATERNAL PLASMA HUMAN CYTOMEGALOVIRUS (HCMV) DNA POSITIVELY CORRELATES WITH VIRAL SHEDDING AND ALTERED MICROBIOME COMPOSITION IN BREAST MILK

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Human cytomegalovirus (HCMV) is a prevalent β-herpesvirus capable of congenital and postnatal transmission, predominantly through breastfeeding. However, the relationship between systemic maternal viremia and viral shedding into breast milk as well its impact of breast milk microbiome composition remains poorly understood.

Paired plasma and breast milk samples from 21 lactating mothers were analyzed for HCMV DNA using Nested polymerase chain reaction (PCR). Concurrently, 16S rRNA gene sequencing was employed to characterize the milk microbiome and assess viral–microbial associations.

A strong concordance was observed between plasma and milk positivity 12 participants with detectable plasma HCMV DNA consistently exhibited viral DNA in breast milk, whereas 9 plasma-negative participants showed no viral DNA in corresponding milk samples. Microbiome profiling revealed compositional variations between HCMV-positive and -negative samples. Firmicutes remained the dominant phylum in both groups (58.33% vs. 54.53%), while Bacteroidetes were slightly elevated in HCMV-positive milk (36.02% vs. 32.15%). Actinobacteria (0.97% vs. 1.58%) and Proteobacteria (1.32% vs. 5.90%) were reduced in HCMV-positive samples, suggesting viral-associated modulation of microbial homeostasis.

Maternal plasma HCMV DNA positivity strongly correlates with viral shedding into breast milk, implicating systemic infection in mother-to-infant viral transfer. Associated microbiome alterations suggest a potential viral–microbial interplay that may influence infant gut colonization and immune development, underscoring the need for integrated viral and microbiome monitoring during lactation.

YS-5

REGULATORY FUNCTIONS OF CIRCULAR RNAS IN PROSTATE CANCER METASTASIS

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Screening, diagnostic and therapeutic options for prostate cancer remain limited, emphasizing the need to identify novel molecular targets. Circular RNAs (circRNAs) are circular, tissue-specific, single-stranded RNAs whose dysregulated expression has been reported in various cancers. In-silico analyses have shown that hsa_circ_0085494 and hsa_circ_0031408 are upregulated in prostate cancer tissues compared to normal tissues. This study investigated the expression of these circRNAs across different stages of prostate cancer to understand their role in disease progression. The study also assessed their ability to distinguish prostate cancer from benign prostatic hyperplasia (BPH). Serum and tissue samples were collected from patients with BPH (n=24) and primary (n=27), locally advanced (n=11), and metastatic (n=21) prostate cancer. Total RNA was isolated and expression of circRNA was quantified by qRT-PCR. CircRNAs work primarily by binding and blocking the functions of their partner miRNAs. Potential miRNA binding partners were predicted using CircInteractome and corresponding miRNA levels were also measured. Expression of hsa_circ_0085494 was significantly higher in metastatic tissues and sera compared to primary and locally advanced prostate cancer. hsa_circ_0031408 showed a similar pattern in tissues but not in serum. Compared with BPH, hsa_circ_0085494 was markedly upregulated in prostate cancer, while hsa_circ_0031408 showed no significant difference. miR-330-3p, predicted as a target of hsa_circ_0085494, showed no significant variation among groups. Increased expression of hsa_circ_0085494 in prostate cancer and its higher levels in metastatic cases suggest its potential as a screening biomarker and regulator of metastasis. Both circRNAs may serve as promising molecular targets to limit prostate cancer progression.

POSTER PRESENTATIONS: COMPETITIVE

PP-C-1	**Garima Vats** *University of Hyderabad* **Identification of Key Genes during Early-Stage Testicular Development in Catfish**
PP-C-2	**Rutvi Vaja** *Navrachana University, Vadodara* **Neuroprotective Effects of Melatonin Co-Administration against Aluminium Chloride mediated Chronic Toxicity in a Pharmacologically Induced Adult Zebrafish (*Danio rerio*) Model of Alzheimer's Disease**
PP-C-3	**Shivshankar Maurya** *Institute of Science, BHU, Varanasi* ***Phyllanthus niruri* Extract Mitigates Cafeteria Diet Induced Metabolic Alterations and Adipose Tissue Function in Male Mice**
PP-C-4	**Arun Roy** *University of North Bengal, Darjeeling* **Role of Gut Bacteria and Melatonin In Mediating Better Digestive Physiology and Growth of *Cirrhinus Mrigala***
PP-C-5	**Anusree K V** *Central University of Kerala* **Evaluation of Age and Sex Dependent Transcriptional Changes of Genes Associated with Hypothalamic-Pituitary–Gonadal (HPG) Axis in Asian Seabass (*Lates calcarifer*, Bloch, 1790)**
PP-C-6	**Lakshmi K Nalinan** *Central University of Kerala* **Investigating Age Dependent Differential Gene Expression Profile of Hypothalamic-Pituitary-Interrenal (HPI) Axis in male Asian Seabass (*Lates calcarifer*, Bloch 1790)**
PP-C-7	**Bhawna Kumari** *University of Lucknow* **Age-Dependent Novel Expression of Vaspin and its Receptor GRP78 in the Testis of Mice at Different Stages of Postnatal Development in Mice**
PP-C-8	**Bini Kanyal** *AIIMS, New Delhi* **Investigating the Effect of Serotonin on Trophoblast Functions: An in Vitro Study**
PP-C-9	**Sripriya Bulusu** *Navrachana University, Vadodara*

	Early-Life Macronutrient Exposure Alters Pubertal Timing through Hypothalamic microRNA Networks
PP-C-10	**Poulomi Sarkar** *CSIR-IICB, Kolkata* **Decoding the Role of Epigenetic Modifiers and Trophoblast-Exosomes in Vascular Smooth Muscle Cell Plasticity and Spiral Artery Remodeling**
PP-C-11	**Mahamadtezib Khatri** *The Maharaja Sayajirao University of Baroda* **Desynchrony of miR-122-SREBP1-PPARα Oscillations in Chronodisruption and its Implication in MASLD**
PP-C-12	**Bhumika Patel** *The Maharaja Sayajirao University of Baroda* **Melatonin Alleviates Anxiety and Depression by Reducing Hippocampal Inflammation in a Model of Diet and Circadian Disruption**
PP-C-13	**Helly Shah** *The Maharaja Sayajirao University of Baroda* **Carbon Monoxide Disrupts KHSRP–6PGD Coupling to Restore miR-34a Balance and Lipid Homeostasis in Hepatocytes**
PP-C-14	**Mahima Sharma** *AIIMS, New Delhi* **Impact of Elevated Estrogen-to-Androgen Ratio on Primary Mouse Prostate Cells**
PP-C-15	**Drashti Mehta** *Navrachana University, Vadodara* **Deciphering the Role of Kisspeptin - Paclitaxel Combination in Modulating Adhesion and Tumor Microenvironment in Triple Negative Breast Cancer**
PP-C-16	**Souparnika B.R** *Dr. ALM Post Graduate Institute of Basic Medical Sciences, Chennai* **Targeting the ALDH1A1 with Pharmacological Inhibitor A37 Suppresses Migration and Induces Death of Metastatic Cervical Cancer Cells**
PP-C-17	**Rushigandha Salunke** *ICMR-NIRRCH, Mumbai* **Dynamic Changes of TLR9 Across Progressive Stages of Triple-Negative Breast Cancer**
PP-C-18	**Ananya Breed** *ICMR-NIRRCH, Mumbai* **Trop2 Drives Aggressive Traits and Spheroid Formation Linked to Poor Survival in High-Grade Serous Ovarian Cancer**

PP-C-19	**Junita Desouza** *ICMR-NIRRCH, Mumbai* **GPER1 (G Protein-Coupled Estrogen Receptor 1) Activation: A Novel Strategy for Chemoprevention of Prostate Cancer**
PP-C-20	**Soumya Rastogi** *AIIMS, New Delhi* **To elucidate the role of Tacedinaline in Reproductive Competence Using *C. elegans* as Model System**
PP-C-21	**Apoorva Pawar** *ICMR-NIRRCH, Mumbai* **Distinct Temporal Regulation of Trop2 and EpCAM Expression in Rat and Human Placenta**
PP-C-22	**Khushi Gupta** *AIIMS, New Delhi* **Endometriosis and Circulating MicroRNAs: A Detailed Analysis and Validation of Reported Biomarkers in the Indian Population**
PP-C-23	**Anamika Gupta** *AIIMS, New Delhi* **Effect of CPT1 Inhibition Mediated Endoplasmic Reticulum Stress on Calcium Oscillations in Mice Oocytes**
PP-C-24	**Millo Kanya** *AIIMS, New Delhi* **Effect Of Sperm Cryopreservation on Mitochondrial Transferase Expression and Sperm Motility**
PP-C-25	**Neha Choudhari** *Department of Biotherapeutics Research, MAHE, Manipal* **Role of GSK3α in Regulating the RNA Demethylase FTO during Spermatogenesis in Mice**
PP-C-26	**Silpa K G** *Amala Cancer Research Centre, Thrissur, Kerala* **Consequence of Dysregulated Glycolipid Metabolism in Sertoli Cells and its Impact on Spermatogenesis**
PP-C-27	**Aayushi Taneja** *AIIMS, New Delhi* **Exploring the Role of DNA Damage and Alternative Lengthening of Telomeres Pathways in Sperm Telomere Elongation with Paternal Aging**
PP-C-28	**Sonali Kumari Singh** *AIIMS, New Delhi*

	BPA and Heavy Metal–Driven Epigenetic Alterations in Idiopathic Hypospermatogenesis
PP-C-29	**Deepshikha Arya** *ICMR-NIRRCH, Mumbai* **Dynamics of Core and Modified Histones in Sperm of Infertile Men with Clinical Varicocele: Pre- and Post-Treatment**
PP-C-30	**Roshan Dadachanji** *ICMR-NIRRCH, Mumbai* **Delineating the Hemostatic Signature of Indian Women with Polycystic Ovary Syndrome**
PP-C-31	**Manisha Kumari** *ICMR-NIRRCH, Mumbai* **Lipidomic Profiling Revealed Alterations in Lipid Metabolic Landscape in Follicular Microenvironment in Women with PCOS**
PP-C-32	**Medini Samant** *ICMR-NIRRCH, Mumbai* **Exploring the Role of Vitamin D Pathway Genetic Variants in Susceptibility to Polycystic Ovary Syndrome**
PP-C-33	**Komal Khade** *ICMR-NIRRCH, Mumbai* **Integrative Metagenomic and Metabolomic Analysis to Elucidate the Role of Gut Microbiota in the Pathophysiology of PCOS**
PP-C-34	**Madhanraj Akilandeswari Pugalendhi** *Vellore Institute of Technology, Vellore* **Propionic Acid Attenuates Metabolic Dysfunctions in PCOS Rat Model**
PP-C-35	**Deepanjali Ghadge** *ICMR-NIRRCH, Mumbai* **Endometrial Receptivity and Implantation rate in Endometriosis: Are They Affected Adversely?**
PP-C-36	**Vaibhavi Srivastava** *Gujarat University, Ahmedabad* **Endometrial Gene Expression Signature of Inflammatory Pathway and its Role on ART Outcomes in Indian Women.**
PP-C-37	**Priyal Mehta** *The Maharaja Sayajirao University of Baroda* **Understanding the Lipidemic Milieu of Spleen in PCOS Rodent Model**
PP-C-38	**Vivek Kumar** *AIIMS, New Delhi*

	Deciphering Steroid Biosynthesis Pathway Gene Variants in PCOS Phenotype A with Elevated 17-OHP
PP-C-39	**Bindusha Das** **Navrachana University** **Studying the Influence of Melatonin and Probiotic Metabolites in a Hyperandrogenic in vitro Environment**
PP-C-40	**Annis Inncia Gnana Thiravaim** *Vellore Institute of Technology, Vellore* **Illuminating the Differential Regulation of PPAR-γ and Aromatase by Pharmacotherapeutics in *Danio rerio* Ovaries: Implications for PCOS Intervention**
PP-C-41	**Kajal Sihag** *AIIMS, New Delhi* **Mitochondria-Induced Endoplasmic Reticulum Stress: A Potential Mechanistic Link to Idiopathic Premature Ovarian Insufficiency**
PP-C-42	**Bipradip Saha** *ICMR-NIRRCH, Mumbai* **Evaluating the Effect of Antioxidants in Endocrine Disrupters and High-Fat Diet Induced Polycystic Ovary Syndrome (PCOS) like Condition**
PP-C-43	**Mousumi Bal** *ICMR-NIRRCH, Mumbai* **Comparative Transcriptomic Analysis of SUSD2^{+} Endometrial Mesenchymal Stem Cells and Whole-Tissue Lesions in Endometriosis**

PP-C-1

IDENTIFICATION OF KEY GENES DURING EARLY-STAGE TESTICULAR DEVELOPMENT IN CATFISH

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Teleost fishes exhibit a diverse range of reproductive strategies leading to species specific sex determination system. Depending on the vast range of teleost species, sex determination and differentiation mechanisms vary in their sexual patterns; therefore, sexual plasticity in gonadal development is inevitable through a phenomenon referred to as gonadal transdifferentiation. The present research aims to identify novel factors/genes during sex steroid induced gonadal transdifferentiation in catfish. Catfish (*Clarias magur*) larva around 15 to 20 days post hatchlings obtained by in vitro fertilization were utilized for experimentation. Treatments of sex steroids (methyl testosterone (MT) ,150 µg/L each, continuously for 15 days was carried out after hatching. Mesonephric gonadal complex (sexually undifferentiated / differentiating gonads) was dissected out from control and treated fish at 35 dph for transcriptome analysis to study differentially expressed genes related to gonadal transdifferentiation. Compared to control, the following genes/factors, including, tbx21, star, bmp2, creb, fgf11, foxn3, amh, tph1b, wnt7b, sox3, bmpr2a, gfra3 were found to be significantly upregulated upon MT treatment, while foxn3, tns3, znf34 and nf4a2b were found to be significantly downregulated during the crucial window of gonadal transdifferentiation in C. magur. The expression pattern was validated through qPCR analysis. Histology analysis depicted traces of ovotestis in the MT-treated fish at 150 dph as compared to only testis/ovary in control fish. Overall, this study highlighted several novel genes related to teleostean gonadal transdifferentiation in catfish.

PP-C-2

NEUROPROTECTIVE EFFECTS OF MELATONIN CO-ADMINISTRATION AGAINST ALUMINIUM CHLORIDE MEDIATED CHRONIC TOXICITY IN A PHARMACOLOGICALLY INDUCED ADULT ZEBRAFISH (*DANIO RERIO*) MODEL OF ALZHEIMER'S DISEASE

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Emerging evidence implicates the brain–gut axis and sex-specific biological factors as key determinants of Alzheimer's disease (AD) progression and therapeutic response. Although melatonin exhibits potent antioxidant and neuroprotective properties, its efficacy across brain and gut systems in classic experimental in vivo model systems, particularly using sex as a biological variable, remain underexplored. The study hypothesized that melatonin provides protection against $AlCl_3$-induced oxidative stress, cholinergic dysfunction, and gut-brain axis disturbances, thereby attenuating AD-like pathology. Adult male and female zebrafish(ZFs) were co-administered with chronic melatonin and $AlCl_3$ treatment for 28 days. Post-treatment analyses included histopathological evaluation of brain and gut tissues, quantification of oxidative stress in brain and gut, cholinergic activity, assessment of behavioural parameters, measure of cognitive impairments, locomotor activity, and anxiety-like responses. Melatonin co-administration, restored antioxidant defenses with elevated CAT and GSH activity and reduced LPO levels. Cholinergic activity increased and suppression of elevated AChE activity. Behavioural impairments, including memory deficits, locomotor abnormalities, and anxiety-like behaviours, were significantly ameliorated. Histopathological analyses confirmed substantial protection against neuronal vacuolization, gliosis, astrocytic dysmorphology, and gut epithelial damage. Moreoever, females exhibited more pronounced improvements across biochemical, behavioural, and histological parameters, highlighting sex-dependent therapeutic efficacy. These findings demonstrate that melatonin exerts potent multi-targeted neuroprotective and enteroprotective effects in a pharmacologically induced experimental AD ZF model, with stronger efficacy in females. This underscores the translational potential of melatonin and emphasizes the need for sex-stratified approaches in future preclinical and clinical studies to optimize precision-based interventions for AD.

PP-C-3

PHYLLANTHUS NIRURI EXTRACT MITIGATES CAFETERIA DIET INDUCED METABOLIC ALTERATIONS AND ADIPOSE TISSUE FUNCTION IN MALE MICE

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Obesity is a chronic and multifactorial disease characterized by excessive body fat that poses significant health risk, including increased incidence of type 2 diabetes and cardiovascular disease. Based on the available reports *Phyllanthus niruri* acts as hepato-tonic that effectively ameliorates hepatic disturbances caused by calorie rich diet in mice. However, there exist an acute scarcity of reports that addresses the effect of *Phyllanthus niruri* on adipose tissue function and whole-body metabolic physiology. The present study aims to study the protective effect of hydro-alcoholic extract of *Phyllanthus niruri* on the cafeteria diet-induced obesity in male C57BL/6 mice. The mice were randomly divided into four groups (n=6/group) namely, control, cafeteria diet (CD), *Phyllanthus niruri* extract (PEx) and CD+ PEx. The mice were fed with regular chow diet/cafeteria diet and drinking water ad libitum for a period of 8 weeks. The oral dose of PEx (200mg/kg) was administered from week three onwards till eighth week. The food intake, water and sucrose consumption was recorded every day, while body weight was recorded every week. *P. niruri* treatment significantly reduced the body weight gain, fasting glucose, and serum triglyceride and cholesterol levels compared to the untreated CD fed obese mice. The histological examination showed reduced lipid accumulation in liver and reduced adipocyte hypertrophy, indicating improved lipid/nutrient handling. The molecular markers of lipogenesis and lipolysis showed significant improvement in PEx treated group compared to CD fed mice. The present study demonstrates that *P. nirurii* extract effectively mitigates metabolic alterations associated with consumption of cafeteria diet by improving glucose homeostasis, lipid metabolism and adipose tissue function.

PP-C-4

ROLE OF GUT BACTERIA AND MELATONIN IN MEDIATING BETTER DIGESTIVE PHYSIOLOGY AND GROWTH OF *CIRRHINUS MRIGALA*

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The present study evaluated growth performance, feed efficiency, gut bacterial abundance, and segmental variations in melatonin levels, digestive enzyme activities, anti-oxidative agents, and oxidative stress parameters within the gut of *Cirrhinus mrigala*, following the administration of probiotics and synbiotics. Fingerlings of *C. mrigala* (average weight: 27±0.29 g) were randomly distributed into three distinct dietary treatment groups in triplicate: (a) a standard diet (SD/control); (b) a probiotics-rich diet (PBRD); and (c) a synbiotics-rich diet (SBRD). Two probiotic strains, *Bacillus subtilis* (KX269836) and *Bacillus sonorensis* (KF623291), were incorporated into the standard diet at a concentration of 1×10^9 cfu/g to formulate both the PBRD and SBRD, with fructooligosaccharide included as a prebiotic (0.6%, w/w) in the SBRD formulation.

The study involved a 120-day feeding trial, during which growth performance and feeding metrics were documented, followed by the analysis of gut tissue samples. The results indicated that the SBRD group exhibited the highest weight gain, improved digestion, and increased bacterial abundance compared to the other groups. The group fed PBRD demonstrated intermediate results, while the SD group yielded the lowest outcomes. Although no significant segment-wise variations in melatonin levels were detected in the control group, significantly higher melatonin concentrations were identified in the anterior intestine (AI) and posterior intestine (PI) of the SBRD-, and PBRD-fed group. Additionally, the SBRD-fed group exhibited the most elevated enzyme activities, with moderate activities noticed in the PBRD-fed group. Dietary interventions of synbiotics and probiotics resulted in reduced oxidative stress and enhanced antioxidant capacity. Remarkably, throughout all examined parameters, synbiotics consistently proved to be more efficacious than probiotics alone. Collectively, these findings suggest a significant correlation between gut microbiota and melatonin production, indicating that combined dietary stimulators may play a crucial role in enhancing digestive efficacy and mitigating gut stress, ultimately contributing to improved growth outcomes.

PP-C-5

EVALUATION OF AGE AND SEX DEPENDENT TRANSCRIPTIONAL CHANGES OF GENES ASSOCIATED WITH HYPOTHALAMIC-PITUITARY–GONADAL (HPG) AXIS IN ASIAN SEABASS (*LATES CALCARIFER*, BLOCH, 1790)

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Asian Seabass (*Lates calcarifer*) is a sequential protandrous hermaphrodite teleost. It initially matures as a male (< 2 years) and subsequently (> 3-4 years) changes its sex to female. Like most vertebrates, reproduction in fishes is primarily governed by the hypothalamic–pituitary–gonadal (HPG) axis. Notably, the function of the HPG axis also gets modulated by multiple intrinsic and extrinsic factors, including germ cell number, temperature, salinity and stress etc. In this study, we have investigated the sex and age specific transcriptional differences of some of the key HPG-axis genes (viz., Kiss1, Kiss2, Kissrα, kissrβ, Gnrh1, Gnrh2, Gnrh3, Gnrhr, Lhβ, Fshβ, Fshr, Lhr, Ar, and Er1) in *L. calcarifer*. Brain and gonadal tissues from juvenile (< 1 year) and adult fishes [males (≥ 2 years) and females (≥ 4 years)] were examined. Our data revealed that Kiss1, Kissrα, Gnrh1, Gnrh3 and Fshβ mRNAs were elevated in the juvenile male brain, whereas Kiss2, Kissrβ, Gnrh2, Gnrhr and Lhβ mRNAs were predominantly expressed in the adult male brain. On the other hand, Fshr, Er1 and Ar mRNAs were highly expressed in juvenile testes, while Lhr expression was found to be enriched in the posterior brain of adult males. Furthermore, a distinct sexually dimorphic transcriptional pattern of gonadotropin receptors was observed indicating sex-specific differential regulation of reproductive signalling. These findings provide novel insights into the developmental and sex-dependent regulation of the HPG axis in a marine teleost, with potential future implications for critical understanding of the breeding biology of this species.

PP-C-6

INVESTIGATING AGE DEPENDENT DIFFERENTIAL GENE EXPRESSION PROFILE OF HYPOTHALAMIC-PITUITARY-INTERRENAL (HPI) AXIS IN MALE ASIAN SEABASS (*LATES CALCARIFER*, BLOCH, 1790)

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Asian Seabass (*Lates calcarifer*) is predominantly found in coastal-brackish water bodies of the Indo-Pacific region. In India, *L. calcarifer* is commercially cultured in coastal aqua-farms due to its fast growth rate, ability to tolerate varying salinity levels and high marketvalue. However, high stocking density is a crucial stress factor that limits the productivity ofthese aqua-farms. Such stressful conditions lead to prolonged elevation of plasma cortisol levels resulting sex-specific disruption of the steroidogenic activity necessary for gametogenic development affecting gonadal output. The hypothalamic–pituitary–interrenal (HPI) axis plays a central role in stress regulation in teleosts by maintaining internal homeostasis through cortisol signalling. In this study, we have examined the transcriptional profiles of key HPI-axis genes (Crh, Acth, Acthr, Gr) in L. calcarifer. The brain regions (anterior, mid, posterior) and interrenal tissues were examined in juvenile (< 1 year) and adult (2-3 years) male fishes. Our data demonstrated an elevated Acthr mRNA expression in the posterior brain of adults compared to juveniles, whereas Gr expression was found to behigher in the posterior brain of juveniles. Notably, no age-related variation was observed in Acth expression. Collectively, our findings highlight the developmental differences of HPI-axis gene expression profile that can be manipulated for stress management in aqua-farms.

PP-C-7

AGE-DEPENDENT NOVEL EXPRESSION OF VASPIN AND ITS RECEPTOR GRP78 IN THE TESTIS OF MICE AT DIFFERENT STAGES OF POSTNATAL DEVELOPMENT IN MICE

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Vaspin (visceral adipose tissue-derived serine protease inhibitor), a serpin family adipokine, was initially identified in visceral adipose tissue and later recognized for its metabolic and anti-inflammatory effects across organs. Its biological activity is primarily mediated through binding with the 78-kDa glucose-regulated protein (GRP78). Although the vaspin–GRP78 interaction modulates several signaling pathways, its role in the male reproductive system remains unclear. Therefore, we aimed to investigate the expression pattern and potential role of vaspin and GRP78 in the mouse testis across different stages of post-natal development. Firstly, we performed immunohistochemistry to localize vaspin and GRP78 in the testis of neonatal, pre-pubertal, pubertal, adult, and aged mice. Vaspin expression was minimal in neonatal and pre-pubertal stages, peaked at puberty, then declined in adults, with a marked decrease in aged mice. GRP78 showed a gradual increase from neonatal to adult stages, followed by a sharp decline in aged mice. These results were supported by RT-PCR and immunoblotting. We also measured the testosterone in serum by ELISA and performed its correlation study with vaspin and GRP78 expression in the testis. The reduced expression in aged testes correlated with morphological degeneration and decreased functional activity, indicating potential involvement of the vaspin–GRP78 axis in maintaining testicular integrity and metabolic balance during aging. Therefore, these findings highlight vaspin as a novel metabolic regulator in testicular physiology and suggest its decline may contribute to age-associated testicular dysfunction.

PP-C-8

INVESTIGATING THE EFFECT OF SEROTONIN ON TROPHOBLAST FUNCTIONS: AN IN VITRO STUDY

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Serotonin (5-hydroxytryptamine) functions not only as a central neurotransmitter but also as an important signalling molecule influencing endocrine and developmental processes in early embryogenesis. Existing evidence suggests that the placental serotonergic system, including MAOA and 5-HT receptors, modulates trophoblast activity. However, direct effects of serotonin on trophoblast functions have not been documented. In the present study, we investigated how serotonin affects trophoblast proliferation, migration, and hormone production. Human trophoblast JEG-3 cells were cultured in DMEM containing 10% Fetal Bovine Serum and exposed to serotonin concentrations ranging from 0.01 μM to 10 μM for different time intervals. Cell proliferation was assessed by MTT assay, and cell migration was evaluated by a wound healing assay. To determine endocrine effects, levels of hCG, progesterone, and estradiol in the culture media were measured using specific immunoassays. Understanding the effect of serotonin on placental functions will help in deciphering the mechanisms controlling normal physiology of pregnancy.

PP-C-9

EARLY-LIFE MACRONUTRIENT EXPOSURE ALTERS PUBERTAL TIMING THROUGH HYPOTHALAMIC MICRORNA NETWORKS

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Pubertal timing is modulated by early-life nutrition through epigenetic mechanisms that remain incompletely understood. This study investigated how macronutrient-specific diets influence hypothalamic microRNA (miRNA) expression and pubertal onset in female Wistar rats. Animals were exposed to various diets including, High-fat (HFD), High-carbohydrate (HCD), High-protein (HPD), and Cafeteria diets (CafD) from postnatal day 21-42. Followed by a comprehensive analysis of phenotypic markers, hormone levels, ovarian histology, and hypothalamic microRNA (miRNA) transcriptomics. Results suggested that energy-dense diets (HFD, HCD) significantly advanced vaginal opening by 4-5 days compared to controls and increased body weight, serum Luteinizing Hormone (LH), Follicle Stimulating hormone (FSH), and Estradiol levels compared to control. Small RNA sequencing revealed extensive miRNA reprogramming, with over 470 differentially expressed miRNAs in each dietary group. Key findings included upregulation of miR-30b (targeting Mkrn3, a pubertal inhibitor), downregulation of miR-199 (targeting Kiss1, a pubertal activator), and altered expression of let-7 family miRNAs affecting developmental timing genes. Quantitative PCR validation confirmed inverse relationships between regulatory miRNAs and their target mRNAs involved in HPG axis control. In-silico structural modelling supported the thermodynamic stability of predicted miRNA-mRNA interactions. Functional enrichment analysis revealed convergence on GnRH signalling, MAPK pathways, and neuroendocrine regulation. These findings suggest that early nutritional environments may influence hypothalamic miRNA networks, potentially contributing to long-term neuroendocrine modulation.

PP-C-10

DECODING THE ROLE OF EPIGENETIC MODIFIERS AND TROPHOBLAST-EXOSOMES IN VASCULAR SMOOTH MUSCLE CELL PLASTICITY AND SPIRAL ARTERY REMODELING

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Uterine spiral artery remodelling (SAR) is critical for proper fetal development, relying on the trophoblast-mediated phenotypic switching of vascular smooth muscle cells (VSMCs) from a contractile to a synthetic state. Impaired SAR contributes to conditions such as intrauterine growth restriction (IUGR) and maternal-fetal morbidity. This phenotypic switch likely involves epigenetic modifications, regulated by "writers," "erasers," and chromatin remodelling factors. Co-culture of E16.5 rat primary trophoblast cells with A7r5 VSMCs induced VSMC dedifferentiation, marked by altered genotypic markers. As expected, Trophoblast-co-cultured VSMCs elicited alterations in the expression of epigenetic "writers" and "erasers." This was accompanied by downregulation of 13 chromatin remodelling genes, confirmed by qPCR arrays. Similar gene suppression was observed in the E16.5 rat metrial gland, an entry point of uterine spiral arteries, in vivo. Furthermore, antisense oligonucleotide-mediated knockdown of chromatin remodelling genes inhibited the phenotypic transition of VSMCs, confirming their regulatory role. Notably, in the metrial gland of IUGR rats, chromatin remodelling factors and VSMC markers showed reciprocal expression patterns indicating their role in in VSMC plasticity. We further showed that blocking exosome release from trophoblasts reduced VSMC plasticity. Uptake of exosomes by VSMCs were also confirmed. Next-generation sequencing of small RNAs from trophoblast-derived exosomes identified 238 miRNAs, reinforcing their role as functional cargo. In conclusion, our findings highlight that trophoblast exosome-mediated epigenetic regulation of VSMCs is essential for SAR. Disruption of this cell-to-cell communication may underlie pregnancy complications such as IUGR. [This work was supported by grants from Department of Biotechnology, BT/PR49910/MED/97/642/2023]

PP-C-11

DESYNCHRONY OF MIR-122-SREBP1-PPARA OSCILLATIONS IN CHRONODISRUPTION AND ITS IMPLICATION IN MASLD

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MicroRNA-122 (miR122) is liver specific and a key post-transcriptional regulator of genes governing of lipid metabolism and circadian homeostasis. Dysregulation of miR-122 has been implicated in metabolic-dysfunction associated steatotic liver disease (MASLD); however, its rhythmic interplay with major lipid regulators such as SREBP1 and PPARα under chronodisruptive conditions remains unexplored. We hypothesize that aberrant oscillations of hepatic miR-122 underlie the dampening of SREBP1 and PPARα rhythmicity, thereby aggravating lipid dyshomeostasis in MASLD. C57BL/6J mice were subjected to photoperiod manipulation-induced chronodisruption (CD), high fat-high fructose diet (H), and their combination (HCD) for 18 weeks. Liver and serum samples were collected at 6-hour Zeitgeber intervals (ZT0–ZT24) to assess rhythmic expression of miR-122, SREBP1, and PPARα. Preliminary findings reveal disrupted temporal patterns of miR-122 in H, CD, and HCD groups, coinciding with attenuated oscillations of SREBP1 and PPARα transcripts. Notably, increased hepatic miR-122 expression at ZT12 corresponded with reduced PPARα and elevated SREBP1 levels, suggesting reciprocal regulation. These data indicate that aberrant miR-122 rhythmicity contributes to impaired lipid regulatory cycles by repressing PPARα-mediated β-oxidation and promoting SREBP1-driven lipogenesis. The observed desynchrony between miR-122 and its metabolic targets under chronodisruptive and dietary stress provides mechanistic insight into miR-122–clock–lipid axis dysfunction and implies towards a bigger role of miR-122 in MASLD.

PP-C-12

MELATONIN ALLEVIATES ANXIETY AND DEPRESSION BY REDUCING HIPPOCAMPAL INFLAMMATION IN A MODEL OF DIET AND CIRCADIAN DISRUPTION

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Emerging from the interplay of metabolic and circadian disruptions, this investigation delineates the neurobehavioral sequelae of a high-fat-high-fructose (H) diet and chronodisruption (CD) via photoperiodic shifts, both individually and synergistically (HCD). These perturbations were measured by increased anxiety in the marble burying, elevated plus maze, and hole board tests, and depressive behavior in the sucrose preference, forced swim, and tail suspension tests. These changes were correlated with significantly elevated levels of thyroid hormones (T3, T4, TSH) and hippocampal pro-inflammatory cytokines (Tnf-α, Il-1β, Il-4, Il-6, Il-10, Il-12, Il-17, Mcp-1, Nf-κb). Exogenous melatonin treatment (HM, CDM, HCDM groups) resulted in moderate to significant amelioration of these neurobehavioral deficits and inflammatory markers. Furthermore, while the H, CD, and HCD groups showed non-significant decreases in mRNA of hippocampal BDNF-TrkB pathway genes (Bdnf, Trkb, Nt-3, Nt-4, Psd-95, Syn-1) with no change in protein levels, melatonin treatment significantly improved these transcripts. This study is the first to report on the compounded effects of H and CD, demonstrating melatonin`s potential as an effective anxiolytic and anti-depressive agent via modulation of hippocampal inflammation and the BDNF-TrkB pathway.

PP-C-13

CARBON MONOXIDE DISRUPTS KHSRP–6PGD COUPLING TO RESTORE MIR-34A BALANCE AND LIPID HOMEOSTASIS IN HEPATOCYTES

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KH-type Splicing Regulatory Protein (KHSRP) functions as an RNA-binding protein that governs pre-mRNA splicing and microRNA maturation. Its altered expression has been associated with metabolic imbalance and lipid accumulation in hepatocytes. We hypothesize that KHSRP upregulates miR-34a expression by binding to downstream controller sequences of 6-phosphogluconate dehydrogenase (6-PGD) pre-mRNA and that this interaction is redox-sensitive and disrupted by carbon monoxide (CO). Oleic acid (0.5 mM, 24 h) treatment in HepG2 cells induced prominent lipid accumulation, elevated KHSRP expression, and its distinct nuclear localization accompanied by a reduction in 6-PGD transcripts. Co-treatment with CO donor CORM-A1 abolished KHSRP nuclear enrichment, normalized miR-34a levels. RNA immunoprecipitation confirmed KHSRP–6-PGD coupling under oleic acid exposure. Moreover, siRNA-mediated KHSRP knockdown mimicked CO treatment by preventing lipid accumulation and reinstating 6-PGD expression. Collectively, these findings identify KHSRP as a key molecular switch orchestrating miR-34a-driven lipid imbalance. Its redox-dependent dissociation by CO highlights KHSRP–6PGD coupling as a novel regulatory checkpoint and a promising therapeutic target for the correction of dyslipidaemia associated with metabolic stress.

PP-C-14

IMPACT OF ELEVATED ESTROGEN-TO-ANDROGEN RATIO ON PRIMARY MOUSE PROSTATE CELLS

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The prostate is the largest accessory gland of the male reproductive system and produces key components required for sperm survival and activity. It is made up of two main compartments—the epithelium and the stroma—whose coordinated interactions are vital for maintaining normal gland function. Sex hormones, especially estrogen and dihydrotestosterone (DHT), play crucial roles in regulating prostatic growth, differentiation, proliferation, and overall activity; disturbances in these hormone levels can contribute to prostate disorders. Primary cell cultures provide a valuable system for investigating these interactions because they closely mimic the genetic diversity and structural features of the native organ. Due to the challenges in obtaining human tissue, mouse models are widely used, as they possess a highly similar genome and comparable cellular organization. This study aimed to investigate the effects of age dependent estrogen: androgen ratio alterations on the mice primary prostate cells.

Male C57BL/6 mice aged 12–14 weeks were euthanized, and their urogenital tracts were collected. The prostate lobes were isolated and dissociated to obtain a single-cell suspension. Cells were then separated using a Percoll gradient and identified through immunostaining to confirm specific cell types. Stromal cells were cultured with increasing concentrations of E2 while maintaining constant levels of DHT, and their proliferation was assessed using an MTT assay. In addition, stromal and epithelial cells were co-cultured under both normal and an altered E2:DHT ratio of 2:1 (20 pM E2 and 10 nM DHT), and cell proliferation in these co-cultures was evaluated using Calcein AM staining.

The primary cells were effectively isolated and fractionated. Higher E2:DHT ratios led to a marked increase in stromal cell proliferation. Both stromal and epithelial cells exhibited substantial growth at the 2:1 E2:DHT ratio, emphasizing the role of sex steroids in regulating cellular interactions and proliferation.

Our results suggest that age-related hormonal changes substantially impact prostate cell physiology, which potentially play a role in the early development of BPH and prostate cancer. Additional studies are needed to further explore the mechanisms involved.

PP-C-15

DECIPHERING THE ROLE OF KISSPEPTIN - PACLITAXEL COMBINATION IN MODULATING ADHESION AND TUMOR MICROENVIRONMENT IN TRIPLE NEGATIVE

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Kisspeptin, encoded by the KISS1 gene, is a classic metastasis suppressor in breast cancer, and has also been linked recently to chemoresistance modulation. Paclitaxel (PTX) resistance remains a significant challenge in Triple-negative breast cancer (TNBC). This resistance is notably driven by an aggressive tumor phenotype, CSC presence, and resistance to detachment-induced apoptosis (anoikis). The primary objective of this study is to investigate the potential role of Kisspeptin as a chemosensitizing modulator of Paclitaxel in the highly aggressive MDA-MB-231 and MDA-MB-468 breast cancer cell lines. MTT-based proliferation analysis, kisspeptin 10 exhibited IC10 and IC50 values of 12.13 nM (KP10) and 110.21 nM (KP50) for MDA-MB-231, and 9.31 nM (KP10) and 88.35 nM (KP50) for MDA-MB-468. Anoikis assay were used to assess the functional characterisation and alterations in detachment-mediated apoptotic resistance which showed efficacy of Kisspeptin–Paclitaxel combinatorial treatment. The molecular analyses, including gene expression profiling and protein expression, were employed to elucidate the regulatory framework governing kisspeptin-enhanced paclitaxel sensitivity, specifically focusing on CSCs, proliferation and DNA repair pathway markers. In addition, in-silico biomarker analyses for publicly available datasets were integrated with molecular assays to model Kisspeptin-mediated remodelling of the tumor microenvironment, which can represent a novel mechanism for overcoming chemoresistance in breast cancer.

PP-C-16

TARGETING THE ALDH1A1 WITH PHARMACOLOGICAL INHIBITOR A37 SUPPRESSES MIGRATION AND INDUCES DEATH OF METASTATIC CERVICAL CANCER CELLS

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Cervical cancer is the fourth most common cause of cancer incidence and mortality among women. Cancer stem cells (CSCs) play a vital role in tumor recurrence, drug resistance and metastasis. Aldehyde dehydrogenase 1A1 (ALDH1A1), a key CSC marker, promotes cell self-renewal, survival and chemoresistance, which ultimately drives tumor aggressiveness, cancer progression and metastasis. In this study, we determined the effects of pharmacological inhibitor of ALDH1A1, A37, on ME-180 human metastatic cervical cancer cell line.

ME-180 cells were treated with various concentrations of A37 (6.25, 12.5, 25, 50, 75 and 100 μM) for 24 hours to evaluate the cell proliferation (MTT assay). Subsequently, the cells were exposed to 50 and 75 μM concentrations of A37 and determined for cell migration (wound healing assay), clonogenic survival (crystal violet staining), cancer stemness (tumorsphere assay), and protein expression (western blot analysis). The statistical significance was assessed by one-way ANOVA followed by SNK test.

A37 treatment markedly reduced cell proliferation, migration, clonogenic survival capacity and tumorosphere formation of cervical cancer cells. Notably, A37 decreased the expression of epidermal growth factor receptor, nuclear factor of activated T cells 1, estrogen receptor alpha, cyclin D3 and poly(ADP-ribose) polymerase proteins. Interestingly, A37 increased the expression of autophagy related proteins ATG5 and ATG16, and apoptotic protein caspase-3.

Pharmacological inhibition of ALDH1A1 with A37 suppressed the proliferation, survival, migration and stemness, while inducing apoptosis and autophagy of cervical cancer cells, underscoring the therapeutic potential of ALDH1A1 inhibitor in the treatment of metastatic cervical cancer.

PP-C-17

DYNAMIC CHANGES OF TLR9 ACROSS PROGRESSIVE STAGES OF TRIPLE-NEGATIVE BREAST CANCER

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Triple Negative Breast Cancer(TNBC) is a subtype of breast cancer, which lacks hormone receptors(ER/PR/Her2). These BC patients are frequently associated with chemo-resistivity and tumor recurrence. Recently, TNBC is considered an immunogenic subtype of BC, making it a good candidate for treatment with immunotherapeutic approaches. TLR (Toll-like receptor), a family of pattern recognition receptors(PRR) has been previously associated with immunoregulatory roles in TNBC and other cancers. But, the role of TLR in TNBC progression still remains to be explored. As understanding the role of TLR and associated genes in tumor initiation, maintenance, progression, and intervention becomes to study in human samples. In the present study we have developed and characterized a murine syngeneic TNBC model. For development of syngeneic mouse model, we injected 4T1 cells into 4th inguinal mammary gland via nipple by intraductal surgery. The H&E images of mouse tumor tissues obtained were confirmed for ductal carcinoma in situ(DCIS) (preinvasive) and invasive carcinoma(IC) (invasive). Also, the mouse TNBC stages obtained were found to mimic the progressive stages of human TNBC. We further characterized these tumor tissues for cell proliferation index, invasiveness, and epithelial content. In this we found progressive loss of alpha smooth muscle actin expression, increase in proliferative cell nuclear antigen and cytokeratin expression as the tumor progressed from DCIS to IC. Additionally, screening the expression pattern of TLR9 in DCIS and IC tumor tissue revealed its significant progressive increase. These results indicate that TLR9 might play an important role in TNBC tumor progression.

PP-C-18

TROP2 DRIVES AGGRESSIVE TRAITS AND SPHEROID FORMATION LINKED TO POOR SURVIVAL IN HIGH-GRADE SEROUS OVARIAN CANCER

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Trophoblast cell surface antigen 2 (Trop2) is overexpressed in ovarian cancer (OC) compared to normal ovarian tissue. Notably, many high-grade serous ovarian cancer (HGSOC) cell lines exhibit elevated Trop2 expression. As HGSOC represents the most aggressive OC subtype with the poorest five-year survival, we investigated the functional significance of Trop2 overexpression in this context. Stable Trop2 knockdown clones of the OVCAR5 cell line demonstrated reduced clonogenic potential. Interestingly, they showed increased vimentin expression and enhanced adhesion to fibronectin-coated surfaces but diminished invasive capacity in matrigel invasion assays. Given that most HGSOC patients present with extensive peritoneal dissemination, we further examined Trop2 expression in ascites from treatment-naïve ovarian cancer patients. Immunohistochemical analysis of ascitic cell blocks (n = 36) revealed Trop2-positive cells in 97.2% (35/36) patients and 44.4% (16/36) displayed Trop2-positive vimentin-negative spheroid-like clusters. Ascitic cells were subjected to qRT-PCR (n = 39) and high Trop2 transcript levels (above median) were significantly associated with the presence of Trop2-positive spheroids ($p = 0.0003$). Immunoblotting confirmed correlation between Trop2 transcript and protein levels. Further, Kaplan–Meier survival analysis revealed that patients with high Trop2 transcript expression had significantly poorer five-year survival outcomes ($p= 0.034$). Together, these findings suggest that Trop2 contributes to the aggressive phenotype of HGSOC by promoting clonogenicity and invasion while facilitating the formation of Trop2-positive spheroids in ascites, which are associated with adverse clinical outcomes.

PP-C-19

GPER1 (G PROTEIN-COUPLED ESTROGEN RECEPTOR 1) ACTIVATION: A NOVEL STRATEGY FOR CHEMOPREVENTION OF PROSTATE CANCER

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Prostate Cancer (PCa) is the second most commonly diagnosed cancer among men, and the number of annual new cases is projected to double by 2040. Given the long latency period of PCa development, preventive strategies are critically needed. G protein–coupled estrogen receptor 1 (GPER1), a plasma membrane–localized receptor, has been shown to exhibit cell context–dependent roles in tumor progression across multiple organ systems. Emerging evidence suggests that GPER1 acts as a tumor suppressor in PCa; however, its chemopreventive potential remains unexplored. Flow cytometry analysis revealed a significant reduction in GPER1-positive cell frequency in high-grade human PCa compared to benign prostatic hyperplasia (BPH) tissues, consistent with observations from public datasets. In the TRAMP (Transgenic Adenocarcinoma of Mouse Prostate) model, GPER1 expression and GPER1-positive cell frequency were significantly higher at the high-grade prostatic intraepithelial neoplasia (HGPIN) stage but declined at the well-differentiated carcinoma (WDC) stage relative to age-matched controls. Methylation profiling indicated increased promoter methylation of GPER1 at the HGPIN stage, followed by a decrease at the WDC stage. Pharmacological activation of GPER1 using its agonist G1, but not when co-administered with the antagonist G15, inhibited the progression of HGPIN to carcinoma in TRAMP mice. In vitro, GPER1 silencing in LNCaP, PC3, and RWPE-1 cells enhanced migration and invasion. Mechanistically, GPER1-mediated epithelial-to-mesenchymal transition (EMT) was regulated via the miR-200a–ZEB2–E-cadherin axis and other metastasis-associated genes. Collectively, these findings demonstrate a protective role of GPER1 signalling in PCa progression and its potential as a promising chemopreventive target.

PP-C-20

TO ELUCIDATE THE ROLE OF TACEDINALINE IN REPRODUCTIVE COMPETENCE USING *C. ELEGANS* AS MODEL SYSTEM

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Epigenetic mechanisms play a crucial role in regulating gene expression during reproductive development. Among these modifications, histone acetylation is considered to be a highly dynamic process which can be targeted using drugs such as HDAC inhibitors. HDACis have been reported to enhance somatic cell reprogramming efficiency and improve developmental competence in cloned embryos. In this study, we sought to investigate the effects of these genome-wide modulators on reproduction in *C. elegans*. Sub-lethal doses of HDACi, Tacedinaline were selected using microtitre plate based bacterial clearing assay. Brood assay was performed on L4 worms exposed to Tacedinaline (500uM) for 48 hrs which exhibited significant increase in brood size of drug treated worms as compared to control. Oocyte morphology analysis indicated dose-dependent improvements in oocyte quality. RNA-Seq was performed in the drug treated worms to decipher the mechanisms governing improved reproductive capacity in worms. Tacedinaline induced widespread transcriptional activation in gonads, highlighting the potential for histone acetylation to regulate genes involved in reproduction. Gene Ontology analysis revealed involvement of neuropeptide signaling, extracellular matrix remodeling, and RNA polymerase II activity, all of which could influence reproductive efficiency. Our findings underscore the importance of careful dose optimization and highlights the translational relevance of *C. elegans* in studying histone acetylation-related pathways, with potential implications for human diseases involving chromatin regulation. Further exploration of the molecular mechanisms underpinning HDAC inhibitor effects could provide new insights into reproductive health.

PP-C-21

DISTINCT TEMPORAL REGULATION OF TROP2 AND EPCAM EXPRESSION IN RAT AND HUMAN PLACENTA

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Transmembrane glycoproteins, Trop2 and EpCAM, belonging to the tumor-associated calcium signal transducer (TACSTD) family, were initially reported as human trophoblast cell surface antigens and later extensively studied in cancers. Despite this, the temporal expression and functions they may serve in different stages of placentation remain largely unelucidated. To address this, the current study conducted a comparative analysis of Trop2 and EpCAM expression across gestation in human and rat placenta, which share relevant similarities along with certain disparities. Distinct species-specific differences in temporal expression patterns were observed; rat placenta exhibited an increase in Trop2 and EpCAM expression towards late gestation, correlating with progressive placental growth, in contrast to human, where expression remains stable from first trimester to term. The peak expression of both proteins, early in human and late in rat, in each species aligns with its respective window of maximal trophoblast invasion. Trop2 and EpCAM were co-localized in the villous trophoblasts in human placenta, while in rat placenta, Trop2 was restricted to labyrinth zone, in contrast to EpCAM, which was also expressed in the junctional zone. In silico promoter analysis showed species-specific transcription factor binding sites with ASCL2, which regulates extravillous trophoblast differentiation, found only in the human Trop2 and rat EpCAM promoter. TEAD4 sites, which mark the proliferative trophoblast progenitors, were observed in both promoters in the rat, but were absent in human EpCAM promoter. In the conclusion, our findings suggest distinct roles for Trop2 and EpCAM in gestational regulation of trophoblast invasion in human and rat placentation.

PP-C-22

ENDOMETRIOSIS AND CIRCULATING MICRORNAS: A DETAILED ANALYSIS AND VALIDATION OF REPORTED BIOMARKERS IN THE INDIAN POPULATION

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Endometriosis is a complex gynecological condition in which tissue similar to the inner lining of the uterus grows outside the uterus, causing pain and infertility. Diagnosis of endometriosis typically requires invasive procedures like laparoscopy. MicroRNAs (miRNAs) have evolved into popular non-invasive indicators for endometriosis and other illnesses. Several studies explored the diagnostic role of miRNAs in endometriosis but no definitive diagnostic marker has been identified yet. The aim of the study is to comprehensively analyze the available miRNAs datasets and validate the consistently identified miRNAs in Indian women. Nine circulating miRNAs were identified by an extensive review based on their consistent expression patterns and stability. Women with advanced-stage endometriosis (n = 12) and controls (n = 11) were recruited and their plasma samples were collected based on clinical symptoms, CA-125 levels, ultrasound, MRI findings, and laparoscopic confirmation. Nine miRNAs (miR-451a, let-7b, miR-150-5p, miR-17-5p, miR-3613-5p, miR-20a-5p, miR-342-3p, miR-125b-5p, and miR-21-5p) were selected and quantified using qRT-PCR. Receiver Operating Characteristic (ROC) analysis was performed to assess their diagnostic potential. Among them, miR-451a and miR-20a-5p exhibited significantly lower expression in endometriosis patients (n = 12) compared to controls (n = 11). miR-451a showed distinct trends compared to previous studies, while miR-20a-5p was consistent with earlier research. Although encouraging, these results are based on a small sample size. To properly evaluate the diagnostic potential of these miRNAs as endometriosis biomarkers, larger, multi-center studies are required in our population.

PP-C-23

EFFECT OF CPT1 INHIBITION MEDIATED ENDOPLASMIC RETICULUM STRESS ON CALCIUM OSCILLATIONS IN MICE OOCYTES

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Oocyte activation is a fundamental step in fertilization, involving a series of biochemical and structural changes triggered by repetitive calcium (Ca^{2+}) oscillations. These oscillations regulate meiotic resumption, cortical granule exocytosis, and polyspermy block, ensuring successful fertilization and embryonic development. Disruption in calcium signaling results in Oocyte Activation Deficiency (OAD), a leading cause of fertilization failure in assisted reproductive technologies (ART). Mitochondria and the endoplasmic reticulum (ER) maintain a functional interaction that is essential for calcium homeostasis and energy balance during oocyte activation. Carnitine Palmitoyltransferase 1 (CPT1), a rate-limiting enzyme in mitochondrial fatty acid β-oxidation, is responsible for transporting long-chain fatty acids into mitochondria for ATP generation. Inhibition of CPT1 by etomoxir disrupts fatty acid oxidation, leading to reduced ATP synthesis, lipid accumulation, and ER stress, which can adversely affect calcium oscillations and oocyte activation.

Aim was to investigate the impact of CPT1 inhibition–induced endoplasmic reticulum stress on calcium oscillations during oocyte activation.

C57BL/6 mouse oocytes were divided into four groups: (1) Negative control, (2) Positive control, (3) Experimental control (Tunicamycin, 10 µg/ml; ER stress inducer), and (4) Experimental (Etomoxir, 300 µM; CPT1 inhibitor). Artificial oocyte activation (AOA) was performed using 7% ethanol, and calcium dynamics were analyzed by immunofluorescence microscopy.

Etomoxir-treated oocytes exhibited diminished calcium oscillations similar to Tunicamycin-treated oocytes, confirming that CPT1 inhibition induces ER stress and suppresses calcium signaling required for oocyte activation.

CPT1-mediated mitochondrial activity is crucial for maintaining ER homeostasis and calcium dynamics, and its inhibition compromises oocyte activation and fertilization potential.

PP-C-24

EFFECT OF SPERM CRYOPRESERVATION ON MITOCHONDRIAL TRANSFERASE EXPRESSION AND SPERM MOTILITY

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Mitochondria is the powerhouse of the cell. The midpiece of sperm contains mitochondria which plays a central role in sperm energy metabolism by generating ATP through fatty acid β-oxidation thus playing a vital role in sperm motility. Disruption of enzymes such as carnitine palmitoyltransferase 1 (CPT1), essential for FAO, can impair ATP synthesis and energy homeostasis, leading to reduced sperm motility post-thaw but the role CPT1 in sperm cryopreservation is not yet explored.

Aim was to study the effect of sperm cryopreservation induced mitochondrial impairments on sperm motility METHOD: This study aimed to evaluate mitochondrial beta oxidation transferase CPT1 expression in pre and post-cryo samples in male patients (n=10). The motility, concentration, vitality, and morphology will be checked to assess the baseline semen parameters. For evaluating mitochondrial β-oxidation parameters, Carnitine palmitoyltransferase 1 (CPT1) expression was assessed by immunofluorescence. Fluorescence intensity was visualized under a confocal microscope to compare pre- and post-cryo expression levels.

CPT1 expression was positively correlated with sperm motility which shows its integral role in energy production. Post-cryo semen parameters (motility and vitality) were downregulated. Further there was a decreased expression of CPT1 following cryopreservation.

CPT1 is the rate limiting factor for β-oxidation which is essential for sperm motility. Post-thaw decrease in motility is one of the cause of decreased fertilization potential in cryopreserved sperm. Oxidative stress has been linked to post-thaw cryo damage but the specific molecular impairment has not been explored. Our study concludes that CPT1 might play a central role in ATP production in the mitochondria of sperm which is necessary for motility and therefore may be one of the target of enhancing sperm motility in post thaw samples.

PP-C-25

ROLE OF GSK3A IN REGULATING THE RNA DEMETHYLASE FTO DURING SPERMATOGENESIS IN MICE

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Idiopathic male infertility is an increasing global concern characterised by defects in semen release, low sperm count, and abnormalities in sperm morphology and motility. Understanding its underlying causes requires detailed insight into the processes of spermatogenesis and sperm maturation. Glycogen synthase kinase 3α (GSK3α), a key signaling enzyme, plays an isoform-specific role in regulating sperm motility, epididymal maturation, capacitation, and hyperactivation. Methylation of mRNA is essential for maintaining transcript stability, cytoplasmic transport, and translation. The mRNA demethylase FTO (Fat Mass and Obesity-Associated Protein) is a target of GSK3α-mediated phosphorylation, suggesting a regulatory link between these enzymes in male fertility. This study explores the high-affinity spatiotemporal interaction between FTO and GSK3α in mouse testis to understand post-transcriptional control mechanisms in spermatogenesis. Expression analyses revealed that both Gsk3a and Fto increase between days 18–34 postpartum, peaking during spermatid formation and sperm morphogenesis. Co-immunoprecipitation demonstrated a preference of FTO for GSK3α over GSK3β, while GSK3α knockout testes exhibited reduced m6A methylation, indicating elevated FTO activity. Computational protein–protein docking further supported the energetically favourable interaction between GSK3α and FTO. Notably, a missense mutation in FTO (Cys326>Ser) introduced an additional GSK3 phosphorylation site, leading to teratozoospermia in a clinical case. Together, these results establish that GSK3α regulates FTO activity in a paralog-specific manner during murine spermatogenesis.

PP-C-26

CONSEQUENCE OF DYSREGULATED GLYCOLIPID METABOLISM IN SERTOLI CELLS AND ITS IMPACT ON SPERMATOGENESIS

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Lipids found in the plasma membrane of germ cells are crucial for fertilization, and approximately 5-8% of all polar lipids in mammalian species are glycolipids. The major glycolipid in sperm is the sulphated glycolipid SGG, which is present in both head and tail fractions of spermatozoa and facilitates sperm-egg interaction during fertilization. During spermatogenesis, approximately 50% of germ cells undergo apoptosis, and Sertoli cells phagocytose the apoptotic germ cells. However, the fate of sulphated glycolipids in phagocytosed germ cells has not been investigated thoroughly. In this study, we present data on lysosomal enzymes involved in the catabolism of sulphated glycolipids in the cellular constituents of seminiferous epithelium from both juvenile and adult mice testes. The key enzymes responsible for the degradation of sulphated glycolipids, viz., arylsulfatase A (ARSA) and galactosylceramidase (GALC), were found to be enzymatically active in the testis and in cells of the seminiferous epithelium, particularly the Sertoli cells. Additionally, enzyme activity across different zones of the seminiferous tubule was also determined; both ARSA and GALC exhibited increased activity in zones representing the spermiation stage, suggesting enhanced activity of these enzymes correlated with apoptotic germ cell clearance in the seminiferous epithelium by phagocytosis. We are currently focusing on delineating the consequences of impaired sulphated glycolipid metabolism in SC function. Investigating impaired glycolipid metabolism in SCs and its impact on spermatogenesis may prove useful in providing therapeutic options to males with defective ARSA and GALC activity.

PP-C-27

EXPLORING THE ROLE OF DNA DAMAGE AND ALTERNATIVE LENGTHENING OF TELOMERES PATHWAYS IN SPERM TELOMERE ELONGATION WITH PATERNAL AGING

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Unlike somatic cells, sperm telomere length (STL) exhibits an unusual increase with advancing paternal age. Telomeric ends are protected by the shelterin complex, which prevents activation of DNA damage response pathways. The alternative lengthening of telomeres (ALT) pathway—a recombination-based mechanism typically seen in cancer cells—can maintain telomeres independently of telomerase. However, its role in germline telomere maintenance during aging remains poorly understood. Although germline stem cells generally show high telomerase activity, the mechanisms driving sperm telomere elongation and potential involvement of ALT with increasing male age are yet to be established.

Aim was to investigate the underlying mechanisms contributing to increased STL with advancing paternal age, focusing on DNA damage accumulation and the possible activation of ALT pathways.

Semen samples were collected from infertile men aged 25–35 years (n=20) and 36–50 years (n=27), along with fertile controls aged 25–45 years (n=12). Semen parameters were assessed as per WHO guidelines. Sperm DNA integrity was evaluated using nuclear decondensation and DNA fragmentation assays. STL was quantified by qPCR, while γ-H2AX, a DNA damage marker, was detected by immunofluorescence and quantified using ImageJ. Expression levels of shelterin proteins (TRF1, TRF2), ALT regulators, and telomerase components (TERT, TERC) were analyzed using qPCR.

STL was significantly higher in the older infertile group (36–50 years) compared to the younger infertile group (25–35 years), yet remained lower than in fertile controls. DNA fragmentation and γ-H2AX levels were elevated in older infertile men, indicating higher DNA damage. TRF2 expression was reduced in infertile men compared to controls but increased with age. ALT regulator expression was upregulated in older infertile men, while TERT and TERC levels showed no significant variation.

The increase in STL with advancing paternal age may represent a compensatory response to age-related DNA damage, potentially mediated by activation of ALT pathways to preserve germline integrity.

PP-C-28

BPA AND HEAVY METAL–DRIVEN EPIGENETIC ALTERATIONS IN IDIOPATHIC HYPOSPERMATOGENESIS

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Idiopathic hypospermatogenesis (HS), a subtype of non-obstructive azoospermia (NOA), is a key cause of severe male infertility characterized by reduced germ cell production without a clearly defined etiology. Emerging evidence suggests that environmental exposures, particularly to endocrine-disrupting chemicals (EDCs) such as Bisphenol A (BPA) and heavy metals (As, Cd, Pb, Hg), may influence spermatogenic function via epigenetic alterations, particularly DNA methylation. However, the specific relationship between EDC exposure and methylation changes in idiopathic HS remains inadequately understood. This study investigated DNA methylation changes linked to high EDC exposure in idiopathic HS, aiming to identify altered genes and pathways as potential biomarkers or therapeutic targets. A prospective cohort of infertile men with idiopathic hypospermatogenesis (HS) and normospermatogenesis was recruited from the Urology Department, AIIMS, New Delhi. Comprehensive clinical, hormonal, genetic (karyotype, Yq microdeletion), and ultrasonographic evaluations were performed, with testicular biopsy for histological confirmation. EDC exposure was assessed via BPA ELISA and heavy metal quantification using ICP-MS, stratifying HS patients into four exposure subgroups. Testicular DNA from archived biopsies underwent genome-wide methylation profiling using microarray technology. Bioinformatic analyses identified differentially methylated regions, associated genes, and disrupted biological pathways. Methylation microarray analysis suggested distinct DNA methylation patterns in high-EDC subgroups, particularly involving genes associated with testicular development, hormonal signaling, and germ cell apoptosis. This study establishes a novel link between environmental toxicants and epigenetic alterations in idiopathic hypospermatogenesis, highlighting EDC-induced DNA methylation changes as potential biomarkers for male infertility.

PP-C-29

DYNAMICS OF CORE AND MODIFIED HISTONES IN SPERM OF INFERTILE MEN WITH CLINICAL VARICOCELE: PRE- AND POST-TREATMENT

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Varicocele is one of the leading causes of male infertility. Although treatment for varicocele is known to improve semen parameters, more than 50% affected men still remain infertile. Varicocele affects spermatogenesis through multiple mechanisms; however, elevated oxidative stress has been highlighted as a key factor underlying varicocele-associated infertility. Excessive oxidative stress during spermatogenesis can compromise mitochondrial function, thereby leading to aberrant epigenetic modifications in spermatozoa. Notably, human sperm retain approximately 5-15% of nucleosomes, which are enriched at loci critical for developmental processes. A subset of these loci can escape epigenetic reprogramming, underscoring their functional relevance. In view of this, our study aimed to evaluate histone and modified histone profiles in the sperm of men with clinical varicocele, both before and after treatment. We analyzed retained nucleosome content in infertile men with varicocele (n = 50) and compared with healthy fertile men (n = 30). Varicocele patients (n = 25) were followed up, and semen samples were recollected three months post-treatment. Using flow cytometry, we quantified the relative abundance of core histones H3 and H4, the histone variant H2AX, protamine-2, and modified histones H3K4me3 and H3K9me3. Our findings revealed significantly elevated levels of core histones (H3, H4), histone variant H2AX, and modified histone H3K4me3 and H3K9me3 in varicocele patients compared with fertile controls. It was noted that varicocele treatment helps in partial restoration of these histone levels. Increased retention of H3 and H4 correlated positively with sperm DNA fragmentation, suggesting defective chromatin packaging and enhanced susceptibility to oxidative damage.

PP-C-30

DELINEATING THE HEMOSTATIC SIGNATURE OF INDIAN WOMEN WITH POLYCYSTIC OVARY SYNDROME

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Polycystic ovary syndrome (PCOS) is a common endocrinopathy affecting nearly 10% of women of reproductive age and a leading cause of anovulatory infertility. Beyond its reproductive manifestations, PCOS is increasingly recognized as a systemic condition associated with insulin resistance, obesity, dyslipidemia, and chronic low-grade inflammation. These metabolic disturbances may contribute to a prothrombotic state through alterations in coagulation and fibrinolysis pathways, predisposing affected women to cardiovascular and thromboembolic complications. However, the precise mechanisms linking PCOS with hemostatic imbalance remain incompletely understood. This study aims to investigate the profiles of coagulation and fibrinolysis markers in plasma from women with PCOS compared with age- and BMI-matched healthy controls, which is relatively unexplored in Indian scenario. Citrated plasma and serum samples were collected from women diagnosed with PCOS according to Rotterdam criteria and from healthy regularly menstruating control participants. Anthropometric, hormonal and biochemical characterization of all study participants was carried out, and hemostatic parameters were assessed in platelet-poor plasma. In our study, we observed no significant difference in common screening assays namely, prothrombin time (PT), activated partial thromboplastin time (aPTT), but significantly decreased thrombin time in PCOS women. Moreover, fibrinogen levels were reduced while fibrin degradation product levels were increased, indicating altered fibrinogen turnover. Women with PCOS exhibited significantly elevated plasma levels of tPA, PAI-1, and plasminogen compared with controls suggesting impaired fibrinolytic efficiency in them. Overall, our findings indicate that women with PCOS are characterized by altered coagulation and fibrinolytic profiles which could partially explain higher prevalence of cardiovascular and thrombotic disorders.

PP-C-31

LIPIDOMIC PROFILING REVEALED ALTERATIONS IN LIPID METABOLIC LANDSCAPE IN FOLLICULAR MICROENVIRONMENT IN WOMEN WITH PCOS

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Polycystic ovary syndrome (PCOS) is the most common endocrinopathy affecting reproductive-aged women. Women with PCOS are at increased risk of insulin resistance, obesity, dyslipidemia, T2D, and CVD. The etiology of PCOS is enigmatic. Infertility and miscarriages are common in PCOS, primarily due to poor oocyte quality influenced by the follicular microenvironment comprising granulosa cells (GCs) and follicular fluid (FF). Folliculogenesis is a metabolically active process, alterations in the metabolic composition of the follicular microenvironment may impair oocyte competence. Increasing evidences indicate alterations in lipid metabolic pathways such as steroid hormone biosynthesis, sphingolipid metabolism, and fatty acid metabolism in PCOS. Therefore, this study investigates the dysregulation of lipid metabolism in follicular microenvironment and its potential contribution to PCOS pathophysiology. FF and GCs were collected from PCOS and control women undergoing in vitro fertilization (IVF). Lipidomic profiling of FF was performed using O-HRLCMS, and gene expression analysis using RT² Profiler arrays in GCs. We found 1104 lipid ions among which 79 were significantly downregulated and 84 were significantly upregulated in PCOS. Supervised multivariate analysis revealed clear separation between PCOS and control groups, indicating distinct lipidomic profiles. Triglycerides (TGs) were significantly elevated, whereas phosphatidylcholines (PCs) and lysophosphotidylcholines (LPCs) were significantly decreased in PCOS. Transcriptomic profiling of genes associated with fatty acid metabolism (FAM) pathways identified eight significantly altered genes in GCs, primarily involved in TG metabolism and fatty acid transport. Overall, our results indicate altered lipid metabolism in the follicular microenvironment leading to poor oocyte quality and poor reproductive outcomes in PCOS women.

PP-C-32

EXPLORING THE ROLE OF VITAMIN D PATHWAY GENETIC VARIANTS IN SUSCEPTIBILITY TO POLYCYSTIC OVARY SYNDROME

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Polycystic ovary syndrome (PCOS) is a multifactorial endocrinopathy affecting 5–20% of women of reproductive age, characterized by hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology. Vitamin-D plays a vital role in reproductive and metabolic regulation. Whole exome sequencing (WES) in our study helped identify functional variants associated to PCOS and revealed vitamin-D metabolism pathway as one of the important pathways. As vitamin-D deficiency is prevalent in PCOS, we focused to study variants in Vitamin-D pathway genes. WES performed on 125 well-characterized women with PCOS identified functional variants in key vitamin-D metabolism genes. We identified one variant in LRP2 (rs2075252) in 124 women, VDR (rs2228570) in 116 women, CUBN (rs62619939) among 32 women, CASR (rs1801725, rs1801726, rs1042636) in 35, 1 and 53 women respectively and GC (rs9016, rs4588, rs7041) in 125, 66 and 95 women respectively. Comparison with South-Asian controls from gnomAD_v2.1.1 database revealed significant allele frequency differences for all variants. These polymorphisms were subsequently validated using Sanger sequencing in an independent cohort comprising 100 PCOS women and 100 controls. Genotypic and allelic frequencies were analyzed for association with PCOS. All identified polymorphism were successfully validated in both groups. However, no statistically significant difference was observed in genotypic or allelic distribution between PCOS cases and healthy controls. This pioneering Indian study explores collective role of multiple vitamin-D pathway genes in shaping the genetic and metabolic profile of PCOS. Larger nationwide studies are warranted to replicate these findings and to further elucidate the potential role of vitamin-D pathway variants in PCOS pathophysiology.

PP-C-33

INTEGRATIVE METAGENOMIC AND METABOLOMIC ANALYSIS TO ELUCIDATE THE ROLE OF GUT MICROBIOTA IN THE PATHOPHYSIOLOGY OF PCOS

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Polycystic Ovary Syndrome (PCOS) is a complex endocrine and metabolic disorder in reproductive-aged women, marked by hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology. Emerging evidence implicates gut microbiota dysbiosis in PCOS, linking it to intestinal permeability, chronic inflammation, and metabolic disturbances. Acting as an endocrine organ, the gut microbiota influences metabolism, immunity, and the gut–brain axis, and its imbalance can disrupt host physiology. Understanding these interactions offers scope for microbiota-targeted therapies to improve metabolic and reproductive outcomes. Most studies, mainly in European and Chinese women, have used 16S_rRNA sequencing to assess taxonomic composition and allows prediction of functional potential of gut microbiota in PCOS. Therefore, this study investigated gut microbiota using shotgun-metagenomics, which provided insights into functional profile of gut microbiota. We carried out shotgun metagenomics of 24 women with PCOS and 16 controls. The hormonal characterization of the recruited participants showed increased levels of LH: FSH ratio, testosterone and decreased SHBG in PCOS group as compared to controls. The alpha diversity indies were low in PCOS as compared to control, however were not significant. We currently didn't detect significant alteration in beta diversity between PCOS and control group. Further we carried out, fecal metabolomics which provide a functional readout of microbial activity, thus enhancing our understanding of the role of gut microbiota in PCOS pathophysiology. We found significantly altered levels of metabolites like choline, amino acids and branch chain amino acid, between PCOS and control. Currently we are investigating functional profiles of gut-microbiota and differential abundance analysis.

PP-C-34

PROPIONIC ACID ATTENUATES METABOLIC DYSFUNCTIONS IN PCOS RAT MODEL

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Polycystic ovary syndrome (PCOS) is a complex metabolic and endocrine disorder characterized by hyperandrogenism, chronic anovulation, polycystic ovaries, and hyperinsulinemia. Considering the metabolic and inflammatory components of PCOS, this study examined the therapeutic potential of propionic acid (PA) in a letrozole-induced PCOS rat model. PCOS was induced in female rats through oral administration of letrozole (1 mg/kg BW). After 21 days of letrozole administration, the rats were treated with either a low or high dose of PA via oral or intranasal routes. We observed metabolic abnormalities, such as fasting hyperglycemia and dyslipidemia, elevated hepatic (SGOT, SGPT, ALP, total bilirubin, and direct bilirubin) and renal markers (creatinine, urea, and uric acid), and hormonal imbalance (high testosterone and low progesterone) in PCOS-induced rats. Furthermore, histopathological examinations of the ovary revealed the presence of multiple cysts and the absence of corpora lutea. Of note, PA treatment restored testosterone and progesterone levels to normalcy, stabilized glucose and lipid levels, and normalized hepatic and renal markers. PA treatment also improved glucose homeostasis and decreased triglycerides and total cholesterol, suggesting enhanced lipolysis, which is a key factor in PCOS regulation. Histological observations showed restoration of follicular morphology in PA-treated rats. Conclusively, PA normalized the biochemical, metabolic, and histological changes associated with PCOS, indicating its potential as a therapeutic agent.

PP-C-35

ENDOMETRIAL RECEPTIVITY AND IMPLANTATION RATE IN ENDOMETRIOSIS: ARE THEY AFFECTED ADVERSELY?

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Endometriosis is frequently accompanied by subfertility, with ~50% of affected women experiencing impaired fecundity. Chronic inflammation, immune dysregulation, oxidative stress and extracellular matrix remodeling have been implicated in compromised endometrial receptivity, all of which are reported to be present in endometriosis. We performed a systematic review of the transcriptomic studies of mid-secretory phase eutopic endometrium in endometriosis, curating Endometriosis-Associated Genes (EAGs; endometriosis vs controls), Receptivity-Associated Genes (RAGs; mid-secretory vs early-secretory/proliferative), and Infertility-Associated Genes (IAGs; mid-secretory in recurrent implantation failure/unexplained infertility vs fertile controls). Across datasets totaling 319 participants, 11 genes showed opposite trend in their expression between EAGs versus RAGs, whereas EAGs and IAGs showed 9 genes with a concordant trend in their expression. We also conducted a meta-analysis (2014–2024; PubMed, Scopus, Google Scholar) of 40 studies evaluating assisted reproductive technology (ART) outcomes in women with and without endometriosis. The number of oocytes retrieved were lower in women with endometriosis compared to fertile control ($p<0.00001$). The number of oocytes retrieved was significantly lower in women with endometrioma ($p<0.00001$) and peritoneal lesions ($p<0.0001$) compared infertile cases without endometriosis; oocytes retrieved was also lower in endometrioma versus peritoneal disease ($p<0.001$). Stratified analyses showed reduced oocyte numbers in stage I/II ($p<0.05$) and stage III/IV ($p<0.00001$) endometriosis when compared with infertile cases without endometriosis, whereas no difference between stages I/II and III/IV of women with endometriosis. By contrast, implantation and clinical pregnancy rates did not differ between women with and without endometriosis. Collectively, our analysis suggests that the infertility experienced in women with endometriosis is not because of a marked derangement in the expression of receptivity associated genes. Ovarian factors are likely to contribute more to subfertility observed in women with endometriosis.

PP-C-36

ENDOMETRIAL GENE EXPRESSION SIGNATURE OF INFLAMMATORY PATHWAY AND ITS ROLE ON ART OUTCOMES IN INDIAN WOMEN

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Endometrial receptivity during the mid-secretory window of implantation (WOI) is crucial for successful implantation, yet recurrent implantation failure (RIF) remains a significant challenge. This study aimed to gain valuable insights into the inflammatory pathways involved in the receptivity profile of the endometrium in our sample population.

Endometrial biopsies from Indian women undergoing Assisted Reproductive Technology (ART) with progesterone treatment group (PTG) were divided into recurrent implantation failure (RIF; n=34) and control (CON; n=33) groups. Total RNA (RNA Integrity Number, i.e., RIN ≥7) was isolated and analyzed using the Agilent SurePrint G3 8×60K microarray platform. Differentially expressed genes (DEGs; fold change >2, $p<0.05$) were identified and functionally annotated in GeneSpring using GO and KEGG pathways.

PTG identified 3,788 DEGs (3,643 upregulated; 145 downregulated). Key inflammatory pathways contributed 122 dysregulated genes, including the JAK–STAT pathway (47), the MAPK pathway (46), the Toll-like receptor (TLR) pathway (17), and the NF-κB pathway (12). Several important genes, such as LIFR, IL6, VEGFA, CSF1, and ERBB4, were also identified, all of which are known to play roles in endometrial receptivity. These genes suggest that cytokine signaling, immune modulation, angiogenesis, growth factor signaling, and cell–cell communication are essential for endometrial receptivity.

This study highlights specific inflammatory pathway genes involved in RIF and identifies several potential biomarkers of displaced WOI. These findings provide a foundation for understanding RIF and aid in developing cost-effective diagnostic tools and personalized embryo transfer strategies to improve ART outcomes.

PP-C-37

UNDERSTANDING THE LIPIDEMIC MILIEU OF SPLEEN IN PCOS RODENT MODEL

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Polycystic ovary syndrome (PCOS) is a complex endocrine–metabolic disorder characterized by reproductive, metabolic, and immune dysregulation, affecting nearly one-third of women worldwide. Its etiology involves disruption of the hypothalamic–pituitary–gonadal axis and insulin resistance, creating an altered endocrine and metabolic state that promotes chronic low-grade inflammation through immune activation. PCOS is marked by an imbalance in immune cell populations, with increased macrophages and lymphocytes secreting pro-inflammatory cytokines that impair insulin signaling and exacerbate metabolic dysfunction. Interestingly, the spleen—a major reservoir and regulator of immune cells—has recently been implicated in reproductive physiology, with evidence suggesting an association between splenic function and ovarian activity. Additionally, the spleen exhibits structural and functional alterations under dyslipidemic conditions, highlighting its potential role in metabolic regulation. In this study, a letrozole-induced PCOS model was established by administering 0.5 mg/kg body weight of letrozole daily for 21 days to 2–3-month-old virgin Charles Foster female rats. Post-treatment, biochemical analysis of lipid parameters by spectrophotometry. and transcript profiling of key lipid metabolism regulators were performed to investigate the spleen's contribution to dyslipidemic pathology. The findings revealed alterations in blood and splenic leukocyte populations, suggesting the possible causes of oligo-ovulation, along with disruptions in splenic lipid metabolism, indicating that the spleen may be directly or indirectly involved in metabolic dysregulation in PCOS.

PP-C-38

DECIPHERING STEROID BIOSYNTHESIS PATHWAY GENE VARIANTS IN PCOS PHENOTYPE A WITH ELEVATED 17-OHP

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Polycystic Ovary Syndrome (PCOS) is a prevalent endocrine disorder affecting 8-13% of women. Exclusion of congenital adrenal hyperplasia (CAH) & non-classical CAH is mandatory before labeling as PCOS. Elevated 17-hydroxyprogesterone (17-OHP) is a marker of CAH, particularly with 21-hydroxylase deficiency. This study, we have explored the 17-OHP level in relation to gene variants of steroidogenic pathways using whole-exome sequencing. This hospital-based observational study involving 106 patients diagnosed with PCOS phenotype A and 26 age-matched fertile female controls. Whole blood samples were collected for serum and genomic DNA isolation to assess 17-OHP levels via ELISA. Whole-exome sequencing was conducted on 80 PCOS patients. The analysis was carried out to look for pathogenic/likely pathogenic variants in genes associated with PCOS. The evaluation of the variants in association with the 17-OHP levels was conducted. We have found that 17-OHP levels were significantly elevated compared to controls. ROC analysis demonstrated excellent discrimination, with a cut-off >0.77 ng/mL. The cohort was stratified into (i) High 17-OHP group (n= 39) and (ii) Normal 17-OHP group (n= 7). In group 1, 63 rare coding variants in 45 genes was detected. Functional annotations of these genes indicated their involvement in ovarian folliculogenesis (FOXO3, AMH), gonadotropin action (AMHR2, AR, etc), steroid hormone biosynthesis (POR, CYP21A2, etc), and metabolic syndromes such as obesity (FTO, ADIPOQ) and insulin resistance (IRS1, UCP2, UCP3). Cumulatively, 2 pathogenic variants, 23 likely pathogenic variants, and 43 variants of uncertain significance. In the normal group, 10 variants in 9 genes related to steroid biosynthesis (e.g., AKR1C3, STAR) were identified, including 1 likely pathogenic and 9 of uncertain significance. There were significant differences in frequencies of the variants in the cohort when compared to the population databases ($p<0.05$). This research expands our understanding of the mutational landscape contributing to PCOS. A 17-OHP level exceeding 0.77 ng/mL in PCOS may serve as a biomarker associated with steroidogenic variants in ~46% of patients. These findings have the potential to enhance the utility of diagnostic genetic screening for the disorder characterized by elevated 17-OHP levels.

PP-C-39

STUDYING THE INFLUENCE OF MELATONIN AND PROBIOTIC METABOLITES IN A HYPERANDROGENIC IN VITRO ENVIRONMENT

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Hyperandrogenism disrupts follicular maturation and alters ovarian steroidogenic pathways. To model this endocrine imbalance in vitro, SKOV3 ovarian epithelial cells were exposed to testosterone, modeling the androgen-dominant environment. MTT assays confirmed that testosterone within the concentration range (1-80 nM) maintained cell viability, validating its suitability for mechanistic interrogation. Biochemical and metabolomic profiling revealed and rogen-driven oxidative stress and metabolic perturbations in SKOV3 cells. Subsequent treatment with melatonin and Bifidobacterium-derived secondary metabolites demonstrated restoration of redox balance. Further studies are underway to analyze possible normalization of steroidogenic activity such as gene expression analyses of FSHR, LHCGR, 17β-HSD, 3β-HSD, MT1, and MT2. Melatonin is well known to influence steroidogenic pathways, however co-stimulation of potential bifidobacterial metabolites may contribute to enhanced cellular redox balance. This study outlines an advanced in-vitro approach to analyze modulation of hyperandrogenism, paving the way for expedited testing of potential biomolecules intended for therapeutic amelioration of Poly Cystic Ovarian Syndrome (PCOS).

PP-C-40

ILLUMINATING THE DIFFERENTIAL REGULATION OF PPAR-Γ AND AROMATASE BY PHARMACOTHERAPEUTICS IN *DANIO RERIO* OVARIES: IMPLICATIONS FOR PCOS INTERVENTION

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Polycystic ovarian syndrome (PCOS) is a multifactorial endocrine disorder characterized by dysregulated steroidogenesis, anovulation, and hyperandrogenism. Aberrant estradiol synthesis, driven by aromatase, along with the paradoxical role of PPAR-γ, contributes to its complex pathophysiology. This study evaluates the modulatory efficacy of known drug molecules on the expression dynamics of PPAR-γ and aromatase in *Danio rerio* ovarian tissue. Sixty adult female zebrafish were randomized into six groups: untreated control, DMSO vehicle, metformin, letrozole, clomiphene citrate, and propionic acid. Following 14 days of treatment, RT-qPCR was performed to assess gene expression in ovaries, complemented by in-silico pathway analysis and molecular docking to explore drug-target interactions. All drugs (except letrozole) upregulated PPAR-γ relative to controls. Metformin increased PPAR-γ while suppressing aromatase, suggesting attenuation of estrogen synthesis to modulate steroidogenesis. In contrast, letrozole downregulated PPAR-γ and elevated aromatase, indicating a compensatory mechanism to restore estrogen levels. Clomiphene citrate induced a two-fold increase in PPAR-γ and elevated aromatase expression, likely reflecting feedback inhibition of estradiol synthesis and subsequent gonadotropin stimulation. Similarly, propionic acid also enhanced PPAR-γ and aromatase expression, positioning it as a potential modulator of ovarian steroidogenesis. Additionally, RNA-seq analysis of granulosa cells from PCOS patients revealed significant dysregulation in ovarian steroidogenesis (27.45%) and TNF signaling (12.8%). Molecular docking confirmed favorable binding affinities of PPAR-γ to clomiphene citrate (−5.0 kcal/mol) and propionic acid (−3.10 kcal/mol), suggesting their potential to modulate PPAR-γ activity. These findings reveal propionic acid as a promising modulator of ovarian steroidogenesis, offering translational relevance for PCOS management.

PP-C-41

MITOCHONDRIA-INDUCED ENDOPLASMIC RETICULUM STRESS: A POTENTIAL MECHANISTIC LINK TO IDIOPATHIC PREMATURE OVARIAN INSUFFICIENCY

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Premature Ovarian Insufficiency (POI) affects ~1% of women before 40 years, yet its molecular mechanisms remain unclear. Mitochondria and endoplasmic reticulum (ER) interact through mitochondria-associated membranes (MAMs), regulating energy production, calcium signaling, and lipid metabolism essential for folliculogenesis. However, the role of mitochondria- and ER-related genes in POI is poorly understood.

Aim was to elucidate mitochondrial and ER functional pathways affecting follicular dynamics in POI.

Idiopathic POI patients (n=30) and fertile controls (n=30) were investigated. Whole exome sequencing (WES) and in-silico modelling identified mitochondrial/ER gene variants. RT-qPCR, immunofluorescence, and ATP assays assessed expression of mitochondrial, ER stress, and β-oxidation markers. For in vivo validation, C57BL/6 mice (3–5 weeks, n=4/group) were treated with mitochondrial inhibitors (Cyclophosphamide, Etomoxir, Omeprazole, L-amino Carnitine, Etoposide, Mildronate). Post-treatment, ovaries and systemic tissues were examined histologically, and serum FSH AMH levels quantified.

WES revealed multiple alterations in genes involved in mitochondrial fatty acid transport and metabolism, including variants of uncertain significance in nuclear and mitochondrial genes. POI patients showed altered expression of mitochondrial and ER stress markers with reduced ATP levels compared to controls. Inhibitor-treated mice demonstrated reduced body weight, ovarian shrinkage, decreased follicle count, increased atresia, and histopathological changes in liver, kidney, and spleen. Elevated serum FSH & AMH confirmed successful induction of POI phenotype.

Mitochondrial fatty acid metabolism and ER homeostasis are crucial for ovarian function. Their disruption impairs β-oxidation, induces ER stress, and drives POI pathogenesis. Identified genetic alterations may serve as biomarkers and therapeutic targets for early POI intervention

PP-C-42

EVALUATING THE EFFECT OF ANTIOXIDANTS IN ENDOCRINE DISRUPTERS AND HIGH-FAT DIET INDUCED POLYCYSTIC OVARY SYNDROME (PCOS) LIKE CONDITION

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Polycystic ovary syndrome (PCOS) is a complex endocrine and metabolic disorder affecting 8-21% of women of reproductive age worldwide. Factors such as endocrine disruptors and high-fat diets contribute to the development of PCOS, and antioxidants like curcumin, vitamin E, and CoQ10 may help alleviate its symptoms. This study investigated the therapeutic effects of antioxidants in a rat model of PCOS induced by bisphenol A (BPA) and a high-fat diet. Immature female Wistar rats were divided into four groups: Vehicle Control (corn oil), BPA (100 µg/kg body weight), High-Fat Diet (35% fat), and High-Fat Diet + BPA. BPA was administered daily via oral gavage, and the high-fat diet was given for 54 days. Throughout the experiment, weekly body weight, vaginal opening, and estrous cycles were monitored. Adulthood assessments included serum hormonal levels, glucose tolerance, and ovarian histology to confirm PCOS-like conditions. After establishing the model, the rats were divided into two subgroups: untreated and antioxidant-treated. The antioxidant cocktail consisted of curcumin (155 mg/kg), vitamin E (10.5 mg/kg), and CoQ10 (20 mg/kg), administered daily for 25 days. Results showed that the high-fat diet combined with BPA disrupted estrous cyclicity, increased body weight, and reduced glucose tolerance, while also causing hormonal imbalances and cystic ovarian morphology. The antioxidant treatment improved estrous cyclicity, glucose tolerance, and other biochemical parameters, indicating potential benefits for managing PCOS.

PP-C-43

COMPARATIVE TRANSCRIPTOMIC ANALYSIS OF SUSD2^{+} ENDOMETRIAL MESENCHYMAL STEM CELLS AND WHOLE-TISSUE LESIONS IN ENDOMETRIOSIS

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Endometriosis (EMs) is a chronic estrogen-dependent disorder characterised by the growth of endometrial-like tissues outside the uterus. Despite its unclear etiology, increasing evidence suggests that endometrial Mesenchymal Stem cells (eMSCs) contribute to lesion formation and maintenance through their regenerative and immunomodulatory properties. However, the molecular mechanisms underlying this contribution are not yet fully understood. We performed RNA sequencing (RNA-Seq) on MACS-sorted SUSD2+ eMSCs and whole-tissue from subtypes of EM- ovarian endometrioma (OMA), deep infiltrating endometriosis (DIE), eutopic endometrium of endometriosis (EEU), and control endometrium (CEU). This study aims to elucidate the role of eMSCs in the pathogenesis by comparing their transcriptomic profiles with whole endometriotic tissues. In the eMSC dataset, significantly differentially expressed genes (DEGs) were detected only when comparing endometriosis subtypes (OMA; 1596 DEGs and DIE; 758 DEGs) and eutopic endometrium (EEU; 1752 DEGs) with controls (CEU). However, whole-tissue data showed a greater number of DEGs in ectopic lesions (OMA, DIE) compared to endometrium, due to presence of various cell types, reflecting greater heterogeneity. We further performed comparative analysis of two datasets, which revealed a subset of overlapping DEGs across OMA (94 DEGs), DIE (61 DEGs) when compared with controls. Among ectopic lesions, the maximum number of common DEGs was observed in OMA, which further complements the eMSCs phenotype of OMA. Similarly, 61 common DEGs are present in DIE between two datasets, reflecting the significant contribution of eMSCs to the DIE lesion. Among ectopic lesions, 10 DEGs were shared by both DIE and OMA. Although a significant number of DEGs were observed in the EEU in the eMSCs dataset, those were not identified in the whole tissue transcriptome. Furthermore, pathway analysis of the overlapping DEGs revealed a similar set of pathways, including signalling pathways (AGE-RAGE, AMPK, cGMP-PKG, TGF-beta), ECM-receptor interaction, and cancer pathways. These findings demonstrate that eMSCs exhibit disease-specific transcriptional changes associated with endometriosis lesions.

POSTER PRESENTATIONS: NONCOMPETITIVE

PP-NC-1	**Harshita Johari** Navrachana University, Vadodara **Exploring Sex Pheromone Communication in Moth Pests for Integrated Pest Management**
PP-NC-2	**Zeel Shah** *The Maharaja Sayajirao University of Baroda* **Development of Nano-based delivery system of Aloe derived bio-actives for management of Polycystic Ovarian Syndrome**
PP-NC-3	**Remi Rana** *The Maharaja Sayajirao University of Baroda* **Impact of Estrogen Receptor Agonist Diarylpropionitrile on Mitochondrial Function and Neuroendocrine Alterations in PCOS Brain**
PP-NC-4	**Divyank Varshney** *BRIC-NIBG, Kalyani, West Bengal* **Multi-Omics Integration Identifies Epigenetic Mediators of Genetic Risk in Preterm Birth**
PP-NC-5	**Shobha Uday Sonawane** *ICMR-NIRRCH, Mumbai* **Epigallocatechin-3-gallate (EGCG) Ameliorates Cypermethrin-Induced Alterations in Spermatogenesis and DNA Methylation in F1 Male Rats**
PP-NC-6	**Jithin Biju** *Manipal Academy of Higher Education, Manipal* **Comparative Analysis of Sperm RNA Yield and Purity Across Three Isolation Protocols Reveals Method-Specific Variation**
PP-NC-7	**Samiksha Ghadshi** *Ramnarian Ruia College, Mumbai* **Insights Gained During a Microbiology Internship at OM Diagnostic Laboratory, Pune**
PP-NC-8	**Jumna Sherin** *University of Calicut, Kerala* **Phytochemical Modulation of Growth Enhancement and immunological Function in Tilapia by Curcumin (*Oreochromis mossambicus*)**
PP-NC-9	**Ladeeda** *University of Calicut, Kerala* **Impairment of Embryonic Vascular Patterning and Organelle Homeostasis in Zebrafish on Exposure to Tin Disulfide Nanoparticles**

PP-NC-1

EXPLORING SEX PHEROMONE COMMUNICATION IN MOTH PESTS FOR INTEGRATED PEST MANAGEMENT

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Moth pests continue to severely threaten agriculture globally and in Gujarat, including the Vadodara region, causing major losses in crops like cotton, groundnut, cucurbits, soybean, and vegetables. Recent studies (2022–2024) highlight increased larval infestations, pesticide resistance, and over-reliance on chemical control, leading to environmental degradation and declining effectiveness. A preliminary survey by the PI across four agricultural zones in Vadodara identified 2,723 insect individuals across 59 species, with major representation from Coleoptera and Lepidoptera. Molecular docking studies revealed strong binding interactions between pheromone ligands and Spodoptera frugiperda odorant receptors—SfuOR56 showed highest affinity to (Z)-9 tetradecenyl acetate (–6.8 kcal/mol), while SfuOR62 showed strong binding to (E)-7 dodecenyl acetate and (Z)-11 hexadecenyl acetate, indicating their potential roles in pheromone detection. Moreover, GC-MS and receptor-ligand studies, will be carried out and the obtained results will be elucidated to formulate strategies for control this pest species in the agricultural fields.

PP-NC-2

DEVELOPMENT OF NANO-BASED DELIVERY SYSTEM OF ALOE DERIVED BIO-ACTIVES FOR MANAGEMENT OF POLYCYSTIC OVARIAN SYNDROME

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Polycystic Ovarian Syndrome (PCOS) is a prevalent endocrine disorder characterized by insulin resistance, hyperandrogenism, and ovarian dysfunction, leading to reproductive anomalies (Diamanti et al., 2012). Current therapeutic interventions primarily involve steroid analogues or ovulatory agents, which often cause adverse effects upon prolonged use. This has led to an increasing interest in identifying safe and effective herbal alternatives for managing reproductive health. Previous studies from our laboratory have demonstrated that Aloe vera gel—a multifaceted medicinal herb—can restore ovarian structure and function (Maharjan et al., 2010; 2014) and reduce associated comorbidities such as dyslipidaemia (Desai et al., 2012) in PCOS rodent models. Further, in vitro investigations have revealed that the non-polar phytocomponents of Aloe vera are likely responsible for these beneficial effects (Dey et al., 2021). In the present study, non-polar bio actives were extracted using the Soxhlet extraction method and isolated through column chromatography. However, bioavailability studies of nonpolar extract of Aloe, indicated that bioactive fractions were present at very low concentrations In vivo, possibly due to poor solubility and limited bioavailability of lipophilic compounds. To overcome these challenges, nanotechnology-based formulations are being developed to enhance solubility, stability, targeted delivery, and sustained release of the bioactive components. This study focuses on developing nanoparticles encapsulating Aloe vera bio actives and evaluating their in vitro release profiles in a dose- and time-dependent manner. Such nanoparticle-based Phyto formulations could provide a potent, efficacious, and safer therapeutic alternative for PCOS management, while enhancing the pharmacological relevance of Aloe vera.

PP-NC-3

IMPACT OF ESTROGEN RECEPTOR AGONIST DIARYLPROPIONITRILE ON MITOCHONDRIAL FUNCTION AND NEUROENDOCRINE ALTERATIONS IN PCOS BRAIN

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Among women of reproductive age, poor lifestyle and metabolic disorders manifest as PCOS. PCOS is a multi-etiological hyperandrogenic disorder affecting 5-15% of Indian women. The hyperandrogenic state can aggravate obesity, acne, hirsutism and aloepecia thereby leading to psychological disorders. This could be associated with altered neurohormones, neurotransmitters, neurosteroids and neuropeptides, along with its receptors.

Moreover, PCOS affected brain depicts a pro-inflammed state with an altered redox microenvironment indicating mitochondrial dysfunction. Apart, from being the powerhouse of the cell the mitochondria regulate redox balance and synthesize neurosteroids.

As PCOS brain exhibits imbalance between androgen and estrogen levels, hence hormone therapy could be an effective approach. Emerging evidences suggest that estrogen significantly impacts mitochondrial biogenesis, fusion/fission, mitophagy, oxidative phosphorylation and reactive oxygen species production. However, no studies highlight the role of mitochondrial steroid receptors with mitochondria physiology. Thereby, targeting Estrogen Receptor (ER) based therapeutics for mood disorders could be good approach.

In present study, Estrogen Receptor (ER) agonist- Diarylpropionitrile (DPN) at 0.01mg is chosen as steroid modulator drug administered to letrozole-induced PCOS rat model for 15 days daily subcutaneously. Spectrophotometric analysis of mitochondrial complexes along with mitochondrial dynamics related genes were assessed by real-time PCR. Neurosteriod alterations was assessed by western blotting and behavioural paradigms were conducted to assess anxiety-like states. DPN treated rats demonstrated restoration of gonadal steroid levels along with reduced ovarian cysts, with mitochondrial function restoration. This study attempts to identify the role of ER-mediated mitochondrial modulation in brain microenvironment of PCOS.

PP-NC-4

MULTI-OMICS INTEGRATION IDENTIFIES EPIGENETIC MEDIATORS OF GENETIC RISK IN PRETERM BIRTH

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Preterm birth (PTB) remains the leading cause of neonatal morbidity and mortality worldwide, yet the molecular mechanisms linking genetic variation to PTB risk are yet to be elucidated. We conducted the first genome-wide association study (GWAS) of spontaneous PTB in South Asian women from the GARBH-Ini cohort, identifying 40 population-specific and 212 trans-ethnic variants. To investigate their regulatory roles, we profiled genome-wide DNA methylation (~850,000 CpG sites) in maternal peripheral blood collected longitudinally at 11–14, 18–20, and 26–28 weeks of gestation and performed methylation quantitative trait loci (meQTL) analysis (±1 Mb from each SNP). We identified 33 meQTLs ($p < 2.89 \times 10^{-6}$) across the three time points, with several SNPs regulating CpG methylation at EGLN3, MED24, and ZNF668 genes. The risk alleles of the SNPs modulated the DNA methylation levels of these genes at each of the three time points in a consistent manner. The expression levels of these genes were also differentially expressed in specific cell types of placentae from preterm and term deliveries. Pathway enrichment of meQTL-associated genes revealed significant involvement of hypoxia signalling, apoptosis, and immune dysregulation. These findings elucidate how some of the SNPs modulate the risk of preterm delivery in women by epigenetically regulating gene expression, leading to placental dysfunction. These novel insights have significant potential for developing methods for early prediction of PTB risk in women and therapeutic targeting.

PP-NC-5

EPIGALLOCATECHIN-3-GALLATE (EGCG) AMELIORATES CYPERMETHRIN-INDUCED ALTERATIONS IN SPERMATOGENESIS AND DNA METHYLATION IN F1 MALE RATS

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Epigallocatechin-3-gallate (EGCG), the predominant catechin in green tea, exhibits potent antioxidant, anti-inflammatory, and epigenetic modulatory properties, including inhibition of DNA methyltransferases (DNMTs). This study evaluated the protective effect of EGCG on the sperm epigenome and reproductive functions in F_1 male rats perinatally exposed to cypermethrin (CYP). Pregnant dams (F_0) received corn oil (vehicle control) or CYP (25 mg/kg body weight/day) from gestation day 6 to postnatal day (PND) 22. At PND 50, CYP-exposed F_1 males were orally supplemented with EGCG (10 mg/kg body weight/day) for 60 days. At PND 120, F_1 males were sacrificed to assess body and reproductive organ weights, sperm parameters, testicular histopathology, and germ cell subpopulations by flow cytometry. Perinatal CYP exposure significantly reduced body and reproductive organ weights, sperm count, and motility, and caused disorganization of the germinal epithelium. EGCG supplementation markedly restored sperm quality and testicular architecture. Flow cytometry revealed an increased proportion of elongating and elongated spermatids in EGCG-treated males. CYP exposure upregulated Dnmt1, Dnmt3a, and Dnmt3b, with downregulation of Dnmt3l, whereas EGCG partially normalized these expression levels. Methylation analysis of H19 DMR CpG sites showed a non-significant trend toward hypomethylation in CYP-exposed males compared to controls. These findings suggest that EGCG mitigates CYP-induced testicular and sperm impairments in F_1 male rats, possibly through modulation of DNA methylation and DNMT expression. EGCG may thus serve as a promising dietary intervention against environmental reproductive toxicity.

PP-NC-6

COMPARATIVE ANALYSIS OF SPERM RNA YIELD AND PURITY ACROSS THREE ISOLATION PROTOCOLS REVEALS METHOD-SPECIFIC VARIATION

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Isolation of high-quality RNA from spermatozoa remains a major challenge due to the compact nature of sperm and the low amount of RNA species compared to somatic cells. This study compared RNA yield and purity using three isolation protocols—Trizol only (TZ), with 2-mercaptoethanol (TB), and Trizol combined with mercaptoethanol and heat (TBH). RNA concentration and $OD_{260/280}$ ratios were quantified spectrophotometrically. Data were analysed using one-way ANOVA followed by Tukey's post hoc test, after confirming normality (Shapiro–Wilk) and homogeneity of variances (Levene's test). RNA yield varied significantly among protocols ($F(2,9)=22.48$, $p=0.0003$). The TB method yielded the highest RNA concentration (157.12 ± 33.0 ng/µl), followed by TZ (101.09 ± 34.9 ng/µl) and TBH (24.79 ± 5.54 ng/µl). Post hoc analysis confirmed significantly higher yields in TB and TZ compared to TBH ($p<0.001$ and $p=0.0096$, respectively). RNA purity also differed significantly ($F(2,9)=7.667$, $p=0.0114$). Although TZ showed the highest mean A260/280 ratio (2.56 ± 0.14), this exceeds the optimal range (1.8–2.1) for pure RNA, suggesting possible DNA carryover. TB (1.98 ± 0.35) and TBH (2.05 ± 0.12) displayed purity consistent with intact RNA. In conclusion, both RNA yield and quality are strongly influenced by the extraction method. The TB protocol achieved maximal yield, while TBH produced RNA of optimal purity. Method selection should balance yield with purity to ensure reliable downstream sperm transcriptomic analysis in male reproductive research.

PP-NC-7

INSIGHTS GAINED DURING A MICROBIOLOGY INTERNSHIP AT OM DIAGNOSTIC LABORATORY, PUNE

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The internship program at OM Diagnostic Laboratory, Pune, provided a valuable opportunity to gain practical experience in clinical microbiology. During the training, I learned various laboratory techniques including sample collection, media preparation, culturing, staining, and identification of microorganisms. I also observed procedures such as Gram staining, antibiotic sensitivity testing, and biochemical tests for bacterial identification. The experience enhanced my understanding of aseptic techniques, biosafety, and the importance of quality control in diagnostic laboratories. In addition to technical skills, the internship improved my teamwork, communication, and record-keeping abilities. Overall, the internship bridged the gap between theoretical knowledge and real-world laboratory applications, strengthening my foundation for future research and clinical practice in microbiology.

PP-NC-8

PHYTOCHEMICAL MODULATION OF GROWTH ENHANCEMENT AND IMMUNOLOGICAL FUNCTION IN TILAPIA BY CURCUMIN (*OREOCHROMIS MOSSAMBICUS*)

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Curcumin, a bioactive compound derived from Curcuma longa, has gained increasing attention in comparative endocrinology for its modulatory effects on growth and immune regulation in aquatic species. This study investigates the endocrine-linked growth response and immunological outcomes of dietary curcumin supplementation in fish. Growth parameters, including weight gain, feed conversion ratio, and specific growth rate, were recorded to assess overall performance. To determine cellular viability and oxidative status, live/dead cell assays using acridine orange–ethidium bromide (AO/EB) staining and reactive oxygen species (ROS) measurement via DCFDA assay were performed. Curcumin-treated groups exhibited improved growth indices compared to controls, along with enhanced cell viability and reduced ROS generation, indicating its protective role against oxidative stress. Flow cytometry analysis of PBMCs further demonstrated that curcumin supplementation enhanced cellular viability, reduced oxidative stress, and improved mitochondrial function, supporting its immunoprotective role. Blood haematology profiles showed improved physiological status in curcumin-treated fish, indicating better systemic health and immune competence. We have also earlier reported a significant increase in n-3 (omega-3) and n-6 (omega-6) polyunsaturated fatty acids in tilapia suggesting the improved the nutritional value in tilapia, an important food fish. These results highlight the potential of curcumin as a nutraceutical agent capable of influencing endocrine-regulated growth mechanisms and strengthening immune responses in fish. This study underscores the relevance of phytochemicals in advancing sustainable aquaculture practices through improved growth and immunophysiological health.

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PP-NC-9

IMPAIRMENT OF EMBRYONIC VASCULAR PATTERNING AND ORGANELLE HOMEOSTASIS IN ZEBRAFISH ON EXPOSURE TO TIN DISULFIDE NANOPARTICLES

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Vascular Patterning Growth Factor (VEGF) plays a pivotal role in angiogenesis, a vital process during embryonic development. Zebrafish embryos (*Danio rerio*), owing to their optical transparency, rapid development and conserved vascular architecture, serve as an ethical and efficient alternative to traditional animal models for studying angiogenesis, in alignment with 3Rs principles. This study investigates the impact of tin disulfide nanoparticles (SnS_2 NPs), which are widely utilised in photocatalysis, lithium-ion batteries, photodetectors, and environmental remediation, on vascular development at 72 and 96 hours post fertilisation (hpf).

Exposure to SnS_2 NPs resulted in a dose-dependent alteration in VEGF patterning, as evidenced by confocal microscopy. Real-time PCR (q-PCR) analysis further revealed a dose-dependent increase in VEGF expression, reflecting a compensatory molecular response to vascular disruption. Mitochondrial assessment using mitosight (mitochondria-specific fluorescent dye) indicated increased mitochondrial stress signals, while lysosight (lysosome-specific fluorescent dye) analysis indicated elevated lysosomal activity, collectively demonstrating heightened organelle perturbation with increasing nanoparticle exposure.

Overall, SnS_2 NPs caused an upregulation of vascular, mitochondrial and lysosomal stress responses, confirming their potential to disrupt embryonic angiogenesis and cellular hemostasis. These findings reinforce the zebrafish embryo as a sensitive, scalable, and ethically favourable model for evaluating nanoparticle-induced developmental toxicity.


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REFERENCE LABORATORIES


GENEX
Science and beyond....


MEDGENOME


Genotypic
genomics simplified


METROPOLIS
The Pathology Specialist


DSS
INSPIRED BY TECHNOLOGY

Comprehensive Research Services

From Ultra-Long Reads with Oxford Nanopore to Q40+ Data, We Deliver It All.

Genotypic
genomics simplified

Whole Genome Methylation Sequencing Using Oxford Nanopore

Enables direct detection of 5mC methylation on native DNA molecules - without the need for bisulfite conversion - offering single-molecule resolution with long-read advantages. This approach captures epigenetic patterns across entire genomes, enabling comprehensive methylome profiling even in complex or repetitive regions.

Key Highlights:

- Native molecule sequencing avoids harsh bisulfite conversion, preserving DNA integrity and minimizing conversion errors.
- Long reads enhance mapping confidence in repetitive and GC-rich regions.
- Enables long-range epigenetic patterning, critical for understanding gene regulation and chromatin dynamics.
- Validated data shows >99% gene-level overlap with Illumina short-read methylome datasets.
- Suitable for plant and human genomes, providing insights into developmental biology, cancer, and neurological disorders.

Cell line Characterization

Accurate cell line characterization using long- and short-read sequencing ensures gene integrity, copy number, splicing, and mutation detection. Direct RNA sequencing reads native RNA end-to-end, preserving strand specificity, resolving isoforms, and detecting RNA modifications like m6A for true transcriptome and epitranscriptome insights.

Key Highlights:

- No cDNA conversion or PCR – Avoids biases and preserves native RNA structure.
- Strand-specific full-length transcript reads – Ideal for isoform identification and splicing analysis.
- Direct detection of RNA modifications (e.g., m6A, pseudouridine) – No chemical enrichment required.
- Poly-A tail length analysis – Supports studies on transcript stability and processing.
- Real-time sequencing – Enables dynamic decisions during the run.
- Excellent for complex transcriptomes – Especially useful in neurobiology, cancer, and developmental biology research.

Unlock Cellular Insights With Single-Cell Sequencing

Explore the biology of individual cells with unmatched clarity. Our Single-Cell Sequencing solutions - powered by 10x Genomics and Oxford Nanopore platforms - uncover rare cell types and distinct molecular signatures often missed in bulk analyses.

Key Highlights:

- Immune responses and clonal diversity Tumor heterogeneity and microenvironment
- Developmental trajectories and lineage mapping
- Gene regulatory networks and epigenetic states
- Isoform diversity and RNA modifications with full-length nanopore sequencing

Achieve Complete Genomic Resolution With T2T Sequencing

Telomere-to-Telomere (T2T) sequencing delivers gapless, reference quality assemblies - capturing repetitive, GC-rich and complex regions missed by standard methods.

Key Highlights:

- Resolves centromeres, telomeres, and satellite repeats
- Enables accurate gene annotation and structural variant detection
- Ideal for plant, human, and complex genomes
- Supports evolutionary studies and epigenetic mapping
- Combines ultra-long Nanopore reads with HiFi/short reads for hybrid accuracy

Next-Gen Proteomics With SomaLogic's SOMAmer® Technology

Profile thousands of proteins with unmatched specificity, sensitivity, and reproducibility -using just microliters of sample. Our high-throughput SomaScan® platform empowers deep biomarker discovery in a single scalable assay.

Ideal for investigating:

- Measures up to 11,000 proteins per sample
- Works on serum, plasma, CSF, and other biological matrices
- Wide dynamic range: detects high- and low-abundance proteins
- High reproducibility with <5% CV and minimal cross-reactivity
- Suitable for translational research, drug discovery, and clinical trials

Advance Your Research With Our Comprehensive Research Solutions

- Microarray
- RNA Sequencing
- ChIP sequencing
- ATAC Sequencing
- Exome Sequencing
- Amplicon sequencing
- Methylome Sequencing
- Direct RNA sequencing
- Degradome/PARE-seq/RAD-seq/GBS
- Metagenome/metavirome sequencing/16s/ ITS
- Mitochondria and Chloroplast genome sequencing
- Epigenome/Bisulfite/meDIP/meRIP sequencing
- Targeted re-sequencing
- miRNA/small RNA Sequencing
- Whole Genome Sequencing
- Whole Genome Metagenome sequencing

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