IFPA 2015
The Placenta: influence and impact

September 8-11, 2015
Brisbane Exhibition & Convention Centre
Queensland, Australia

PROGRAM
Robinson Research Institute’s Core Facilities
Local experts providing high quality, efficient and cost effective services

Adelaide Research Assay Facility (ARAF)
Specialised, high-throughput and high-sensitivity assays of physiologically important analytes
ARAF provide a one-stop-shop for researchers who require analyses but may not have ready access to the expertise, reagents or equipment to undertake them.

The facility services and consults to academic and commercial clients who require specialised measurements of analytes in human or animal biological fluids, or cell culture / tissue extracts.

Gene Silencing & Expression Facility (GSEx)
Construct design, virus production and pilot transduction studies
GSEx provide gene manipulation services in a fully equipped PC2 laboratory - managed by a team with >20 years experience in viral transduction technology from Stanford University and Immunex.

The facility offers custom production of lentiviral, AAV and retroviral vectors, stock viruses, CRISPR, non-viral vector and other cell and molecular biology services.

SA Genome Editing Facility (SAGE)
Custom Knock Out (KO) mice within weeks of order
SAGE uses cutting-edge genome editing technology to generate mutant mice for a wide range of applications.

Utilising CRISPR technology, SAGE directly modify the genome of zygotic embryos - offering fast generation of KO animals (3-6 weeks from injection), lower cost and complete control over the genetic background.

Visit the Robinson Research Institute website to learn more
adelaide.edu.au/robinson-research-institute
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On behalf of the Local Organising Committee (LOC), welcome to Brisbane and to IFPA 2015, The Placenta: Influence and Impact. The meeting will provide an update of our understanding of the mechanisms underlying placental development and the impact of dysfunctional placentation on fetal development and the subsequent disease risk profile of the offspring. The LOC has developed a diverse scientific program for delegates to engage in, including 13 featured lectures and 12 workshops. In addition, the translational and clinical focus of the meeting has been expanded. I acknowledge and sincerely thank all those who have committed time and effort to organizing the conference and especially, the Scientific Committee, Marie-Claire Morley (abstract submission) and the team at ICMS (Professional Conference Organizer).

Thanks to our academic sponsors: Griffith University; Mater Research; the Robinson Research Institute; and UQCCR, and to our major corporate sponsor, Shimadzu Australia.

I hope that you find the meeting productive and informative, and take all opportunity to nurture the collaborative and personal relationships that make IFPA such a unique and rewarding organisation.

Best wishes
Greg Rice, Chairman, IFPA 2015

I would like to extend a very warm welcome to attendees of the 2015 meeting of the International Federation of Placenta Associations. The Australia-New Zealand Placental Research Association and University of Queensland’s Centre for Clinical Research, our hosts for this meeting, have excelled! Our location here on Brisbane’s beautiful South Bank, the conference venue, speakers and program is the recipe for a wonderful meeting. From xenobiotics to stem cells, from epigenetics to immunology, this meeting has not just something for everyone but a range of topics and ideas to expand the mind.

A special note about our early career researchers (ECRs): In IFPA we like to make sure all our ECRs feel a part of the conference. We want them to get the most out of these occasions, meeting with senior and junior investigators alike. For some, this can be a little intimidating, so let me ask all our experienced researchers to take time out to meet with the ECRs and discuss science, careers and collaboration with them.

Another aspect of the ECR experience is the travel awards: We would like to express our heartfelt thanks to Professor Y.W. (Charlie) Loke for his continuing generosity in funding the Loke Travel Awards. We would also like to extend our appreciation to Dr. Les Myatt for his efforts in obtaining the NIH grant that funds travel for ECRs from the United States (and which funds the NIH Lecture). Finally we would like to thank Elsevier for their continued support of IFPA, especially through the Elsevier Trophoblast Research Award.

I look forward to greeting all of you at the start of another event to be remembered.

Nick Illsley
President, IFPA
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# COMMITTEES

## Local Organising Committee
- Vicki Clifton
- Paul Dawson
- Greg Duncombe
- Sebastian Illanes
- Nick Illsley
- Murray Mitchell
- Karen Moritz
- Les Myatt
- Tony Perkins
- Greg Rice (Chairman)
- Claire Roberts
- Carlos Salomon (ECR Chairman)
- David Simmons

## Scientific Advisory Committee
- Philippe Boeuf
- Larry Chamley
- Jo James
- Jeff Keelan
- Martha Lappas
- Jonathan Morris
- Padma Murthi
- John Newnham
- Kristy Pringle
- Roger Smith
- Luis Sobrevia
- Stephen Tong

## Awards Committee
- Estela Bevilacqua
- Alicia Jawerbaum

## International Scientific Committee (abstract review committee)
- Estela Bevilacqua
- Graham Burton
- Anthony Carter
- Larry Chamley
- Gernot Desoye
- Danièle Evain-Brion
- Berthold Huppertz
- Nick Illsley
- Yoshiki Kudo
- Alicia Jawerbaum
- Thomas Jansson
- Rebecca Jones
- Martin Knöfler
- Fiona Lyall
- Lopa Leach
- Les Myatt
- D. Michael Nelson
- Christiane Pfarrer
- Christopher Redman
- Alessandro Rolfo
- Yoel Sadovsky
- Annetine Staff
- Tullia Todros
- Melissa Westwood
- Aureo Yamada
- Stacy Zamudio
Kent Thornburg

Professor Thornburg received his Ph.D. in developmental physiology and studied cardiovascular physiology as an NIH postdoctoral fellow at Oregon Health & Science University. He is M. Lowell Edwards Chair, Professor of Medicine, Director of the Center for Developmental Health at the Knight Cardiovascular Institute, and Director of the Bob and Charlee Moore Institute for Nutrition & Wellness.

Kent has expertise in cardiopulmonary physiology, placentology, and developmental programming. He studies the roles of the placenta and the intrauterine environment as programming agents for adult-onset chronic disease and he leads studies on maternal diet and body in regulating fetal growth in women of Oregon. He is the principal investigator on NIH funded studies including maternal-fetal signaling, training in translational cardiovascular research, thyroid hormone and heart development and placental function. He collaborates with scientists in England, New Zealand, France, Finland and Australia.

He served as editor of the journal, Placenta, as consulting editor for Pediatric Research and on the editorial board of the American Journal of Physiology. Additionally, Dr. Thornburg serves on advisory panels at the National Institutes of Health, the American Heart Association and the Children's Heart Foundation and recently served as co-chair of the task force to determine the 10-year vision of the developmental origins of health and disease for the National Institute of Child Health and Human Development. He also served as a Distinguished Editor for the NIH Center for Scientific Review.

Contact: Knight Cardiovascular Institute
Oregon Health & Science University Portland,
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John Challis

Professor John Challis commenced his position as Pro Vice-Chancellor for Health and Medical Research in February 2014 for a two-year period. His role is to provide academic leadership in driving the institution and its collaborators, particularly in Western Australia, to increase the WA share medical and health sciences funding from all sources.

John also holds the title of University Professor Emeritus at the University of Toronto, and Adjunct Professor at the University of British Columbia and Simon Fraser University. He is Principal of LHC, Life Health Consulting, in Vancouver BC.

John completed his training at the Universities of Cambridge, UC San Diego and Harvard and held a junior research fellowship at Wolfson College, University of Oxford, before moving to Canada. More recently he served as Chair of the Department of Physiology at the University of Toronto, and later as Vice President Research and Associate Provost. He was the Scientific Director of the Canadian Institutes for Health Research, Institute of Human Development, Child and Youth Health and served as President and CEO of the Michael Smith Foundation for Health Research In Vancouver BC. Author of more than 500 peer review papers and chapters, he has trained more than 100 graduate students and postdoctoral fellows, and held in excess of $25 million research funding. He is a Fellow of the Royal College of Obstetricians and Gynaecologists, the Institute of Biology, the Canadian Academy of Health Sciences and of the Royal Society of Canada.

Contact: Office of the Pro Vice-Chancellor, The University of Western Australia

Email: john.challis@uwa.edu.au
Yoel Sadovsky

Director, Magee-Womens Research Institute
Elsie Hilliard Hillman Chair of Women’s Health University of Pittsburgh

Yoel received his MD degree from the Hebrew University in 1986, followed by OBGYN residency at Washington University and maternal-fetal medicine and postdoctoral research fellowships at UCSF. He then returned to Washington University and served as Director, Division of Maternal-Fetal Medicine and Ultrasound between 1999-2007. In 2007, he assumed Directorship of Magee-Womens Research Institute at the University of Pittsburgh. In 2014 he was appointed Associate Dean, Women’s Health Research and Reproductive Sciences, and in 2015 he was honored by the University as a Distinguished Professor in Obstetrics and Gynecology.

Dr. Sadovsky seeks to decipher placental function and adaptation to injury, focusing on trophoblast-specific long non-coding and microRNAs and their communication among the fetal, placental, and maternal compartments. He also interrogates trophoblast trafficking of lipids. His laboratory is funded by several NIH grants, and his investigation resulted in more than 130 articles and 20 chapters and invited publications. Dr. Sadovsky has served as a member of the NICHD Advisory Council and currently serves on the NICHD Board of Scientific Directors. He also serves as an Editor for the journal Placenta. In 2004, Dr. Sadovsky received the Society for Gynecologic Investigation’s (SGI) President’s Achievement Award, and was recently elected President of that society for 2016-17. In 2013, Dr. Sadovsky was elected to the Institute of Medicine, and in 2014 he was elected Member, Association of American Physicians (AAP). Dr. Sadovsky was awarded the 2013 Cozzarelli Prize in biomedical science from the National Academy of Sciences.

Contact: Magee-Women’s Research Institute
204 Craft Avenue, A608, Pittsburgh, PA 15213
Email: ysadovsky@mwri.magee.edu

Derek Wildman

Professor Wildman is Professor of Molecular and Integrative Physiology Research Theme Leader In Computational Genomic Medicine at the School of Molecular and Cellular Biology, University of Illinois and Editor-In-Chief Molecular Phylo-genetics And Evolution. Dr Wildman completed his PhD at New York University and a post-doctoral fellowship at Wayne State University. Derek moved to the University of Illinois in 2014.

The overall goal of Derek’s research is to determine the genomic underpinnings of human phenotypes. Current research in his laboratory has two directions.

Firstly, elucidating the evolutionary genomic history of mammalian and especially human pregnancy. This research takes advantage of comparative genomic data to test hypotheses about genes implicated in human evolution. Comparative approaches are used to develop a better understanding of major obstetrical syndromes. A long-term goal is to accurately describe the evolution of parturition in mammals.

Secondly, the evolution of mammals in general and primates in particular. Derek’s approach is to infer well-supported phylogenetic trees in primates, and to use these trees as the foundation upon which hypotheses about positive Darwinian selection in human evolution are tested. Technologies used include genome sequencing, transcriptomics, qPCR, and epigenomics. He also develops high-throughput computational methods for genomic analysis. These techniques have allowed Derek’s team to understand patterns of natural selection and expression patterns in genes involved in many human phenotypes including those of pregnancy and the brain.

Contact: School of Molecular and Cellular Biology, University of Illinois, Urbana, IL
Email: wildmand@illinois.edu
Matthew Rätsep

Matthew T. Rätsep is a Ph.D. candidate in the Pharmacology & Toxicology graduate program at Queen’s University in Kingston, Ontario, Canada. His current research is focused on placental growth factor (PGF), an angiogenic protein whose deficiency is implicated in the development of preeclampsia, a severe gestational disorder.

Specifically Matthew’s research has centered on the role of PGF in mediating fetal brain development and cognitive function in mice and humans. He hopes to further this line of work through pursuing a position in the pharmaceutical industry. Matthew previously completed his B.Sc. and M.Sc. studies in Health Sciences at Brock University in St. Catharines, Ontario, Canada. Shortly after coming to Queen’s University, Matthew was awarded the Frederick Banting and Charles Best Canada Graduate Scholarship to fund his Ph.D. work.

As part of his Ph.D., Matthew participated in a 6-month student exchange at the State University of Campinas (UNICAMP) in Brazil. Matthew has presented his work at many national and international conferences, and has been recognized with numerous presentation awards. These include the Best Oral Presentation at the 2013 Latin American Symposium on Maternal-Fetal Interaction (SLIMP) in Foz do Iguaçu, Brazil, the Best Platform Presentation at the 2014 Society for the Study of Reproduction (SSR) meeting in Grand Rapids, USA, and the Trophoblast Research New Investigator Award for best poster presentation at the 2014 International Federation of Placenta Associations (IFPA) meeting in Paris, France.

Contact: Department of Biomedical and Molecular Sciences, Queen’s University, Kingston, Ontario, Canada

Email: m.ratsep@queensu.ca

Anthony Carter

Anthony M. Carter is Emeritus Reader at the Institute for Molecular Medicine, University of Southern Denmark. He was educated at Latymer Upper School and Magdalene College Cambridge.

Anthony received his doctorate from the University of Lund, Sweden. He has served as Dean of Medicine at the University of Southern Denmark and been a Visiting Professor at the Universities of Toronto and Western Ontario.

His research has encompassed placental circulation, placental gas exchange, fetal endocrinology and the control of fetal growth. In recent years he has focused on comparative studies of the mammalian placenta and the evolution of placentation.

Contact: Kardiovaskulær og Renal Forskning Odense C, Denmark

Email: acarter@health.sdu.dk
Roger Smith AM

Roger Smith MB, BS, Hons., FRACP, FRANZCOG is Director of the Department of Endocrinology John Hunter Hospital and Co-Director of the University of Newcastle Priority Research Centre in Reproductive Science and Director of the Mothers and Babies Research Centre. He was awarded his Bachelor of Medicine & Surgery from the University of Sydney and his PhD from the University of London.

Rogers research focuses on clinical and basic science aspects of the physiology of human birth and the pathophysiology of premature birth. He is an internationally recognised leader in the pathophysiology of human pregnancy and published in Nature, Nature Medicine and the New England Journal of Medicine and Scientific American. He is also a recognised leader in the field of maternal health in Indigenous Australians.

Contact: School of Medicine and Public Health
Faculty of Health and Medicine, John Hunter Hospital, Newcastle, NSW, Australia

Email: roger.smith@newcastle.edu.au

David Olson

David M. Olson, Ph.D., FRCOG is Professor of Obstetrics and Gynecology, Pediatrics and Physiology at the University of Alberta in Edmonton, Canada. His work is dedicated to improving maternal-child health, especially discovering new means to diagnose and treat preterm birth. Educated at Augustana College (Sioux Falls, SD), the University of Minnesota, St. Louis University, and Western University (London, Canada), he directed or co-directed the University of Alberta Perinatal Research Centre, the CIHR Group in Perinatal Health and Disease, the CIHR Strategic Training Initiative in Maternal-Fetal-Newborn Health Research, and the AIHS Interdisciplinary Preterm Birth and Healthy Outcomes Team. He currently directs the international GAPPS Inflammatory Pathways to Preterm Birth and PREBIC Optimal Pregnancy Environment Risk Assessment (OPERA) teams. He is a founding board member of the Child Health Research Institute and The Mogenson Trust (both Western University), The Alberta Centre for Child, Family and Community Research and has been elected to serve as an officer in several national and international societies, including President of the Canadian Investigators in Reproduction. He founded and organized the Western Perinatal Research Meeting in Banff from 1993-2013 and then the Canadian National Perinatal Research Meeting in 2014. Raising >$30M for research, he has supervised >140 trainees and published >150 papers on prostaglandins, steroids and inflammatory mediators on birth and fetal development. He is patenting intellectual property and translating it to improve women’s pregnancy and newborn health. In 2008 he received his university’s first President’s Achievement Award for leadership; in 2009 he was elected a Fellow ad eundem of the Royal College of Obstetricians and Gynaecologists and in 2015 received the Regional Achievement Award from the Society of Obstetricians’s and Gynecologists Canada.

Contact: Department of Physiology, University of Alberta, Edmonton, Alberta, Canada T6G 2S2
Email: david.olson@ualberta.ca
9:00   IFPA Executive Meeting (by invitation), Concord Room

12:30  Lunch (Executive Committee) (by invitation)

14:20  Welcome

14:30  Plenary Session 1
       Chair: Nicholas ILLSLEY (Hackensack University Medical Centre, USA)

       KEY 1 The Placenta is the Culprit in Programming Chronic Disease
       Kent THORNBURG (The Moore Institute, Oregon Health & Science University, USA)

15:00  Plenary Session 2
       Chair: Murray MITCHELL (UQ Centre for Clinical Research, University of Queensland, AUS)

       Reflections on Preterm Birth
       John CHALLIS (University of Western Australia, AUS)

15:30  Coffee Service

16:00  NIH Lecture
       Chair: Les MYATT (Center for Pregnancy and Newborn Research, University of Texas Health Science Center at San Antonio, USA)

       TBA
       Yoel Sadovsky (Magee-Women’s Research Institute Elsie Hilliard Hillman Chair of Women’s Health Research)

17:00  End of Day

18:30  Opening Reception – Boulevard Foyer
       LOKE and Awards
       ANZPRA ECR Awards

Note to Delegates: The underlined prefix of the title of the presentation (e.g. KEY 1) in this Program Book relate to the reference of the abstract published in Placenta. Abstracts received after the deadline for publication in Placenta can be found in the Late Breaking Abstract section of this booklet.
PROGRAM – Wednesday 9th September 2015

7:30  Awards Committee – Boulevard Room 1

8:30  Oral Presentations Session 1 – Boulevard Auditorium
Chairs: Claire ROBERTS (University of Adelaide, AUS) and Carlos SALOMON (UQCCR, AUS)

O1.1  Placenta and obesity: Differences in reproductive immunology and uteroplacental pathology
Terry MORGAN (Oregon Health & Science University, USA)

O1.2  Assaying the enhancer landscape during early placental development to identify trophoblast invasion transcriptional code and gene-enhancer networks
Geetu TUTEJA (Stanford University, USA)

O1.3  Prepregnancy maternal vitamin D deficiency and placental development in mice
Alison GERNAND (Penn State, USA)

O1.4  Mitochondrial function and glucose metabolism in the placenta with gestational diabetes mellitus
Sribalasubashini MURALIMANOHARAN (University of Texas Health Science Center at San Antonio, USA)

10:00  Coffee Service

10:30  New Investigators Session 1 – Boulevard Auditorium
Chairs: Paul DAWSON (Mater Research Institute, AUS) and Manuel VARAS (University of the Andes, Chile)

NI 1.1  Aberrant decidual macrophage activation is implicated in pre-eclampsia through impaired macrophage-trophoblast interactions
Rebecca BUCKLEY (St George's University of London, UK)

NI 1.2  NRG1-mediated formation of an ErbB2/ErbB3 heterodimer in extravillous trophoblasts suppresses apoptosis
Kerstin PLESSL (Medical University of Vienna, Austria)

NI 1.3  Defining a novel in vivo hierarchy amongst vessel resident endothelial progenitors using the human term placenta
Abbas SHAFIEE (University of Queensland, AUS)

NI 1.4  Altered activin receptor ACVR2A expression in pre-eclampsia contributes to abnormal placentation
Hannah EE JUEN YONG (University of Melbourne, AUS)

NI 1.5  Necroptosis: A novel contributor to placental death in pre-eclampsia
Liane BAILEY (University of Toronto, Canada)

NI 1.6  Hypoxia and high glucose modulate the bioactivity of placental exosomes on endothelial cells
Carlos SALOMON (UQCCR, Brisbane, AUS)
PROGRAM – Wednesday 9th September 2015

12:00 Keynote Lecture – Boulevard Auditorium
Chair: Theresa POWELL (University of Colorado Anschutz Medical Campus, USA)

KEY 3 - Aging and the Placenta
Roger SMITH (University of Newcastle, AUS)

12:30 Lunch (and Regional AGMs (PAA, Boulevard Auditorium; EPG, B1; JPA, B2; ANZPRA, B3)

13:30 Elsevier TR Award – Boulevard Auditorium
Chair: Nicholas ILLSLEY (Hackensack University Medical Centre, USA)

TRAL Impact of placental growth factor and preeclampsia on brain development, cognition, and behaviour.
Matthew RÄTSEP (Queens University, CAN)

14:30 Workshops 1-4 (Parallel sessions)

1. Late onset IUGR surveillance and monitoring (Room B1)
2. Nanomedicine applications and exosome biology (Room B2)
3. Placental Epigenetics (Room B3)
4. Placental Pathology (Boulevard Auditorium)

17:00 Poster Session 1 – Boulevard Foyer

18:30 End of Day

18:30 ECR Social Events
PROGRAM – Thursday 10th September 2015

7:30 TR/Placenta Editorial Board Meeting – Boulevard Room 1

8:30 Oral Presentations Session 3 – Boulevard Auditorium
Chairs: Luis SOBREVI (The Pontificia Universidad Católica de Chile) and David SIMMONS (University of Queensland, AUS)

O3.1 Prenatal maternal stress alters placental serotonin system in a foetal sex dependent manner
Joey ST-PIERRE (Université du Québec, CAN)

O3.2 Understanding the placental mechanisms underpinning increased fetal growth in a mouse model of FGR following sildenafil citrate treatment: insight from network analysis
Colin SIBLEY (University of Manchester, UK)

O3.3 Benefits of exercise training for females born small on a high fat diet
Mary WLODEK (University of Melbourne, AUS)

O3.4 Evidence for placental metabolic reprogramming in pregnancy pathologies
Stacy ZAMUDIO (Hackensack University Medical Centre, USA)

10:00 Coffee Service

10:30 New Investigators Session 2 – Boulevard Auditorium
Chairs: Mary WLODEK (University of Melbourne, AUS) and Thomas JANSSON (University of Colorado, USA)

NI 2.1 Blocking leukemia inhibitory factor impairs trophoblast invasion and leads to abnormal placentation and pregnancy loss in mice
Amy WINSHIP (MIMR-PHI Institute, AUS)

NI 2.2 The placental sulfate transporter, Slc13a4, is critical for skeletal development in mice
Joanna RAKOCZY (University of Queensland, AUS)

NI 2.3 Maternal corticosterone exposure in the mouse causes sex specific alterations in placental OGT and O-linked glycosylation
James CUFFE (University of Queensland, AUS)

NI 2.4 Early alcohol exposure alters placental trophoblast differentiation
Jacinta KALISCH-SMITH (University of Queensland, AUS)

NI 2.5 The missing link – How the placenta programs the maternal brain
Hugo CREETH (University of Cardiff, UK)

NI 2.6 Targeted placental delivery of insulin-like growth factor-II increases fetal weight in P0 mice
Anna KING (University of Manchester, UK)
PROGRAM – Thursday 10th September 2015

12:00 Keynote Lecture – Boulevard Auditorium
Chairs: Sebastian ILLANES (University of Los Andes, Chile) and Karen MORITZ (The University of Queensland, AUS)

O2.1 The role of microRNA miR223 in immune adaptation for pregnancy and fetal-placental development
Sarah ROBERTSON (The University of Adelaide, AUS)

12:30 Lunch (and IFPA AGM, Boulevard Auditorium)

13:30 IFPA Award Lecture – Boulevard Auditorium
Chair: Alicia JAWERBAUM (University of Buenos Aires, Argentina)

Phylogenomic origins and evolution of the mammalian placenta
Derek WILDMAN (University of Illinois, USA)

14:30 Workshops 5-8 (Parallel sessions)

5. Lipid mediators of placental function (Room B1)
6. Biomarkers of placental complications of pregnancy (Room B2)
7. Xenobiotics and endocrine disruptors and pregnancy (Room B3)
8. Stem cells of the fetomaternal interface (Boulevard Auditorium)

17:00 Poster Session 2 – Boulevard Foyer

18:30 End of Day – Free Evening
PROGRAM – Friday 11th September 2015

7:30  Awards Committee Meeting – Boulevard Room 1

8:30  Oral Presentations Session 3 – Boulevard Auditorium
      Chairs: Chris REDMAN (University of Oxford, UK) and Terence LAO (Queen Mary Hospital, Hong Kong)

Complications of pregnancy – clinical translation of biomarkers
Sebastian ILLANES (University of Los Andes, Chile)

O4.1 Proton pump inhibitors quench the pathophysiological characteristics of pre-eclampsia in both human and mouse models and represent exciting novel candidate therapeutics
Natalie HANNAN (University of Melbourne, AUS)

O4.2 Morbid adherence of the placenta
Debra HELLER (Rutgers-New Jersey Medical School, USA)

O4.3 Complications of pregnancy – clinical translation of therapeutic interventions
Jon HYETT (Royal Prince Alfred Hospital Sydney, AUS)

10:00  Coffee Service

10:30  New Investigators Session 3 – Boulevard Auditorium
      Chairs: Natalie HANNAN (University of Melbourne, AUS) and Debra HELLER (Rutgers-New Jersey Medical School, USA)

NI 3.1 MRI measures of in vivo placental oxygenation differ between normal and fetal growth restricted pregnancies
Emma Ingram (University of Manchester, UK)

NI 3.2 Acoustic biomarkers of placental pathophysiology and adverse fetal outcome
Diana RIKNAGEL (Aalborg University, Denmark)

NI 3.3 Withdrawn

NI 3.4 An Integrated transcriptional, epigenetic, and clinical analysis of pre-eclamptic placentas
Katherine LEAVEY (University of Toronto, Canada)

NI 3.5 Integrative analysis of placental transcriptome organisation reveals highly conserved regulatory programs and points towards a pre-eclampsia gene cluster
Sam BUCKBERRY (The University of Adelaide, AUS)
## PROGRAM – Friday 11th September 2015

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<th>Time</th>
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<th>Chair(s)</th>
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<tr>
<td>12:00</td>
<td><strong>Keynote Lecture</strong> – Boulevard Auditorium</td>
<td>Boulevard Auditorium</td>
<td>Vicki CLIFFTON (Mater Research Institute, AUS) and James CUFFE (University of Queensland, AUS)</td>
<td>David OLSON (University of Alberta, CAN)</td>
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<td>Uterine – Placental Transitions for Birth and Fetal Development</td>
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<td>12:30</td>
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<td>14:00</td>
<td><strong>Keynote Lecture</strong> – Boulevard Auditorium</td>
<td>Boulevard Auditorium</td>
<td>Padma MURTHI (University of Melbourne, AUS) and Larry CHAMLEY (University of Auckland, NZ)</td>
<td>Tony PERKINS (Griffith University, AUS)</td>
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<td>Multinutrient supplementation and birth outcome</td>
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<td>14:30</td>
<td><strong>Workshops 9-12</strong> (Parallel sessions)</td>
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<td>9. Role of the placenta in fetal programming</td>
<td>(Room B1)</td>
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<td>10. Placental mitochondrial function</td>
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<td>11. Placental transport systems</td>
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<td>12. Placental immunology and infection</td>
<td>(Boulevard Auditorium)</td>
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<td>17:00</td>
<td><strong>Senior IFPA Award Lecture</strong> – Boulevard Foyer</td>
<td>Boulevard Foyer</td>
<td>Stacy ZAMUDIO (Hackensack University Medical Centre, USA) and Alicia JAWERBAUM (University of Buenos Aires, Argentina)</td>
<td>Anthony CARTER (University of Southern Denmark, DK)</td>
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<td>IFPASA Fetal membranes – a focus for the future or a legacy from the past?</td>
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<td>18:00</td>
<td><strong>ECR Balloon Debate</strong> – Boulevard Auditorium</td>
<td>Boulevard Auditorium</td>
<td>Larry Chamley (University of Auckland), Annette Staff (University of Oslo), Vicki Clifton (Mater Medical Research Institute) and Terry Morgan (Oregon Health and Science University).</td>
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<td>20:00</td>
<td><strong>Gala Dinner</strong> – Boulevard Room</td>
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BUSINESS MEETING SCHEDULE

Tuesday 8th September

9:00 - 12:30  IFPA Executive Meeting (by invitation)
              Concord Room

12:30 – 13:30 IFPA Executive Lunch (by invitation)
               Arbour Room

Wednesday 9th September

7:30 – 8:30  Awards Committee
               Boulevard Room 1

12:30 – 13:30 Regional AGMS
              PAA, Boulevard Auditorium; EPG, B1; JPA, B2; ANZPRA, B3

Thursday 10th September

7:30-8:30  TR/Placenta Editorial Board Meeting
           Boulevard Room 1

12:30 – 13:30 IFPA AGM
               Boulevard Auditorium

Friday 11th September

7:30 – 8:30  Awards Committee
             Boulevard Room 1

12:30 – 14:00 Final Poster Judging
              Boulevard Foyer
Parallel session 14:30 – 17:00

Workshop 1: Late Onset IUGR Surveillance and Monitoring

Organizers:
Jon HYETT (Royal Prince Alfred Hospital Sydney, Australia. Email: Jon@fetalmedicine.com)
Greg DUNCOMBE (The University of Queensland, International Health and Medical Services, Qhealth, Australia. Email: g.duncombe@uq.edu.au)

A workshop aiming to define research questions. This workshop will provide an alternative forum for communication between those delegates interested in translational application of basic science research into clinical practice. It aims to provide clinical direction for future basic science research and to encourage clinicians to explore the potential value of recent findings and technologies in clinical practice. The workshop will specifically focus on the potential impact of novel tests for late onset IUGR.

Confirmed speakers and preliminary titles/order are as follows:

14:30-14:50 Whole audience participation - Developing a mind map describing monitoring / interventions for late onset IUGR
Greg DUNCOMBE, University of Queensland, AUS

14:50-15:05 How should we define intrauterine growth restriction?
Jon HYETT, RPA Women and Babies, University of Sydney, AUS

15:05-15:20 Improving classification of placental disease
Roberto OREFICE, Australian National University, Canberra, AUS

15:20-15:40 Limitations of traditional biometry and potential for innovation in relation to late onset IUGR
Speaker (TBC)

15:40-16:00 Assessing uteroplacental and fetal haemodynamics in late onset IUGR
Sailesh KUMAR, University of Queensland, Australia

16:00-16:15 Using MRI to map placental function
Gabriele BOBEK, University of Western Sydney, AUS

16:15-16:35 Forget imaging – it’s all about genomic biomarkers
Claire WHITEHEAD, University of Melbourne, AUS

16:35-17:00 Whole audience participation - Back to the mind map – ranking research questions
Jon HYETT and Greg DUNCOMBE
WORKSHOPS – WEDNESDAY 9th SEPTEMBER

Parallel session 2:30 – 5:00
**Workshop 2: Nanomedicine Applications and Exosome Biology**

**Organizers:**
Jeff KEELAN (School of Women's and Infants' Health, The University of Western Australia, Australia. Email: jeff.keelan@uwa.edu.au)
Carlos SALOMON (UQ Centre for Clinical Research, University of Queensland, Australia. Email: c.salomongallo@uq.edu.au)

During the past decade, there has been an extraordinary explosion of research in the field of extracellular vesicles (EVs), especially in a specific type of EVs originating from endosomal compartments called exosomes. Exosomes are a specific subtype of secreted vesicles which are small (~30–120 nm) and very stable, released from a wide range of cells including the human placenta. This workshop will explore the recent advances in the design and application of nanocarriers for targeted drug delivery in pregnancy with a focus on placental nanovesicles uptake. In addition, the nature and content of endogenous placenta-derived nanovesicles (i.e. exosomes) will be presented and their potential prognostic and diagnostic applications will be discussed.

Confirmed speakers and preliminary titles/order are as follows:

14:30-14:50 Extracellular vesicle biogenesis: are they all the same?
Carlos SALOMON, UQ Centre for Clinical Research, University of Queensland, AUS

14:50-15:10 Immune-modulatory function of placenta-derived exosomes in normal and pre-eclamptic pregnancy
Claudia GÖHNER, University Medical Centre Groningen, NL

15:10-15:30 Therapeutic strategies involving nanoparticle-mediated drug delivery in pregnancy
Jeff KEELAN, The University of Western Australia, AUS

15:30-15:50 Targeting the Trophoblast; nanoparticle mediated placental gene therapy
Helen JONES, Cincinnati Children’s Hospital Medical Center, USA

15:50-16:10 Nanotherapeutics for targeted manipulation of placental growth
Lynda HARRIS, The University of Manchester, UK

16:10-16:30 Targeting the myometrium with nanoparticles
Roger SMITH, University of Newcastle, AUS

16:30-17:00 Question & Answer panel discussion
Brief concluding remarks
WORKSHOPS – WEDNESDAY 9th SEPTEMBER

Parallel session 2:30 – 5:00
Workshop 3: Placental Epigenetics

Organizers:
Karen MORITZ (School of Biomedical Sciences, The University of Queensland, QLD Australia. Email: k.moritz1@uq.edu.au)
Sam BUCKBERRY (The University of Western Australia, WA Australia. Email: sam.buckberry@uwa.edu.au)

Deciphering the role of the epigenome in regulating placental function will be critical to our understanding of how the environment can influence in utero development and pregnancy outcome. Numerous studies have already highlighted important links between altered epigenetic regulation in the placenta and diseases of gestation and early life. In this workshop, we will explore the intersection of nature and nurture in placental development and function and take a look at the recent advances in placental epigenetics.

Confirmed speakers and preliminary titles/order are as follows:

14:30-14:35      Introduction
                  Karen MORITZ, University of Queensland, AUS

14:35-14:55      Moving towards integrative epigenomic analyses
                  Sam BUCKBERRY, The University of Western Australia, AUS

14:55-15:20      Epigenetics, microRNAs and adverse environmental exposures in utero
                  Suyinn CHONG, Mater Research Institute, The University of Queensland, AUS

15:20-15:40      Imprinting and placental development
                  Courtney HANNA, University of British Columbia, CAN

15:40-16:00      DNA Methylation in the Placenta: Cause or Consequence
                  Tina Bianco-Miotto, The University of Adelaide, AUS

16:00-16:20      Atypical telomere length regulation in the human placenta
                  Richard SAFFERY, University of Melbourne, AUS

16:20-17:00      General Discussion

Parallel session 2:30 – 5:00
Workshop 4: Placental Pathology

IFPA – 2015 - Brisbane
Organizers:
Terry MORGAN (Department of Pathology, Oregon Health & Science University, USA. Email: morgante@ohsu.edu)
Yee KHONG (University of Adelaide, Australia. Email: yee.khong@adelaide.edu.au)

Objectives:
- The current obesity epidemic has led to an increase in the prevalence of gestational diabetes and may be a significant independent risk factor for preterm birth and stillbirth.
- The impact on developmental programming is only in the early stages of investigation.
- Placental pathology may provide important insights into key disease mechanisms.

Confirmed speakers and preliminary titles/order are as follows:

14:30-14:45 Introductory Remarks: Relationship between obesity and stillbirth
T. Yee KHONG, University of Adelaide, AUS

14:45-15:15 Placenta and obesity: Differences in reproductive immunology and uteroplacental pathology
Terry MORGAN, Oregon Health & Science University, USA

15:30-16:00 Effects of gestational weight gain on placental pathology on women who are overweight and obese: The results of the LIMIT study
Sarah CASH, University of Adelaide, AUS

16:15-16:45 Gaps in Knowledge and Future Directions
Martha LAPPAS, University of Melbourne, Victoria, AUS

16:45-17:00 Concluding Remarks
T. Yee Khong, University of Adelaide, AUS
WORKSHOPS – THURSDAY 10th SEPTEMBER

Parallel session 2:30 – 5:00
Workshop 5: Lipid Mediators and Placental Function

Organizers:
Denise HEMMINGS (University of Alberta, Canada. Email: denise.hemmings@ualberta.ca)
Christiane ALBRECHT (Institute of Biochemistry and Molecular Medicine, University of Bern, Switzerland. Email: christiane.albrecht@ibmm.unibe.ch)

We will work in small groups during this highly interactive workshop focusing on four areas in the placenta: 1) Regulation and Function of Lipid Transporters, 2) Lipid Transporter Substrates and Their Function, 3) Lipid Signalling and 4) Effective Tools and Models. Each attendee will be given the opportunity to actively contribute in two out of the four areas in a small group format. Summaries highlighting current understanding, the hot topics still to investigate and an extensive list of tools and models with identification of expert researchers willing to train and share their expertise with others will complete the final part of the workshop. Further details with specific questions and approaches for each small group discussion will be provided at the beginning of the workshop. We invite your input as we organize this workshop. While we cannot incorporate all ideas, we are certainly interested in hearing from those of you who want to participate in this workshop.

14:30 10-min overview of workshop topics with suggested areas for further discussion in each
Denise HEMMINGS and Christiane ALBRECHT

14:40 Participants will join their small group discussion of choice for an in depth discussion
facilitated by two of our colleagues. The discussion of each group will be captured in
each group, summarized and reported back in the final 40 minutes of this workshop

15:30 Participants will switch to their second small group discussion of choice

16:20 All participants will join together to hear summaries of all 4 group discussions provided
by the facilitators from each of the groups. Further discussion will follow if time allows.

Regulation and Function of Lipid Transporters
Discussion facilitated by Theresa POWELL (University of Colorado Anschutz Medical Campus,
USA) and Christian WADSACK (University of Graz, Austria)

Lipid Transporter Substrates and Their Function
Discussion facilitated by Alicia JAWERBAUM (University of Buenos Aires, Argentina) and
Denise HEMMINGS (University of Alberta, Canada)

Lipid Signalling
Discussion facilitated by Isabella CANIGGIA (Lunenfeld-Tanenbaum Research Institute, CAN)
and Ed JOHNSTONE (University of Manchester, UK)

Effective Tools and Models
Discussion facilitated by Christiane ALBRECHT (University of Bern, Switzerland) and Rohan
LEWIS (University of Southampton, UK)
Workshops – Thursday 10th September

Parallel session 2:30 – 5:00

Workshop 6: Biomarkers of Placental Function and Complications of Pregnancy

Organizer:
Sebastian ILLANES (Universidad de los Andes, Chile. Email: sillanes@uandes.cl)

The keystone to improving health outcomes remains the timely and accurate diagnosis of the predisposition to, or early detection of disease. Early detection of disease risk and onset is the first step in implementing efficacious treatment and improving patient outcome. In the context of population screening, the objective is to develop new tests that are informative of the risk of asymptomatic early pregnant women subsequently developing complications of pregnancy. Such tests inform clinical decision-making and provide an opportunity for timely and appropriate intervention. The performance of the test determines the quality of the information provided and ultimately the course of patient management. In this workshop the different speakers will focus in their experience in the development of biomarkers for early prediction of placental related diseases, with the idea of an ample discussion in how we translate basic science discoveries to the care of pregnant women.

Confirmed speakers and preliminary titles/order are as follows:

Co-chairs: Jenny MYERS / Sebastián ILLANES

14:30-14:55  Early prediction of GDM: targeting the long term consequences of the disease
Sebastián ILLANES, University of Los Andes, Chile

14:55-15:10  First trimester algorithm for the prediction of GDM
Paula CORREA, ECR, University of Los Andes, Chile

15:10-15:35  Prediction of Preeclampsia: Combining Clinical Risk and Biomarkers
Jenny MYERS - University of Manchester, UK

15:35-15:50  SNPs as biomarkers for pre-eclampsia in the SCOPE and other populations
Prabha ANDRAWEERA, - ECR The University of Adelaide, AUS

16:00-16:20  Novel biomarkers for fetal growth restriction: are we any closer to predicting adverse fetal outcomes?
Clare WHITEHEAD, University of Melbourne, AUS

16:20-16:40  Oral biomarkers in the prediction of placental originated diseases
Alejandra CHAPARRO - University of Los Andes, Chile

16:40-17:00  Lipid Mediator Profiling at Ultra-low Detection Limits using Advanced LCMS/MS Technology
Nigel GRIEVES, Shimadzu Australia
WORKSHOPS – THURSDAY 10th SEPTEMBER

Parallel session 2:30 – 5:00
Workshop 7: Xenobiotics and Endocrine Disruptors and Pregnancy

Organizers:
Padma MURTHI (Monash University, Australia. Email: padma.murthi@monash.edu)
Murray MITCHELL (UQ Centre for Clinical Research, University of Queensland, Australia. Email: murray.mitchell@uq.edu.au)

Endocrine disruptors are exogenous substances that alter endocrine function and consequently cause adverse health effects in an intact organism, its progeny, or subpopulations. They may do so by interfering with the production, release, transport, metabolism, binding, action, or elimination of natural hormones responsible for the maintenance of homeostasis and the regulation of developmental processes. Pregnant women are exposed to various potential endocrine disrupting chemicals through diet, medication use, occupational or environmental activities and other lifestyle factors. Epidemiological studies have associated altered pregnancy and fetal outcomes with exposure to contaminants such as heavy metals, polychlorinated biphenyls, dioxins and pesticides. The main focus of our workshop is to discuss how endocrine disruption leads to change in endocrine-regulated physiology in utero and contributes to adverse pregnancy and neonatal outcomes.

Confirmed speakers and preliminary titles/order are as follows:

14:30-14:50 Opening remarks and Transplacental transfer of endocrine disruptors
Murray MITCHELL, University of Queensland, AUS

14:55-15:05 Maternal exposure to Bisphenol A in pregnancy and adverse gestational outcomes
Bridget MAHER, University of Queensland, AUS

15:10-15:20 Bisphenol A in pregnancy: controversies around measurements of maternal & fetal exposure and their biological interpretation
Jeff KEELAN, The University of Western Australia, AUS

15:25-15:35 Antidepressant alters placental serotonin and estrogen system
Cathy VAILLANCOURT, Université du Québec, CAN

Richard SAFFERY, Murdoch Childrens Research Institute, Uni Melbourne, AUS
### WORKSHOPS – THURSDAY 10th SEPTEMBER

<table>
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<th>Time</th>
<th>Session</th>
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| 15:55-16:05 | Sex specific differences in the placental response to glucocorticoids-mechanisms that may be altered or influenced by endocrine disruption  
Vicki CLIFFTON, Mater Research Institute, Brisbane, AUS |
| 16:10-16:20 | Bisphenol exposure alters placental growth control genes and contributes to abnormal trophoblast and endothelial cell function in vitro  
Padma MURTHI, Monash University, AUS |
| 16:20-16:45 | Question & Answer panel discussion |
| 16:45-17:00 | Concluding remarks by Murray MITCHELL and Padma MURTHI |
Parallel session 2:30 – 5:00

Workshop 8: Stem Cells of the Fetomaternal Interface

Organizers:
Bill KALIONIS (Department of Perinatal Medicine, Royal Women’s Hospital, and Department of Obstetrics and Gynaecology, University of Melbourne, Parkville, Australia. Email: bill.kalionis@thewomens.org.au)

Larry CHAMLEY (Department of Obstetrics & Gynaecology, The University of Auckland, New Zealand. Email: l.chamley@auckland.ac.nz)

Mohamed ABUMAREE (King Saud Bin Abdulaziz University for Health Sciences/King Abdullah International Medical Research Center, Riyadh Saudi Arabia. Email: mohamedabumaree@hotmail.com)

The placenta and uterus are sources of many stem cell populations including cytotrophoblast, mesenchymal, endothelial, haematopoietic and epithelial stem cells. Some of these stem cell populations are intensively studied for their potential use in reparative and regenerative medicine. Accumulating evidence suggests that stem cells are an important new player in the normal and pathological development of the placenta and uterus.

This workshop will showcase both basic and applied research on placental and uterine stem cells. We hope this workshop will be a venue for the exchange of ideas and a starting point for discussion of the many answered questions regarding the role of stem cells.

14:30-14:35 Introduction

14:35-14:55 Why is it a challenge to isolate placental stem cells?
Rebecca PELEKANOS, UQ Centre for Clinical Research, Brisbane, AUS

14:55-15:10 Identification of a trophoblast side-population with low Hoechst staining in human term placentae: Are these Trophoblast Stem Cells?
Teena GAMAGE, The University of Auckland, New Zealand

15:10-15:30 Amnion cell mediated immune modulation during lung injury: controlling the regulatory T cell response
Rebecca LIM, Monash Institute of Medical Research, The Ritchie Centre, AUS

15:30-15:45 The consequences of the interaction between chorionic villous mesenchymal stem cells and human natural killer cells
Abdulaziz ALMOTERY, King Saud Bin Abdulaziz University for Health Sciences, Saudi Arabia
15:45-16:05  Human placental decidua basalis (DBMSCs) modulate the expression of receptors important in mediating the immunosuppressive functions of macrophages in cancer
Mohamed ABUMAREE, King Saud Bin Abdulaziz University for Health Sciences, Saudi Arabia

16:05-16:20  Mesenchymal stem/stromal cells migrate and localise to vascular structures following transplant into human placental explants
Jo JAMES, The University of Auckland, New Zealand

16:20-16:35  Mesenchymal stem cell co-transplantation enhanced the vessel formation capacity of placental derived endothelial colony forming cells in wild type and immunodeficient mice
Abbas Shafiee, University of Queensland and Queensland University of Technology

16:35-16:55  Question & Answer panel discussion

16:55-17:00  Brief concluding remarks
Parallel session 2:30 – 5:00

Workshop 9: Mechanistic role of the placenta in fetal programming

Organizers:
Claire ROBERTS (Robinson Research Institute, University of Adelaide, Australia. Email: claire.roberts@adelaide.edu.au)
Kent THORNBURG (The Moore Institute, Oregon Health & Science University, USA. Email: thornbur@ohsu.edu)

No self-respecting placentologist would believe that the placenta is simply a conduit through which maternal nutrients and oxygen are delivered to the fetus and metabolic wastes are excreted. David Barker was a pioneer in thinking about the role of maternal nutrition in fetal programming for later disease. However, in the last several years of his life David championed the idea that placental shape was a marker of fetal programming for adult disease. This workshop will explore the mechanisms by which the placenta is instrumental in programming and also discuss mechanisms reflected in placental shape at delivery.

Confirmed speakers and preliminary titles/order are as follows:

14:30-14:40  Placenta: much more than just a conduit
Claire ROBERTS, University of Adelaide, AUS

14:45-15:00  Placental shape and adult disease
Kent THORNBURG, Oregon Health Science University, USA

15:05-15:20  Invasion and recruitment of spiral arteries in development of placental size and shape
Graham BURTON, University of Cambridge, UK

15:25-15:40  Mechanisms in placental development that program the fetus
Terry MORGAN, Oregon Health Science University, USA

15:45-16:00  Why study the delivered placenta?
Carolyn SALAFIA, Placental Analytics Institute for Basic Research, USA

16:05-16:20  What can animal models tell us of the role of the placenta in fetal programming?
Hayley Dickinson, Hudson Institute of Medical Research, Monash University, AUS

16:25-16:55  Question & Answer panel discussion

16:55-17:00  Brief concluding remarks
Kent THORNBURG, Oregon Health Science University, USA
Parallel session 2:30 – 5:00
Workshop 10: Placental mitochondrial function

Organizers:
Les MYATT (Center for Pregnancy and Newborn Research, University of Texas Health Science Center San Antonio, USA. Email: myattl@uthscsa.edu)
Tony PERKINS (Griffith University, Gold Coast campus, QLD Australia. Email: a.perkins@griffith.edu.au)

In addition to supplying cellular energy mitochondria have many important roles in placental function including regulation of membrane potential, apoptosis, calcium signaling, steroid synthesis and production of reactive oxygen species. There is now accumulating evidence for defects in mitochondrial respiration/function in pregnancies complicated by preeclampsia, growth restriction, obesity and gestational diabetes, although a mechanistic role in these disorders has yet to be shown. Attention is now focusing on strategies to improve placental mitochondrial function with the objective of improving pregnancy outcomes. This workshop will discuss mitochondrial structure/function, methods for studying mitochondrial respiration and function, evidence for mitochondrial dysfunction in pregnancy pathologies and therapeutic strategies (anti-oxidants, microRNAs).

14:30-14:40 Introductory remarks
Tony PERKINS, Griffith University, Gold Coast, AUS & Les MYATT, University of Texas Health Science Center, USA

14:40-15:05 Overview - Mitochondrial structure function, methods
Holly VAN REMMEN, Oklahoma Medical Research Foundation, USA

15:05-15:30 Dysfunction in pregnancy pathologies (obesity, GDM, preeclampsia, stillbirth)
Leslie MYATT, University of Texas Health Science Center, San Antonio, USA

15:30-15:55 Hypoxia and mitochondria
Amanda SFERRUZZI-PERRI, University of Cambridge, UK

15:55-16:20 Therapeutic Strategies, antioxidants
Olivia HOLLAND, Griffith University, Brisbane, AUS

16:20-16:50 Question & Answer panel discussion
If mitochondrial function is compromised is it cause or consequence? What are the consequences for placental function? If it’s broken should we fix it and what approach should we use: targeted vs non-specific?

16:50-17:00 Summary and conclusions
Tony PERKINS, Les MYATT
WORKSHOPS – FRIDAY 11th SEPTEMBER

Parallel session 2:30 – 5:00

Workshop 11: Placental Transport and its Regulation

Organizers:
Paul DAWSON (Mater Research Institute, University of Queensland, Australia. Email: paul.dawson@mater.uq.edu.au)
Colin SIBLEY (Maternal and Fetal Health Research Centre, University of Manchester, England. Email: colin.sibley@manchester.ac.uk)

A workshop aimed at:
1) Addressing the biological roles and regulation of placental transport, and the impact of perturbed transport on placental, fetal and maternal physiology;
2) Increasing awareness of the known/potential clinical consequences of impaired placental transport;
3) Bringing together research teams to foster collaboration and direct future biomedical and clinical research on placental transport systems.

14:30-14:35 Welcome Colin Sibley & Paul Dawson
14:35-14:55 Adenosine transport and receptors / insulin signaling axis in the placenta endothelium from normal or gestational diabetes pregnancies Luis SOBREVI A, The Pontificia Universidad Católica de Chile, Chile
14:55-15:15 Nitrate and Uteroplacental Function Elizabeth COTTRELL, Maternal and Fetal Health Research Centre, University of Manchester, UK
15:15-15:35 Regulation of placental amino acid transfer by flow, transport and metabolism Rohan LEWIS – Faculty of Medicine, University of Southampton, UK
15:35-15:55 Placental sulfate transport: recent lessons from mouse studies David SIMMONS, University of Queensland, AUS
15:55-16:15 Fatty acids are both nutrients and signaling molecules: why do we know so little about something so important? Theresa POWELL, University of Colorado Anschutz Medical Campus, USA
16:15-16:55 Question & Answer panel discussion
   1. What are the next big issues for placental transport?
   2. What placental transport systems should we try to understand better?
16:55-17:00 Brief concluding remarks
Parallel session 2:30 – 5:00

**Workshop 12: Placental Immunobiology and Infection**

**Organizers:**
Philippe BOEUF (Burnet Institute, Australia. Email: philippe.boeuf@burnet.edu.au)
Martha LAPPAS (The University of Melbourne, Australia. Email: mlappas@unimelb.edu.au)

The placenta forms a physical and immunological barrier, protecting the fetus from the deleterious effects of maternal infection and inflammation. In this workshop, we will present and discuss examples of infectious and/or inflammatory conditions that negatively impact on placental development and functions.

Confirmed speakers and preliminary titles/order are as follows:

14:30-14:40 Welcome and introduction
Martha LAPPAS, The University of Melbourne, & Philippe BOEUF, Burnet Institute, Melbourne

14:40-15:10 T cell immunoglobulin and mucin domain (TIM)3 expression mediates the severity of viral-induced inflammation in fetal membranes
Stella LIONG, The University of Melbourne, AUS

15:10-15:40 Exogenous myostatin and varied oxygen tensions alters the release of cytokines from first trimester placental explants
Hassendrini PEIRIS, The University of Queensland, AUS

15:40-16:10 Induction of apoptosis in human term syncytiotrophoblast by stimulation via TLR3
Kenichiro MOTOMURA, National Research Institute For Child Health And Development, Japan

16:10-16:40 The placental microbiome; microbial presence in nature's transplant
Elise PELZER, Queensland University of Technology, AUS

16:40-16:55 Panel discussion

16:55-17:00 Brief concluding remarks
Anatomy and Pathology

P1.01 Placental glycogen deposition: a cause or consequence of impaired placental development contributing to fetal growth restriction?
Karen Moritz¹, James Cuffe¹, David Simmons¹
¹University of Queensland, St Lucia, Queensland, Australia

P1.02 Term PCSVN features are predicted by ultrasound measures at 11-14 weeks
Carolyn Salafia¹, Theresa Girardi¹², Nadav Schwartz³, Ruchit Shah¹, George Merz², Dawn Misra⁴, Richard Miller⁵⁶, Philip Katzmann⁵⁶, John Moye⁵
¹Placental Analytics, Larchmont, NY, USA, ²Placental Modulation, Institute for Basic Research, Staten Island, NY, USA, ³Hospital of the University of Pennsylvania, Division of Maternal Fetal Medicine, Philadelphia, PA, USA, ⁴Department of Family Medicine and Public Health Sciences, Wayne State University School of Medicine, Detroit, MI, USA, ⁵Obstetrics and Gynecology and Pathology, University of Rochester School of Medicine, Rochester, NY, USA, ⁶National Children's Study, Eunice Kennedy Shriver National Institute of Child Health & Human Development, Bethesda, MD, USA

Pathogenesis of Twin Reversed Arterial Perfusion Sequence Is Different From that of Twin-To-Twin Transfusion Syndrome: Clinicopathological Assessment of 32 Cases of Multiple Pregnancies With Acardia
Taisuke Sato¹³, Kentaro Matsuoka¹, Yuki Ito¹³, Nagayoshi Umehara²³, Aikou Okamoto³, Haruhiro Sago²
¹Department of Pathology, National Center for Children Health and Development, Tokyo, Japan, ²Center of Maternal-Fetal, Neonatal and Reproductive Medicine, National Center for Children Health and Development, Tokyo, Japan, ³Department of Obstetrics and Gynecology, The Jikei University School of Medicine, Tokyo, Japan

Immunohistochemical analysis of STAT1 and STAT5 in human placental disorders
Maja Weber¹, Steffi Treppschuh¹, Ekkehard Schleußner¹, Justine S. Fitzgerald¹, Udo R. Markert¹
¹University Hospital Jena, Department of Obstetrics and Gynecology, Placenta-Lab, Jena, Germany

The effect of antenatal steroids on the maturation of the villi in the placenta - A retrospective study on preterm births between 23 and 27 weeks of gestation in a Stockholm cohort
Miranda Uni Bergström¹, Nikos Papadogiannakis²³, Magnus Westgren¹, Marie-Therese Vinnars¹⁴
¹Department of Clinical Science, Intervention and Technology, Division of Obstetrics and Gynecology, Karolinska Institutet, Stockholm, Sweden, ²Department of Laboratory Medicine, Division of Pathology, Karolinska Institutet, Stockholm, Sweden, ³Karolinska University Hospital, Stockholm, Sweden, ⁴Örnsköldsviks Hospital, Örnsköldsvik, Sweden

Modelling the relationship between maternal blood flow and villus tree density in normal human pregnancy.
Rojan Saghian¹, Joanna James², Sally L. Collins³, Merryn Tawhai¹, Alys R. Clark¹. ¹Auckland Bioengineering Institute, University of Auckland, Auckland, New Zealand; ²Department of Obstetrics & Gynaecology, University of Auckland, Auckland, New Zealand; ³The faculty of Medical Science, University of Oxford, Oxford, UK.

Angiogenesis and Vasculature

P1.07 The influence of the G protein coupled receptor 55 on human placental venous endothelial cells
Julia Kremshefer¹, Monika Siwetz¹, Monika Sundl¹, Ingrid Lang¹, Berthold Huppertz¹, Martin Gauster¹
¹Medical University of Graz, Graz, Austria
P1.08 Chorionic surface vascular networks (CSVN) in high and low ASD risk placentas: caliber, vessel count and Murray's Law
Ruchit Shah1,2, Carolyn Salafia1,2, Theresa Girardi1,2, George Merz1, Dawn Misra3, Craig Newschaffer4, Richard Miller5,6, John Moye7
1Placental Modulation, Institute for Basic Research, Staten island, NY, USA, 2Placental Analytics, Larchmont, NY, USA, 3Department of Family Medicine and Public Health Sciences, Wayne State University School of Medicine, Detroit, MI, USA, 4AJ Drexel Autism Institute, Drexel University, Philadelphia, PA, USA, 5Obstetrics and Gynecology and Pathology, University of Rochester School of Medicine, Rochester, NY, USA, 6National Children's Study, Eunice Kennedy Shriver National Institute of Child Health & Human Development, Bethesda, MD, USA

P1.10 Novel trophoblast-released anti-angiogenic pigment epithelium derived factor (PEDF) limits placental angiogenesis
Jelena Loegl1,2, Erika Nussbaumer3, Ursula Hiden2, Silvija Cvitic2, Ingrid Lang1, Berthold Huppertz1, Gernot Desoye1, Institute of Cell Biology, Histology and Embryology, Graz, Austria, 2Department of Obstetrics and Gynecology, Graz, Austria

Comparative/ Animal Models

P1.11 Identification of seven different isoforms of the glucocorticoid receptor in Guinea Pig placenta: relationship to preterm delivery, sex and betamethasone exposure
Zarqa Saii1, Rebecca Dyson1,2, Hannah Palliser1, Ian Wright1,2, Nick Lu3, Vicki Clifton1
1Robinson Research Institute, Adelaide, Australia, 2Mothers and Babies Research Centre, Newcastle, Australia, 3Illawarra Health and Medical Research Institute, Wollongong, Australia, 4Northwestern University, Chicago, USA

P1.12 Characterisation of the DBA/2J-mated CBA/CaH female murine model of pregnancy loss and IUGR
Kelly Mckelvey1, Vanessa Yenson1, Anthony Ashton1, Jonathan Morris1,2, Sharon McCracken1
1Division of Perinatal Medicine, Kolling Institute, University of Sydney at Royal North Shore Hospital, St Leonards, NSW, Australia, 2Department of Obstetrics and Gynaecology, Royal North Shore Hospital, St Leonards, NSW, Australia

P1.13 High Resolution Placental Maps using MRI and Amira™ Visualization Software
Gabriele Bobek1, Tim Stait-Gardner1, William Price1, Annemarie Hennessy1
1University of Western Sydney, Sydney, Australia

P1.14 Microbiota and the gut-placenta axis in non-human primates (Papio spp.)
Natalia Schlabritz-Lousetvitch1, Xuan Ji Li2, Christopher Rensing3, Bill Taylor3, Caitlin Costelle3, Gene Hubbard4, Gary Ventolini5, Edward Dick5
1Texas Tech University Health Sciences Center at the Permian Basin, Odessa, TX, USA, 2University of Copenhagen, Denmark, 3University of Tennessee Health Sciences Center, Memphis, TN, USA, 4University of Texas health Sciences Center at San Antonio, San Antonio, TX, USA, 5Texas Biomedical Research Institute, San Antonio, TX, USA

P1.15 The near term shift of syndecan protein to an epithelial localization suggests an involvement in placental maturation and release of bovine fetal membranes
Nina Hambruch1, Simone Kumstel1, Vibeke Dantzer2, Christiane Pfarrer1
1Department of Anatomy, University of Veterinary Medicine Hannover, Hannover, Germany, 2Faculty of Health and Medical Sciences, Department of Veterinary Clinical and Animal Sciences, University of Copenhagen, Copenhagen, Denmark

P1.16 Placental-specific hypoxia inducible factor-1 alpha as a model of preeclampsia
Renee Albers1, Melissa Kaufman2, Chanel Keoni3, Martha Hughes2, Bryony Natale2, David Natale2, Thomas Brown1
1Wright State University, Dayton, Ohio, USA, 2University of California San Diego, San Diego, California, USA
Gene Expression and Genomics

P1.17 Adapations to hypoxia during pregnancy: the blind mole rat, Spalax, placenta transcriptome
Derek Wildman1, Michael McGowen2, William Gundling3, Eviatar Nevo3
1University of Illinois, Urbana, IL, USA, 2Queen Mary University of London, London, UK, 3University of Haifa, Haifa, Israel

P1.18 Variability in the expressions of Aurora kinases (AURK) in pre-eclamptic placentae
Reham Balahmar1, Shiva Sivasubramaniam1
1NTU, Nottingham, UK

P1.19 Placenta specific extracellular vesicles’ uptake in primary endothelial cells and effect on gene expression
Tina Cronqvist1, Dione Tannetta1, Stefan Hansson1, Mary Familani1
1Clinical Sciences in Lund, Lund, Sweden, 2University of Oxford, Oxford, UK, 3School of Biosciences, University of Melbourne, Parkville, Australia

P1.20 Antenatal glucocorticoid treatment - Validation of internal reference genes in the human placenta
Hanna Gütling1, Massimo Bionaz2, Deborah M Sloboda3, Loreen Ehrlich1, Wolfgang Henrich1, Andreas Plagemann1, Thorsten Braun1
1Department of Obstetrics and Division of Experimental Obstetrics, Charité - University Berlin, Berlin, Germany, 2Animal and Rangeland Sciences, Oregon State University, Corvallis, Oregon, USA, 3Departments of Biochemistry and Biomedical Sciences, Obstetrics & Gynecology and Pediatrics, McMaster University, Hamilton, Canada

P1.21 Placental development in mouse and human: a comparative study of gene expression across gestation
Francesca Soncin1, Katharine Nelson1, David Natale1, Louise Laurent1, Mana Parast1
1University of California San Diego, La Jolla, California, USA

Hormones and Growth Factors

P1.22 IGF-I intracellular signalling in the human placenta is dependent on clathrin- and caveolin-dependent endocytosis
Magdalena Karolczak-Bayatti1, Lynda Harris1, Melissa Westwood1, John Aplin1
1The University of Manchester, Manchester, UK

P1.23 Reduction in placental AKT1 expression in a mouse model of fetal growth restriction is alleviated by nanoparticle-mediated Trophoblast-specific IGF-1 gene therapy
Kathryn Owens1, Weston Trojan1, Helen Jones1
1Cincinnati Children’s Hospital Medical Center, Cincinnati, Ohio, USA

P1.24 Rapid method for Growth Hormone Receptor exon 3 delete (GHRD3) SNP genotyping from archival human placental samples
Rebecca Pelekanos1, Varda Sardesai1, Marloes Dekker Nitert1,2, Leonie Callaway2, Nicholas Fisk1,3, Penny Jeffery3
1The University of Queensland, UQ Centre for Clinical Research, Brisbane, Qld, Australia, 2The University of Queensland, School of Medicine, Brisbane, Qld, Australia, 3Centre for Advanced Prenatal Care, Royal Brisbane & Women’s Hospital, Brisbane, Qld, Australia, 4Queensland University of Technology, Translational Research Institute, Brisbane, Qld, Australia

P1.25 The role of the embryonic phosphoinositol kinase (pi3k) p110a in regulating placental phenotype and fetal growth
Amanda Sferruzzi-Perri, Ionel Sandovici, Abigail Fowden, Miguel Constancia.
Centre for Trophoblast Research, University of Cambridge, Cambridge, UK
Implantation and Invasion

P1.26 Epac signal enhances decidual prolactin expression via C/EBPβ expression in human endomerial stromal cells
Kazuhiro Tamura1, Mikihiro Yoshie1, Kazuya Kusama1,2, Kazuhiko Imakawa1, Toshihiro Sakurai2, Hanako Bai1, Hirotaka Nishi1, Naoko Kuwabara1, Keiichi Isaka2, Eiichi Tachikawa1
1Dept. of Endocrine and Neural Pharmacology, Tokyo University of Pharmacy and Life Sciences, Tokyo, Japan, 2Laboratory of Theriogenology and Animal Breeding, Graduate School of Agricultural and Life Sciences, The University of Tokyo, Tokyo, Japan

P1.27 Placenta Previa reduced in pregnancies complicated by maternal hepatitis B virus infection
Terence Lao1, Daljit Sahota1
1The Chinese University of Hong Kong, Shatin, Hong Kong, China

P1.28 The epithelial-mesenchymal transition in extravillous trophoblast differentiation: role in abnormally invasive placenta
Nicholas Illsley1, Abdulla Al-Khan1, Sonia DaSilva-Arnold1, Margaret Petroff2, Stacy Zamudio1
1Hackensack University Medical Center, Hackensack, NJ, USA, 2Michigan State University, East Lansing, MI, USA

P1.29 Variations in suture techniques at cesarean section and its relation to complications in subsequent pregnancies
Seiji Sumigama1, Yoshinori Moriyama1, Tomoko Nakano1, Hiroyuki Tsuda1, Tomomi Kotaki1, Yumiko Ito1, Shima Hirako1, Hiroaki Moroi1, Kenji Imai1, Fumitaka Kikkawa1
1Nagoya University Graduate School of Medicine, Nagoya, Japan

P1.30 Two cases of gestational trophoblastic neoplasia with multiple molar emboli-like lung lesions after evacuation of hydatidiform mole
Yuko Tanizaki1, Madoka Yamamoto1, Shigetaka Yagi1, Naoyuki Iwahashi1, Tamaki Yahata1, Sakiko Nanjo1, Mika Mizoguchi1, Aya Kobayashi1, Michihisa Shiro1, Nami Ota1, Yasushi Mabuchi1, Sawako Minami1
1Department of Obstetrics and Gynecology, Wakayama Medical University, Wakayama, Japan

P1.31 Heparin is strongly recommended for juvenile women with recurrent pregnancy loss of caused by anti-phospholipid antibodies compared with thrombophilia
Michiko Suzuki1
1The Jikei University School of Medicine, Tokyo, Japan

P1.32 The expression of ADAM (a Disintegrin and Metalloproteinase) genes in human first-trimester villous and extravillous trophoblast cells
Hironori Takahashi1,2, Dongwei Zhao1, Akioide Okuchuki1, Shigeki Matsubara1, Toshihiro Takizawa1
1Jichi Medical University, Tochigi, Japan, 2Nippon Medical School, Tokyo, Japan

P1.33 The effects of angiogenic growth factors on first trimester trophoblast invasion and proliferation
Prabha Andraweera1, Amanda Highet1, Gary Heinemann1, Claire Roberts1
1School of Paediatrics and Reproductive Health, The Robinson Research Institute, University of Adelaide, Adelaide, Australia

Infection and Inflammation

P1.34 The placental microbiome; microbial presence in nature's transplant
Elise Pelzer1, Flavia Hugens1, Philip Hugenholtz2, Rohan Lourie2, Kenneth Beagley1, Ross Turner4
1Queensland University of Technology, Brisbane, Australia, 2Australian Centre for Ecogenomics, Brisbane, Australia, 3Mater Pathology, Brisbane, Australia, 4The Wesley Hospital, Brisbane, Australia
P1.35  
Is diversification of the endometrial microbiome significant for reproductive success?  
Elise Pelzer1, Dana Willner2, Flavia Huygens1, Melissa Buttini1  
1Queensland University of Technology, Brisbane, Australia, 2Australian Centre for Ecogenomics, Brisbane, Australia

P1.36  
TNF-α upregulates secretion of GM-CSF, CCL5 and IL-10 by human first trimester placenta  
Monika Siwetz1, Amin El-Heliebi1, Astrid Blaschitz1, Ursula Hiden2, Gernot Desoye3, Berthold Huppertz1, Martin Gauster1  
1Medical University Graz, Institute of Cell Biology, Histology and Embryology, Graz, Austria, 2Medical University Graz, Department of Obstetrics and Gynaecology, Graz, Austria

P1.37  
Inhibitory action of lipopolysaccharide-induced caspase-1 expression by alpha-1-antitrypsin in human trophoblasts  
Takanori Okubo1, Kazuhiro Tamura1, Mikihiro Yoshie1, Gen Ishikawa2, Akihito Nakai2, Toshiyuki Takeshita2, Toshiki Matsutani2, Wakanai Ohneda1, Naoko Kuwabara1, Eichi Tachikawa1  
1Dept. of Endocrine and Neural Pharmacology, Tokyo University of Pharmacy and Life Sciences, Tokyo, Japan, 2Division of Reproductive Medicine, Perinatology and Gynecologic Oncology, Nippon Medical School, Tokyo, Japan

P1.38  
Relationship between placental hepatitis B virus (HBV) infection with TLR3 and TNF-α expression in gravidae screened positive for hepatitis B virus infection  
Bo Wah Leung1, Terence, Tzu-Hsi Lao1  
1The Chinese University of Hong Kong, Shatin, Hong Kong

P1.39  
Maternal administration of hydrogen water might be protective for the LPS-induced preterm labor  
Tomoko Nakano1, Tomomi Kotani1, Hiroyuki Tsuda1, Seiji Sumigama1, Fumitaka Kikkawa1  
1Nagoya University, Nagoya, Japan

P1.40  
Induction of apoptosis in human term syncytiotrophoblast by stimulation via TLR3  
Kenichiro Motomura1,2, Naoko Okada1, Akio Matsuda1, Haruhiko Sago2, Hirohisa Saito1, Kenji Matsumoto1  
1National Research Institute for Child Health and Development, Tokyo, Japan, 2National Center for Child Health and Development, Tokyo, Japan

P1.41  
Expression of HIV-1 p24 and CD4 receptor on syncytiotrophoblast and fetal circulation in mother to child transmission of HIV  
Vinogrin Dorsamy1, Camille Vallen1, Jagadisa Moodley2, Thajasvarie Naicker2  
1Optics and Imaging Centre, UKZN, Durban, KwaZulu-Natal, South Africa, 2Women's Health, UKZN, Durban, KwaZulu-Natal, South Africa

P1.42  
Correlation between Intrapartum Maternal Temperature and Placental Histologic Evidence of Inflammation  
Morgen Doty1, Carolyn Salafia2, Susan Shen-Schwarz1, Edwin Guzman1, Claire Alexander1, Sarah Boulton1  
1Saint Peter's University Hospital, New Brunswick, NJ, USA, 2Placental Analytics, Larchmont, NY, USA

P1.43  
Correlation between Clinical Diagnosis of Chorioamnionitis and Placental Histologic Evidence of Inflammation  
Morgen Doty1, Carolyn Salafia2, Susan Shen-Schwarz1, Edwin Guzman1, Claire Alexander1, Sarah Boulton1  
1Saint Peter's University Hospital, New Brunswick, NJ, USA, 2Placental Analytics, Larchmont, NY, USA
Stem Cells

P1.44 Mesenchymal stem/stromal cells migrate and localise to vascular structures following transplant into human placental explants
Sonia Srinivasan¹, Larry Chamley¹, Jo James¹
¹University of Auckland, Auckland, New Zealand

P1.45 Isolation of fetal chorionic villi-derived mesenchymal stem/stromal cells from the human term placenta – why is it a challenge?
Varda Sardesai¹, Nicholas Fisk¹,², Rebecca Pelekanos¹
¹The University of Queensland, Centre for Clinical Research, Brisbane, Qld, Australia, ²Centre for Advanced Prenatal Care, Royal Brisbane & Women’s Hospital, Brisbane, Qld, Australia

P1.46 Human amnion epithelial cells improved pulmonary hypertension associated with bronchopulmonary dysplasia
Dandan Zhu¹, Ruth Muljadi¹, Camden Lo², Kristin Elgass², Euan Wallace¹,², Rebecca Lim¹,²
¹The Ritchie Centre, Clayton, Australia, ²Monash University, Clayton, Australia

P1.47 Placenta derived Mesenchymal Stromal Cells (PDMSCs) Modulate HIF-1a, VEGF and JunB genes in ovarian cancer cells
Domenica Giuffrida¹, Cristian Zenerino¹, Rossella Barrile¹, Anna Maria Nuzzo¹, Tullia Todros¹, Alessandro Rollo¹
¹Dept. of Surgical Sciences, University of Turin, Turin, Italy

P1.48 Amnion cell mediated immune modulation during lung injury: controlling the regulatory T cell response
Jean Tan¹, Shawn Tan¹, Ruth Muljadi¹, Siow Teng Chan¹, Euan Wallace¹,², Rebecca Lim¹,²
¹MIMR-PHI Institute, Clayton, Victoria, Australia, ²Monash University, Clayton, Victoria, Australia

P1.49 Human placental decidua basalis (DBMSCs) modulate the expression of receptors important in mediating the immunosuppressive functions of macrophages in cancer
Mohamed Abumaree¹,²,³, Fayaz Abomaray³,⁴, Khaled Al Saad⁵,⁶, Dunia Jawdat⁷, Abdulaziz Al Khaldi⁸, Ahmed Al Askar⁸, Seham Al Harthy⁸, Abdulmohsen Alkushi⁹,², Bill Kalionis⁹,³, Mohammed Al Jumah¹,²
¹King Saud Bin Abdulaziz University for Health Sciences, College of Science and Health Professions, Riyadh 11481, Saudi Arabia, ²King Abdullah International Medical Research Center, Riyadh 11426, Saudi Arabia, ³King Abdulaziz Medical City, Department of Pathology, Riyadh 11426, Saudi Arabia, ⁴King Abdulaziz Medical City, Division of Cardiac Surgery, Riyadh 11426, Saudi Arabia, ⁵King Abdulaziz City for Science and Technology, Riyadh 11442, Saudi Arabia, ⁶University of Melbourne, Department of Obstetrics and Gynaecology and Department of Perinatal Medicine Pregnancy Research Centre, Royal Women’s Hospital, Parkville, Victoria, Australia

P1.50 Phenotypic and functional characterization of mesenchymal stem cells from decidua parietalis of human term placenta
Mohamed Abumaree¹,²,³, Najla Alshehri², Abdulaziz Almutairi³, Fayaz Abomaray², Ahmed Al Askar², Abdulmohsen Alkushi⁴,², Bill Kalionis³, Mohammed Al Jumah²
¹King Saud Bin Abdulaziz University for Health Sciences, College of Science and Health Professions, P.O. Box 3660, Riyadh 11481, Mail Code 3124, Saudi Arabia, Riyadh 11481, Saudi Arabia, ²King Abdullah International Medical Research Center, Riyadh 11481, Saudi Arabia, ³King Saud Bin Abdulaziz University for Health Sciences, College of Applied Medical Sciences, Riyadh 11481, Saudi Arabia, ⁴University of Melbourne, Department of Obstetrics and Gynaecology and Department of Perinatal Medicine Pregnancy Research Centre, Royal Women’s Hospital, Parkville, Victoria, Australia
P1.51 Phenotypic and functional characterization of mesenchymal stem cells from decidua basils of human term placenta

Mohamed Abumaree1,2, Fawaz Abomaray2, Khaled Al Saad2,3, Dunia Jawdar2, Abdulaiz Al Khaldi4, Ahmed Al Askar7, Seham Al Harthy5, Abdulmohesen Alkushi1,4, Bill Kalionis6, Mohammed Al Jumah2  
1King Saud Bin Abdulaziz University for Health Sciences, College of Science and Health Professions, Riyadh 11426, Saudi Arabia, 2King Abdullah International Medical Research Center, Riyadh 11426, Saudi Arabia, 3King Abdulaziz Medical City, Department of Pathology, Riyadh 11426, Saudi Arabia, 4King Abdullah International Medical Research Center, Riyadh 11426, Saudi Arabia, 5University of Melbourne, Department of Obstetrics and Gynaecology and Department of Perinatal Medicine Pregnancy Research Centre, Royal Women’s Hospital, Parkville, Australia

P1.52 The consequences of the interaction between chorionic villous mesenchymal stem cells and human natural killer cells

Mohamed Abumaree1,2, Abdulaiz Almutairi3, Najla Alshehri2, Fawaz Abomaray2, Ahmed Al Askar2, Abdulmohesen Alkushi1,4, Bill Kalionis6, Mohammed Al Jumah2  
1King Saud Bin Abdulaziz University for Health Sciences, College of Science and Health Professions, Riyadh 11426, Saudi Arabia, 2King Abdullah International Medical Research Center, Riyadh 11426, Saudi Arabia, 3King Saud Bin Abdulaziz University for Health Sciences, College of Applied Medical Sciences, Riyadh 11426, Saudi Arabia, 4King Abdulaziz Medical City, Department of Pathology, Riyadh 11426, Saudi Arabia, 5University of Melbourne, Department of Obstetrics and Gynaecology and Department of Perinatal Medicine Pregnancy Research Centre, Royal Women’s Hospital, Parkville, Australia

P1.53 Identification of a trophoblast side-population with low Hoechst staining in human term placentae: Are these Trophoblast Stem Cells?

Teena Gamage1, Larry Chamley1, Jo James1  
1The University of Auckland, Auckland, New Zealand

P1.54 Mesenchymal Stem Cell Co-transplantation Enhanced the Vessel Formation Capacity of Placental Derived Endothelial Colony Forming Cells In the Wild Type and Immunodeficient Mice

Abbas Shafiee1, Jatin Patel1, Nicholas Fisk1, Kiarash Khosrotehrani1  
1The University of Queensland centre for Clinical Research, University of Queensland, Brisbane,QLD, Australia, Australia

P1.55 Mesenchymal stem cells derived from human term, fetal chorionic villi and the maternal decidua produce ectopic bone in vivo

Gina Kusuma1, Danijela Menicanin2,3, Stan Gronthos2, Mohamed Abumaree4, Shaun Brennecke1, Bill Kalionis5  
1University of Melbourne Department of Obstetrics and Gynaecology and Department of Perinatal Medicine Pregnancy Research Centre, Royal Women’s Hospital, Victoria, Australia, 2Mesenchymal Stem Cell Laboratory, Faculty of Health Sciences, School of Medical Sciences, University of Adelaide, Adelaide, Australia, 3Colgate Australian Clinical Dental Research Centre, School of Dentistry, University of Adelaide, Adelaide, Australia, 4King Abdullah International Medical Research Center, King Saud Bin Abdulaziz University for Health Sciences, Riyadh, Saudi Arabia

P1.56 Placental Contribution to Fetal Neurological Development: Role of Placenta-derived Mesenchymal Stromal Cells (PDMSCs) in Physiological and Preeclamptic Pregnancies

Rossella Barrile1, Cristian Zenerino1, Domenica Giuffrida1, Anna Maria Nuzzo1, Edoardo Terzolo1, Tullia Todros1, Alessandro Rolfo1  
1Dept. of Surgical Sciences, University of Turin, Turin, Italy
P1.57 Syncytiotrophoblast layer 2 expresses MDR1 and BCRP efflux transporters in rodent placenta
Masatoshi Tomi¹, Tomoya Akashi¹, Yoshiya Takaki¹, Tomohiro Nishimura¹, Emi Nakashima¹
¹Keio University of Pharmacy, Tokyo, Japan

P1.58 Angiopoietin Like 4 Inhibits Placental LPL Activity
Theresa Powell¹, Vanessa Ramirez², Evelyn Miller¹, Soumini Vasan¹, Susanne Lager⁴
¹University of Colorado, Aurora, CO, USA, ²Incell Corporation, San Antonio, TX, USA, ³University of Texas Health Science Center San Antonio, San Antonio, TX, USA, ⁴University of Cambridge, Cambridge, UK (4)

P1.59 Angles in normal placental chorionic surface vessel networks follow hydrodynamic bifurcation rules
Theresa Girardi¹,², Carolyn Salafia¹,², Ruchit Shah¹, George Merz³, Dawn Misra³, Richard Miller⁴,⁵, Philip Katzman⁴,⁵, John Moyer⁶
¹Placental Analytics, Larchmont, NY, USA, ²Placental Modulation, Institute for Basic Research, Staten Island, NY, USA, ³Department of Family Medicine and Public Health Sciences, Wayne State University School of Medicine, Detroit, MI, USA, ⁴Obstetrics and Gynecology and Pathology, University of Rochester School of Medicine, Rochester, NY, USA, ⁵National Children's Study, Eunice Kennedy Shriver National Institute of Child Health & Human Development, Bethesda, MD, USA

P1.60 Elementary modelling of coiling effects on blood flow in the umbilical cord
Carolyn Salafia¹, Diana Thomas³, A. David Trubatch¹, Philip Yecko¹
¹The Cooper Union, New York, NY, USA, ²Montclair State University, Montclair, NJ, USA, ³Placental Analytics LLC, Larchmont, NY, USA

P1.61 A 2D porous media model for the placenta vasculature
Zhenxing Wu¹, Parisa Mirbod¹
¹Clarkson University, Potsdam, New York, USA

P1.62 Investigating oxygen uptake efficiency with a computational model of the human placenta
Mabelle Lin¹, Joanna James³, Merryn Tawhai¹, Alys Clark¹
¹Auckland Bioengineering Institute, University of Auckland, Auckland, New Zealand, ²Obstetrics & Gynaecology, Faculty of Medical and Health Sciences, University of Auckland, Auckland, New Zealand

P1.63 The Influence of Maternal Dietary Creatine Intake on Placental Nutrient Transfer in the Spiny Mouse
Stacey Ellery¹,², Zoe Ireland², Rod Snow², David Walker¹,², Hayley Dickinson¹,²
¹The Ritchie Centre, MIMR-PHI Institute of Medical Research, Clayton, Victoria, Australia, ²Department of Obstetrics and Gynaecology, Monash University, Clayton, Victoria, Australia, ³Clinical Research Centre, University of Queensland, Brisbane, Queensland, Australia, ⁴Centre for Physical Activity and Nutrition Research, School of Exercise and Nutrition Sciences, Deakin University, Burwood, Victoria, Australia

P1.64 Identification of important placental nutrient transporters in intrauterine growth restriction and preeclampsia using various strategies
Xiao Huang¹,², Pascale Anderle³,⁴, Lu Hostettler², Michael Lüthi¹,², Marc Baumann¹,³, Daniel Surbek¹,³, Edgar Ontsouka¹,², Christiane Albrecht¹,²
¹Swiss National Center of Competence in Research, NCCR TransCure, University of Bern, Bern, Switzerland, ²Institute of Biochemistry and Molecular Medicine, Faculty of Medicine, University of Bern, Bern, Switzerland, ³Swiss Institute of Bioinformatics, Lausanne, Switzerland, ⁴Department of Obstetrics and Gynecology, University Hospital, University of Bern, Bern, Switzerland

P1.65 Impaired placental thiamine transport in human pregnancy diseases
Xiao Huang¹,², Lu Hostettler², Michael Lüthi¹,², Marc Baumann¹,³, Daniel Surbek¹,³, Edgar Ontsouka¹,², Christiane Albrecht¹,²
¹Swiss National Center of Competence in Research, NCCR TransCure, University of Bern, Bern, Switzerland, ²Institute of Biochemistry and Molecular Medicine, Faculty of Medicine, University of Bern, Bern, Switzerland, ³Department of Obstetrics and Gynecology, University Hospital, University of Bern, Bern, Switzerland
P1.66  Real-Time Assessment Of Fatty Acid Kinetics In The Human Placenta

Kevin Kolahi1, Samantha Louey2, Oleg Varlamov1,2, Kent Thornburg1
1Oregon Health and Science University, Portland, OR, USA, 2Oregon National Primate Research Center, Beaverton, OR, USA

P1.67  A microengineered model of the human placental barrier

Cassidy Blundell1, Ariana Schanzer1, Emily J Su1, Samuel Parry2, Dan Dongeon Huh1
1Department of Bioengineering, University of Pennsylvania, Philadelphia, PA, USA, 2Perelman School of Medicine, University of Pennsylvania, Philadelphia, PA, USA, 3University of Colorado Denver School of Medicine, Denver, CO, USA

Trophoblast Biology

P1.68  Combinatorial Functions of GATA2 and GATA3 are Essential For Early Trophoblast Development and to Balance the Stem vs. Differentiation and Angiogenic Equilibrium in Trophoblast Lineage

Soumen Paul1, Pratik Home1
1University of Kansas Medical Center, Kansas City, KS, USA

P1.69  Angiogenesis-associated pathways in trophoblast pseudovascularization

Amanda Highet1, Sultana Khoda1, Sam Buckberry1, Tina Bianco-Miotto1, Claire Roberts1
1University of Adelaide and Robinson Research Institute, Adelaide, South Australia, Australia

P1.70  Regulatory action of an intracellular Ca2+ in cAMP-induced functional and morphological differentiation in trophoblasts

Mikihiro Yoshie1, Rinna Tamakoshi1, Kazuhiro Tamura1, Gen Ishikawa2, Akihito Nakai2, Toshiyuki Takeshita1, Naoko Kuwabara1, Eiichi Tachikawa1
1Dept. of Endocrine and Neural Pharmacology, Tokyo University of Pharmacy and Life Sciences, Tokyo, Japan, 2Division of Reproductive Medicine, Perinatology and Gynecologic Oncology, Nippon Medical School, Tokyo, Japan

P1.71  Over-expression of IGF-1 via nanoparticle-mediated Trophoblast specific gene delivery reduces AKT1 expression but induces downstream signaling via GSK-beta

Kathryn Owens1, Weston Troja1, Helen Jones1
1Cincinnati Children's Hospital Medical Center, Cincinnati, Ohio, USA

P1.72  Effects of (-)-9-tetrahydrocannabinol (THC) on trophoblast cells proliferation and migration

Xinwen Chang1, Julei Yao1, Qizhi He1, Kai Wang1, Tao Duan1
1Shanghai First Maternity and Infant Hospital, Tongji University School of Medicine, Shanghai, China

P1.73  Placental Exocyst 8 and RalB in Health and Disease

Isabel Gonzalez1, William Ackerman1, Dale Vandre1, John Robinson1
1Ohio State University, Columbus, OH, USA

P1.74  miR-141 is up-regulated in preeclamptic placentae and regulates trophoblast viability and invasion

Stephanie Ospina-Prieto1, Wittaya Chaiwangyen1, Ekkehard Schleussner1, Udo R Markert1, Diana M Morales-Prieto1
1Placenta-Lab, University Hospital Jena, Jena, German

P1.75  CRIPTO1/3 modulates invasion of trophoblast HTR8/SV-neo cell lineage

Carla Bandeira1, Mara Hoshida1, Martin Knofler2, Graciela Panzetta-Dutari4, Susana Genti-Raimondi4, Magali Ridano1, Rossana Francisco1, Estela Bevilacqua1
1Dept of Cell and Developmental Biology, Institute of Biomedical Sciences, University of São Paulo, São Paulo, SP, Brazil, 2Dept of Obstetrics and Fetal-Maternal Medicine, Reproductive Biology Unit, Medical University of Vienna, Vienna, Austria, 3Dept of Obstetrics, Medical School, University of São Paulo, São Paulo-SP, Brazil, 4Dept of Clinical Biochemistry, Faculty of Chemical Sciences, National University of Cordoba, Cordoba, Argentina
P1.76 Periconceptional alcohol exposure causes mid-gestational placental growth restriction and alters trophoblast invasion into the decidua
Jacinta Kalisch-Smith, Marie Pantaleon, David Simmons, Karen Moritz
1The University of Queensland, Brisbane, QLD, Australia

P1.77 Endothelial cytokines influence trophoblast invasion during first trimester of pregnancy
Gregor Weiss, Berthold Huppertz, Monika Siwetz, Ingrid Lang-Olip, Gerit Moser
1Medical University of Graz, Graz, Austria
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**Xenobiotics**

**P2.01** A feto-placental co-culture model shows the complex disruptive effect of the antidepressant fluoxetine and its metabolite norfluoxetine on estrogen biosynthesis and serotonin transport  
Laetitia Laurent1, André-Anne Hudon Thibeault1,2, J Thomas Sanderson1,2, Cathy Vaillancourt1,2  
1INRS-Institut Armand Frappier, Laval, Quebec, Canada, 2BioMed Research Center, Laval, Quebec, Canada

**Oxidative Stress**

**P2.02** Trophoblast mitochondrial biogenesis and functionality is increased with selenium supplementation  
Alisha Khera1, Jessica Vanderlelie1, Olivia Holland1, Anthony Perkins1  
1Griffith University, Gold Coast, Queensland, Australia

**P2.03** Influence of mitochondrial dynamics by selenium in trophoblasts  
Olivia Holland1, Alisha Khera1, Jessica Vanderlelie1, Anthony Perkins1  
1Griffith University, Gold Coast, QLD, Australia

**P2.04** Placental health, the role of anti-oxidant systems and selenium  
Olivia Holland1, Alisha Khera1, Jessica Vanderlelie1, Anthony Perkins1  
1Griffith University, Gold Coast, QLD, Australia

**P2.05** Indoleamine-2,3-Deoxygenase (IDO1) Oxygen-mediated Regulation in Normal, Preeclamptic and Chronic Kidney Disease (CKD) Placentae  
Edoardo Terzolo1, Giorgina Barbara Piccoli2, Anna Maria Nuzzo1, Domenica Giuffrida1, Tullia Todros1, Alessandro Rolfo1  
1Dept. of Surgical Sciences, University of Turin, Turin, Italy, 2Dept. of Clinical and Biological Sciences, University of Turin, Turin, Italy

**Epigenetics**

**P2.06** DNA Methylation Biomarkers for Predicting Pregnancy Complications  
Tina Bianco-Miotto1,2, Carlos Rodriguez Lopez1, Shalem Leemaqz1,2, Sam Buckberry1,2, Dylan McCullough2,3, Zimin Zhuang2,3, Gus Dekker2,3, Mike Wilkinson1, Claire Roberts2,3  
1University of Adelaide, School of Agriculture, Food and Wine, Australia, 2Robinson Research Institute, Adelaide, South Australia, Australia, 3University of Adelaide, School of Paediatrics and Reproductive Health, Australia

**P2.07** Transient and placenta-specific imprinting in human development  
Courtney Hanna1, Maria Penaherrera2,3, Heba Saadeh1, Deborah McFadden2,3, Gavin Kelsey1, Wendy Robinson2,3  
1Babraham Institute, Cambridge, Cambridgeshire, UK, 2University of British Columbia, Vancouver, British Columbia, Canada, 3Child and Family Research Institute, Vancouver, British Columbia, Canada

**Metabolism**

**P2.08** Hypoxia in late mouse pregnancy impairs placental mitochondrial function & nutrient transport  
Josephine Higgins1, Owen Vaughan1, Andrew Murray1, Abigail Fowden1, Amanda Sferruzzi-Perri1  
1Centre for Trophoblast Research, University of Cambridge, Cambridge, UK

**P2.09** Placental biosynthesis and transport of creatine - a fundamental cellular energy metabolite  
Hayley Dickinson1,2, Stacey Ellery1,3, Paul Della-Gatta2, Lobna Ghattas2, Syed Baharom1,3, Miranda Davies-Tuck1,2, Euan Wallace1,3, Joanne Mockler1,3, Rod Snow2, David Walker1,3  
1The Ritchie Centre, Melbourne, Australia, 2Deakin University, Melbourne, Australia, 3Monash University, Melbourne, Australia
P2.10  Trophoblast-derived exosomes under diabetic conditions modulate glucose uptake in skeletal muscle cells involving mTOR pathway
Katherin Scholz-Romero¹, Felipe Zuñiga², Liliana Lamperti², Grace Truong¹, Miharu Kobayashi¹, Gregory Duncombe¹, Murray Mitchell¹, Gregory Rice¹, Carlos Salomon¹
¹Exosome Biology Laboratory, Centre for Clinical Diagnostics, University of Queensland Centre for Clinical Research, Royal Brisbane and Women’s Hospital, The University of Queensland, Brisbane, QLD, Australia, ²Department of Clinical Biochemistry and Immunology, Faculty of Pharmacy, University of Concepcion, Concepcion, Bio-Bio, Chile

Diabetes

P2.11  Feeding diets enriched in PUFAs to pregestational diabetic rats ameliorate metabolic parameters and placental lipoperoxidation in their pregnant offspring.
Evangelina Capobianco, Daiana Fornes, Ivana Linenberg, Veronica White Jawerbaum A, Laboratory of Reproduction and Metabolism, CEFYBO-CONICET- UBA, Buenos Aires, Argentina

P2.12  The microbial community of eutherian origin: effects of maternal microbiota on the placental microbiome
Leanne Ragonesi¹, Flavia Huygens¹, Ross Turner², Philip Hugenholtz³, Rohan Lourie³, Ken Beagley¹, Elise Pelzer¹
¹Queensland University of Technology, Brisbane, Queensland, Australia, ²Wesley Hospital, Brisbane, Queensland, Australia, ³Mater Pathology, Brisbane, Queensland, Australia, ⁴Australian Centre for Ecogenomics, UQ, Brisbane, Queensland, Australia

P2.13  Late pregnancy amniotic fluid Erythropoietin (EPO) levels in women with and without Type 1 diabetes
Giovanna Bernatavicius¹,², James Horn², Clare Thomson¹, Simone McLaughlin¹, David McCance³, Robert Lindsay¹, Fiona Dennison¹, Tom Farrell⁶, Fiona Mackenzie¹, Michael Maresh¹, Jenny Myers¹,²
¹Central Manchester NHS Trust, Manchester, UK, ²Maternal & Fetal Health Research Centre, University of Manchester, Manchester, UK, ³Royal Victoria Hospital, Belfast, UK, ⁴British Heart Foundation Glasgow Cardiovascular Research Centre, University of Glasgow, Glasgow, UK, ⁵MRC Centre for Reproductive Health, University of Edinburgh, Edinburgh, UK, ⁶Jessop Wing, Sheffield Teaching Hospitals Foundation Trust, Sheffield, UK, ⁷Princess Royal Maternity Unit, Glasgow Royal Infirmary, Glasgow, UK

P2.14  First trimester multiparametric model for gestational diabetes prediction
Paula Correa¹, Pia Venegas¹, Greg Rice¹, Carlos Salomon¹, Lazaro Montenegro², Jaime Olguín³, Jaime Roa³, Jorge Cortés³, Jyh Kae Nien¹², Sebastian Illanes¹²
¹Universidad de los Andes, Santiago, Chile, ²Clinica Dávila, Santiago, Chile, ³University of Queensland, Queensland, Australia

P2.15  Influence of type 1 diabetes on the extracellular matrix of the vascular region of mesometrial decidua in mice
Rodolfo Favaro¹, Layra Albuquerque², Zuleica Fortes¹, Telma Zorn¹
¹University of São Paulo, São Paulo, São Paulo, Brazil, ²Butantan Institute, São Paulo, São Paulo, Brazil

P2.16  Opposite trends in seasonality of gestational diabetes mellitus and pregnancy induced hypertensive disorders - A South Australian population study
Petra Verburg¹,², Graeme Tucker¹,², Wendy Scheit¹⁴, Jan Jaap Erwich¹, Claire Roberts¹, Gus Dekker¹,²
¹University of Adelaide, Robinson Research Institute, School of Paediatrics and Reproductive Health, Adelaide, South Australia, Australia, ²University of Groningen, University Medical Center Groningen, Department of Obstetrics and Gynaecology, Groningen, Groningen, The Netherlands, ³SA Health, Epidemiology Branch, Adelaide, South Australia, Australia, ⁴University of Adelaide, The School of Medicine, Adelaide, South Australia, Australia, ⁵Lyell McEwin Hospital, Department of Obstetrics and Gynaecology, Elizabeth Vale, South Australia, Australia
P2.17 Exosomes isolated from obese pregnancies promote TNF-α release from endothelial cells
Omar Elfeky1, Katherin Scholz-Romero1, Miharu Kobayashi1, Gregory Duncombe1, Sherri Longo2, Murray Mitchell1, Gregory Rice1, Carlos Salomon1
1Exosome Biology Laboratory, Centre for Clinical Diagnostics, University of Queensland Centre for Clinical Research, Royal Brisbane and Women’s Hospital, The University of Queensland, Brisbane, QLD, Australia
2Department of Obstetrics and Gynecology, Ochsner Baptist Hospital, New Orleans, Louisiana, USA

Fetal Growth Restriction

P2.18 Severe discordancy in dichorionic diamniotic twin potentially shows preterm labor and poor perinatal outcome
Michihisa Shiro1, Naoyuki Iwahashi1, Tomoko Noguchi1, Tamaki Yahata1, Madoka Yamamoto1, Yuko Tanizaki1, Aya Kobayashi1, Nami Ota1, Shigetaka Yagi1, Sawako Minami1, Kazuhiko Ino1
1Wakayama Medical University, Wakayama, Japan

P2.19 Growth restriction alters expression of placental leptin signalling targets in a sex-specific manner
Deanne Hryciw1, Jessica Briffa1, Lisa Jedwab1, Tania Romano2, Mary Wlodek1
1Department of Physiology, University of Melbourne, Parkville, Australia
2Department of Human Biosciences, LaTrobe University, Bundoora, Australia

P2.20 Maternal Dietary Nitrate Supplementation Alters Fetoplacental Vascular Function in the Endothelial Nitric Oxide Synthase Knockout (eNOS) Mouse
Elizabeth Cottrell1, Mark Wareing1, Elizabeth Cowley1, Sarah Finn-Sell1, Susan Greenwood1, Phillip Baker2, Colin Sibley1
1Maternal and Fetal Health Research Centre; University of Manchester, Manchester, UK
2Liggins Institute; University of Auckland, Auckland, New Zealand

P2.21 Early Identification of fetal growth restriction using 3D ultrasound measured fractional thigh volume
Louise Simcox1, Jenny Myers1, Ed Johnstone1
1University of Manchester, Manchester, UK

P2.22 Decreased placental vitamin D receptor contributes to aberrant cell-cycle gene regulation in human idiopathic fetal growth restriction
Thy Nguyen1,2, Hannah Yong1,2, Anthony Borg2, Shaun Brennecke1,2, Padma Murthi1,2
1The University of Melbourne, Parkville, VIC, Australia
2Royal Women's Hospital, Parkville, VIC, Australia

P2.23 Distinct Intrauterine Growth Trajectories in Infants with Too-Small Placentas
Daphne Landau1, Michelle Bejar1, Carolyn Salafia1, Nayaab Khawar1, Pramod Narula1, Beata Dzugulski1, Jennifer Straughen1, Dawn Misra1, Natan Haratz1
1New York Methodist Hospital, Brooklyn, NY, USA
2The Wayne State University School of Medicine, Detroit, Michigan, USA
3Henry Ford Health Systems, Detroit, Michigan, USA

P2.24 Placental phenotypes and fetal programming of adult diabetes; exploring the relationship
Prutha Mehta1, Bryony Natale1, Christina Schweitzer2, David Natale3
1University of California San Diego, La Jolla, CA, USA
2University of Calgary, Calgary, AB, Canada
3University of California, Los Angeles, CA, USA

P2.25 Of babies with fetal growth restriction (FGR) at term what proportion have placental findings that have implications for future pregnancies
Roberto Orefice1, Jane Dahlstrom1, Allison Kent1, Farah Sethna1
1The Canberra Hospital, Canberra, ACT, Australia
P2.26 Obesity related FTO gene polymorphism and the risk of adverse pregnancy outcomes
Prabha Andraweera1, Gustaaf Dekker1,2, Shalem Leemaqz1, Lesley McCowan1, Claire Roberts1
1School of Paediatrics and Reproductive Health, Robinson Research Institute, University of Adelaide, Adelaide, Australia, 2Division of Women's Health, Lyell McEwin Hospital, Elizabeth Vale, Australia,
3Discipline of Obstetrics and Gynaecology, The University of Auckland, Auckland, New Zealand

P2.27 Tryptophan dilates preconstricted chorionic arteries and endothelial indoleamine 2,3-dioxygenase-1
deficient in intrauterine growth restriction and preeclampsia
Pablo Zardoya Laguardia1, Astrid Blaschitz1, Sasa Frank1, Ingrid Lang-Olip1, Martin Gauster1, Martin
Haeusler1, Mila Cervar-Zivkovic1, Eva Karpf1, Peter Sedlmayr1
1Medical University of Graz, Graz, Styria, Austria

Fetal Membranes
P2.28 Inhibition of Collagen Expression by Cortisol in human Amnion Fibroblasts: A Novel Role of Local
Regeneration of Cortisol in the Rupture of Fetal Membranes
Gang Sun1, Chao Liu1
1Center for Reproductive Medicine, Renji Hospital, Shanghai Jiaotong University School of Medicine,
Shanghai, China

Imaging and Prenatal Diagnosis
P2.29 Serial change in cervical length and the risk of emergent cesarean delivery in placenta previa
Sae Kyung Choi1, Jae Eun Shin1, Sa Jin Kim1
1The Catholic university of Korea, Seoul, Republic of Korea

Immunology
P2.30 Exogenous myostatin and varied oxygen tensions alters the release of cytokines from first trimester
placental explants
Hassendrini Peiris1, Carlos Salomon1, Kanchan Vaswani1, Gregory Duncombe2, Gregory Rice1,
Murray Mitchell1
1The University of Queensland Centre for Clinical Research, Brisbane, Queensland, Australia, 2Royal
Brisbane and Women’s Hospital, Department of Obstetrics and Gynaecology, Brisbane, Queensland,
Australia
P2.31 Immunohistochemical analysis of CD56-positive uNK-cells in the endometrium
Maja Weber1, Bettina Toth2, Isabel Santillan3, Justine S. Fitzgerald1,4, Ekkehard Schleußner1, Udo R.
Markert1
1University Hospital Jena, Department of Obstetrics and Gynecology, Placenta-Lab, Jena, Germany,
2University Hospital Heidelberg, Gynecological Endocrinology and Fertility Disorders, Heidelberg,
Germany, 3Centro Médico Palencia, Madrid, Spain, 4Praxis Klinik am Anger, Kinderwunschzentrum
Erfurt, Erfurt, Germany
P2.32 Upregulation of endothelial chemokines in response to extravillous trophoblast-secreted factors:
potential mechanism for leukocyte infiltration of spiral arteries
Ruhul Choudhury1, Caroline Dunk2, John Aplin3, Stephen Lye2, Lynda Harris1, Rebecca Jones1
1University of Manchester, Manchester, UK, 2Lunenfeld Tanenbaum Research Institute, Toronto,
Canada
Syncytiotrophoblast exosomes guide monocyte maturation and activation of monocytes and granulocytes
Claudia Göhner1,2, Jolien Fledderus2,3, Justine S. Fitzgerald2, Ekkehard Schleußner2, Udo R. Markert2, Marijke M Faas1
1University of Groningen, University Medical Center Groningen, Department of Obstetrics and Gynecology, Groningen, The Netherlands, 2University Hospital Jena, Department of Obstetrics, Placenta-Lab, Jena, Thuringia, Germany, 3University of Groningen, University Medical Center Groningen, Department of Pathology and Medical Biology, Division of Medical Biology, Immunoenocrinology, Groningen, The Netherlands

Syncytiotrophoblast exosomes as well as microvesicles differentially activate cytotoxicity of T- and NK-cells, which is partly lost in preeclampsia
Claudia Göhner1,2, Jolien Fledderus3,1, Justine S. Fitzgerald2, Ekkehard Schleußner2, Udo R. Markert2, Marijke M Faas1, Torsten Plösch1, Sicco A. Scherjon1
1University of Groningen, University Medical Center Groningen, Department of Obstetrics and Gynecology, Groningen, The Netherlands, 2University Hospital Jena, Department of Obstetrics, Placenta-Lab, Jena, Thuringia, Germany, 3University of Groningen, University Medical Center Groningen, Department of Pathology and Medical Biology, Division of Medical Biology, Immunoenocrinology, Groningen, The Netherlands

Insulin receptor A and A1 adenosine receptors expression are required to restore human fetoplacental endothelial function in gestational diabetes mellitus
Tamara Saez1,2, Rocio Salsoso1, Luis Silva1, Carlos Sanhueza1, Fabian Pardo1, Andrea Leiva1, Luis Sobrevia1
1Cellular and Molecular Physiology Laboratory (CMPL), Division of Obstetrics and Gynecology, School of Medicine, Pontificia Universidad Catolica de Chile, Santiago, Chile, 2University Medical Center Groningen (UMCG), Faculty of Medicine, University of Groningen, Groningen, The Netherlands, 3University of Queensland Center for Clinical Research (UQCCR), Faculty of Medicine and Biomedical Sciences, University of Queensland, Brisbane, Australia, 4Faculty of Pharmacy, Universidad de Sevilla, Seville, Spain

Level of Cotinine Inversely Related to the Expression of Placenta VEGF in Pregnant Second Hand Smoker
Norizal Mohd Noor1, Muadz Baharom1, Bahiyah Abdullah2, Nurhuda Ismail2, Methil Kannan Kutty1, Noor Kaslina Mohd Kornain1, Chong Kon Ren1
1Cluster of Laboratory Medicine Science, Faculty of Medicine, Universiti Teknologi MARA, Sg. Buloh, Selangor, Malaysia, 2Cluster of Women & Child Health Science, Faculty of Medicine, Universiti Teknologi MARA, Sg. Buloh, Selangor, Malaysia, 3Cluster of Community Health, Faculty of Medicine, Universiti Teknologi MARA, Sg. Buloh, Selangor, Malaysia, 4Department of Obstetric & Gynaecology, Sungai Buloh Hospital, Sg. Buloh, Selangor, Malaysia

The management of placental abruption
Tomomi Kotani1, Hiroaki Moroi1, Tomoko Nakano1, Hiroyuki Tsuda1, Seiji Sumigama1, Eiko Yamamoto1, Fumitaka Kikkawa1
1Nagoya University Graduate School of Medicine, Nagoya, Japan

The Human Placenta at 41 Weeks of Gestation Shows Evidence of Aging with Shortened Telomeres, DNA Oxidation and Changes in IGFR2, Autophagy and mTOR
Kaushik Maiti1, Zakia Sultana1, John Atiken1, Roger Smith1
1Mother and Babies Research Centre, HMRI, University of Newcastle, NSW, Australia, 2School of biological Science, University of Newcastle, NSW, Australia
P2.39 Use of an in vitro human placental explant culture model to interrogate the effects of starvation on placental function
Zakia Sultana 1, Kaushik Maiti 1, Roger Smith 1
1Mother and Babies Research Centre, HMRI, University of Newcastle, NSW, Australia

P2.40 Restriction of placental growth from conception in the sheep results in changes in placental structure and growth factor expression that are independent of whether the fetus becomes hypoxaemic in late gestation
Song Zhang 1, Paige Barker 1, Kimberley Botting 1, Claire Roberts 2, Christine McMillan 1, Caroline McMillen 1, Janna Morrison 1
1University of South Australia, Adelaide, SA, Australia, 2University of Adelaide, Adelaide, SA, Australia

Preeclampsia

P2.41 The reduction of circulating levels of melatonin contribute to the development of preeclampsia
Yifei Gao 1, ke Zeng 1, Jiayi Wan 1, Mancy Tong 1, Jun Zhao 1, Min Zhao 1, Qi Chen 1,2
1Fudan University of China, Shanghai, China, 2The University of Auckland, Auckland, New Zealand, Nanjing Medical University, Nanjing, China

P2.42 Melatonin and Resveratrol as Potential Treatments for Preeclampsia
Seshini Gurusinghe 1,2, Rebecca Lim 1,2, Siow Teng Chan 1,2, Harmee Singh 1,2, Bryan Leaw 1,2, Rahana Rahman 1,2, Euan Wallace 1,2
1The Ritchie Centre, MIMR-PHI Institute of Medical Research, Melbourne, Victoria, Australia, 2Monash University, Melbourne, Victoria, Australia

P2.43 Dysregulation of oxygen sensing/responsive pathways in pregnancies complicated by idiopathic intrauterine growth restriction and early onset preeclampsia
Sean KM Seeho 1, Jenny H Park 1, Sharon A McCracken 1, Eileen DM Gallery 1, Jonathan M Morris 1
1Royal North Shore Hospital, Kolling Institute, University of Sydney, Sydney, NSW, Australia

P2.44 Circulating pro-renin and its receptor in HIV associated normotensive pregnant and pre-eclamptic women
Thajasvarie Naicker 1, Londiwe Silwane 1, Anushka Ajith 1, Jagidesa Moodley 1
1University of KwaZulu-Natal, Durban, KwaZulu Natal, South Africa

P2.45 Immunolocalization of CCR-5 and ICAM-2 expression in the placenta of normotensive and pre-eclamptic women
Camille Vallen 1, Vinogrin Dorsamy 1, Jagidesa Moodley 1, Thajasvarie Naicker 1
1University of Kwa-Zulu Natal, Durban, South Africa

P2.46 Melatonin regulates autophagy and inflammation in human trophoblastic cells
Lucas Sagrillo Fagundes 1, Eugénia Maria Assunção Salustiano 1, Philippe Wong Yen 1, Cathy Vaillancourt 1
1InRS-Institut Armand Frappier, Laval, QC, Canada

P2.47 Antiphospholipid antibodies affect mitochondrial DNA levels in placental extracellular vesicles: Alarming(g) for preeclampsia
Mancy Tong 1, Qi Chen 1, Chez Viall 1, Joana Desousa 2, Michelle Wise 1, Peter Stone 1, Jo James 1, Lynsey Cree 1, Larry Chamley 1
1Dept Obstetrics and Gynaecology, University of Auckland, Auckland, New Zealand, 2Maternal Fetal Medicine, Auckland City Hospital, Auckland, New Zealand

P2.48 Activating Transcription Factor 3 - a key molecule in the pathogenesis of preeclampsia
Tu‘uhevahau Kaitu‘u-Lino 1,2, Fiona Brownfoot 1,2, Natalie Hannan 1,2, Roxanne Hastie 1,2, Ping Cannon 1,2, Laura Tuohney 1,2, Stephen Tong 1,2
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1Translational Obstetrics Group, Mercy Hospital for Women, Melbourne, Australia, 2Department of Obstetrics and Gynaecology, University of Melbourne, Melbourne, Australia

P2.49 Extravillous trophoblast-derived exosomes promote endothelial cell migration in early pregnancy
Grace Truong1, Sarah Yee1, Katherin Scholz-Romero1, Miharu Kobayashi1, Hassendrini Peiris1, Gregor Duncombe1, Murray Mitchell1, Gregory Rice1, Carlos Salomon1
1Exosome Biology Laboratory, Centre for Clinical Diagnostics, University of Queensland Centre for Clinical Research, Royal Brisbane and Women’s Hospital, The University of Queensland, Brisbane, QLD, Australia

P2.50 Disruption of BMP9 Signalling Contributes to Altered Acid Ceramidase Expression and Processing in Pre-eclampsia
Giovanni Tossetta1, Leonardo Ermini2, Martin Post2, Isabella Caniggia1
1Lunenfeld-Tanenbaum Research Institute, Mount Sinai Hospital, Toronto, Canada, 2The Hospital for Sick Children, Toronto, Canada

P2.51 Detection of CD63+ micro-vesicles, placental alkaline phosphatase and angiogenic markers in gingival crevicular fluid in pre-eclamptic patients
A Chaparro1, E Zuhiga1, D Gaedechens1, V Ramirez2, C Inostroza3, JP Kusanovic4, K Silva4, GE Rice2, SE Ilanes5,6
1Department of Periodontology, Dentistry Faculty. Universidad de los Andes, Santiago, Chile, 2Department of Public Health and Epidemiology. Dentistry Faculty. Universidad de los Andes, Santiago, Chile, 3CIBRO. Oral Biology Center Research. Dentistry Faculty. Universidad de los Andes, Santiago, Chile, 4Department of Obstetrics & Gynaecology of Centro de Salud Hospital Sótero Del Rio, Santiago, Chile, 5Foetal Medicine Unit, department of Obstetrics & Gynaecology and laboratory of reproductive biology. Universidad de los Andes, Santiago, Chile, 6UQCCR, University of Queensland, Brisbane, Chile

P2.52 Serum HtrA3 for early detection of pre-eclampsia and small for gestational age
Yao Wang1, Ying Li1, Fabricio Costa2,3, Jon A Hyett4,5, Guiying Nie1
1MIMR-PHI Institute of Medical Research, Melbourne, Victoria, Australia, 2Monash Ultrasound for Women, University of Melbourne, Melbourne, Victoria, Australia, 3Department of Obstetrics and Gynaecology, University of Melbourne, Melbourne, Victoria, Australia, 4Department of High Risk Obstetrics, Royal Alfred Hospital, Sydney, NSW, Australia, 5Discipline of Obstetrics, Gynaecology and Neonatology, University of Sydney, Sydney, NSW, Australia

P2.53 A comparative morphometric evaluation of normotensive and pre-eclamptic placenta from South African pregnant women
Kaminee Maduray1, Jagidesa Moodley1, Thajasvarie Naicker1
1University of KwaZulu-Natal, Durban, South Africa

P2.54 Down-regulation of placental glucose transporter (GLUT)-1 in pre-eclampsia
Camilla Marinii1,2, Benjamin Lüscher1,2, Marianne Jörger-Messerli1,2, Xiao Huang3,4, Jürg Gertsch5, Matthias A. Hediger1, Christiane Albrecht1, Daniel V. Surbek1,2, Marc U. Baumann1,2
1Department of Obstetrics and Gynaecology, University Hospital of Bern, Bern, Switzerland, 2Laboratory for Prenatal Medicine, Department of Clinical Research, University of Bern, Bern, Switzerland, 3Institute for Biochemistry and Molecular Medicine, University of Bern, Bern, Switzerland, 4Graduate School for Cellular and Biomedical Sciences (GCB), University of Bern, Bern, Switzerland

P2.55 Placental pathologic features of fetal growth restriction and pre-eclampsia in preterm: are placental lesions different?
Kylie Ha-Jin Chang1, Ji-Hee Sung1, Suk-Joo Choi1, Soo-Young Oh1, Jung-Sun Kim1, Cheong-Rae Roh1, Jong-Hwa Kim1
1Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Republic of Korea
P2.56 Expression and Function of Placental Uric Acid Transporter GLUT9
Benjamin Lüscher1,2, Marc U. Baumann1, Camilla Marini1,2, Matthias A. Hediger3, Christiane Albrecht1, Bruno Steiger1, Daniel Surbek1,2
1Department of Obstetrics and Gynecology, University Hospital of Bern, Bern, Switzerland, 2Department of Clinical Research, University of Bern, Bern, Switzerland, 3Institute for Biochemistry and Molecular Medicine, Bern, Switzerland, 4Department of Clinical Pharmacology and Toxicology, University Hospital Zürich, Zürich, Switzerland

P2.57 Wnt5a can be involved in the pathogenesis of preeclampsia through modulation of spiral artery remodelling and pro-and anti-angiogenic factors
Mari Ujita1, Eiji Kondoh1, Kaoru Kawasaki1, Yoshitsugu Chigusa1, Mai Sato1, Hiroshi Takai1, Hikaru Kiyokawa1, Haruta Mogami1, Ikuo Konishi1
1Kyoto University, Kyoto, Japan

P2.58 First trimester decidual stromal cell regulation of trophoblast cells and the effect of TGFβ1
Laura James-Allan1, Alison Wallace1, Guy Whitley1, Judith Cartwright1
1St George's University of London, London, UK

P2.59 Immunohistochemical expression of angiogenic and immunological factors in placenta with preeclampsia and/or fetal growth restriction
Naoyuki Iwahashi1, Tamaki Yahata1, Mika Mizoguchi1, Sakiko Naniyo1, Madoka Yamamoto1, Yuko Tanizaki1, Aya Kobayashi1, Michihisa Shiro1, Nami Ota1, Yasushi Mabuchi1, Shigetaka Yagi1, Sawako Minami1
1Wakayama Medical University, Wakayama, Japan

P2.60 Serum HtrA1 is differentially regulated between early-onset and late-onset preeclampsia
Sonia Soo Yee Teoh1,2, Min Zhao3, Yao Wang1,2, Qi Chen3,4, Guiying Nie1,2
1Implantation and Placental Development Laboratory, Centre for Reproductive Health, MIMR-PHI Institute of Medical Research, Clayton, Victoria, Australia, 2Monash University, Clayton, Victoria, Australia, 3Wuxi Maternity and Children's Health Hospital, Nanjing Medical University, Jiangsu, China, 4Department of Obstetrics and Gynaecology, University of Auckland, Auckland, New Zealand

P2.61 The role of peripheral natural killer cells in HIV associated pre-eclampsia in South Africa
Anushka Ajith1, Jagidesa Moodley1, Thajasvarie Naicker1
1University of KwaZulu-Natal, Durban, South Africa

P2.62 Analysis of lymphatic vessel endothelial hyaluron receptor-1 in the placental bed of HIV associated pre-eclampsia
Onankoy Atshakala Onyangunga1, Jagidesa Moodley1, Thajasvarie Naicker1
1University of KwaZulu-Natal, Durban, South Africa

P2.63 Vascular endothelial growth factor-receptor-3 in placenta of HIV associated normotensive and pre-eclamptic pregnancies
Onankoy Atshakala Onyangunga1, Jagidesa Moodley1, Thajasvarie Naicker1
1University of KwaZulu-Natal, Durban, South Africa

P2.64 Placental lymphatic vascular endothelial hyaluronic receptor-1 in HIV associated pre-eclampsia
Onankoy Atshakala Onyangunga1, Jagidesa Moodley1, Thajasvarie Naicker1
1University of KwaZulu-Natal, Durban, South Africa

P2.65 Characterizing the hematopoietic stem/progenitor cells in the placenta and umbilical cord blood from normal and preeclamptic subjects
Zahra Masoumi1, Mary Familiari2, Mattias Magnusson3, Eva Meze4, Stefan Hansson1
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P2.66 EGF decreases sFlt1 secretion and endothelial dysfunction: a potential therapeutic for preeclampsia
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P2.67 INSR rs2059806 polymorphism and the risk of preeclampsia
Prabha Andraweera1, Gustaaf Dekker1,2, Kathy Gatford1, Shalem Leemaqz1, Lesley McCowan3, 
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Zealand, 4Human Genetics Unit, Faculty of Medicine, University of Colombo, Colombo, Sri Lanka

P2.68 Fetal hemoglobin, α1-microglobulin and hemopexin are specific predictive first and second trimester 
biomarkers of preeclampsia
Ulrik Dolberg Anderson1, Magnus Gram3, Basky Thilaganathan2, Jonas Ranstam4, Bo Åkerström3, 
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P2.69 Atypical E2F contributes to pathophysiology of preeclampsia
Yoshinori Moriyama1, Tomomi Kotani1, Masako Sawada1, Rika Miki2, Fumitaka Kikkawa1
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Nagoya, Aichi, Japan

P2.70 Deciphering the role of the Nodal signaling pathway in preterm birth
Taghreed Heba1,3, Craig Park1,2, Daniel Dufort1,2

P2.71 Vitamin D-binding protein, a novel predictor of human labour: the relationship of serum VDBP and 
25(OH)D3.
Harry Georgiou1,2, Stella Liong1,2, Megan Di Quinzio1,2, 1University of Melbourne, Parkville, 
Victoria, Australia, 2Mercy Hospital for Women, Heidelberg, Victoria, Australia

P2.72 Multivitamin supplementation and pregnancy complications and outcomes: An analysis of the 
Environments for Healthy Living Birth Cohort
Jessica Vanderlelie1, Janelle McAlpine1, Anthony Perkins1, 1Griffith University, Gold Coast, Queensland, Australia
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P2.73 Supraphysiological gestational weight gain worsens gestational diabetes—reduced adenosine transport by reducing human equilibrative nucleosides transporter 1 expression without altering its distribution in human umbilical vein endothelial cells.
Fabian Pardo1, Luis Silva1, Eric Barros1, Barbara Fuenzalida1, Rocio Salsoso1, Andrea Leiva1, Luis Sobrevia1,2
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P2.74 Insulin requires A2B adenosine receptors activation to restore fetoplacental human endothelial function in late-onset preeclampsia
Rocio Salsoso1, Tamara Sáez1, Luis Silva1, Roberto Villalobos1, Marcelo Farias1, Carlos Sanhueza1, Fabián Pardo1, Andrea Leiva1, Luis Sobrevia1,2
1Pontificia Universidad Católica, Santiago, Chile, 2Universidad de Sevilla, Sevilla, Spain, 3University of Queensland, Brisbane, Queensland, Australia

P2.75 Proteomic identification of placental proteins associated with subsequent allergic disease in childhood
Nurul Hayati Mohamad Zainal1,3, Peter Hoffmann2, Astrud Tuck1, Vicki Clifton1,4
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P2.76 Placental mitochondrial methylation and exposure to airborne particulate matter in the early life environment: An ENVIRONAGE birth cohort study
Bram Janssen1, Hyang-Min Byun2, Wilfried Gyselaers3, Wouter Lefebvre4, Andrea Baccerelli5, Tim Nawrot1
1Hasselt University, Diepenbeek, Belgium, 2Harvard School of Public Health, Boston, USA, 3East-Limburg Hospital, Genk, Belgium, 4Flemish Institute for Technological Research, Mol, Belgium, 5Leuven University (KULeuven), Leuven, Belgium

P2.77 Anti-hypertensive Medications methyldopa, labetalol, hydralazine, and clonidine reversed TNF-a inhibited NOS expression in endothelial and trophoblast cellular networks in-vitro
Bei Xu1,3, Angela Makris2,3, Annemarie Hennessy1,3
1School of Medicine, University of Western Sydney, NSW, Australia, 2Renal Unit, Liverpool Hospital, Sydney, NSW, Australia, 3Vascular Immunology Research Laboratory, The Heart Research Institute, University of Sydney, NSW, Australia

P2.78 Hexosamine signaling pathway gene expression is increased in women with gestational diabetes mellitus (GDM)
Charlotte Ramin1, Helen Barrett1,2, Leonie Callaway1,2, Marloes Dekker Nitter1,2
1School of Medicine, The University of Queensland, Australia, 2UQCCR, The University of Queensland, Australia

P2.79 Changes in exosome concentration and exosomal endothelin-1 throughout late gestation and early postpartum in dairy cows
Katherin Scholz-Romero1, Yong Koh1, Susanne Meier2, Chris Burke2, John Roche2, Gregory Rice1, Carlos Salomon1, Murray Mitchell1
1Exosome Biology Laboratory, Centre for Clinical Diagnostics, University of Queensland Centre for Clinical Research, Royal Brisbane and Women’s Hospital, Brisbane, QLD, Australia, 2DairyNZ Limited, Hamilton, New Zealand
P2.80  Selective increase of exosome release from bovine endometrial stromal cells under hypoxic conditions
Yong Koh¹, Katherin Scholz-Romero¹, Gregory Rice¹, Carlos Salomon¹, Murray Mitchell¹
¹ Exosome Biology Laboratory, Centre for Clinical Diagnostics, University of Queensland Centre for
Clinical Research, Royal Brisbane and Women’s Hospital, Brisbane, QLD, Australia

P2.81  Differential expression of microRNAs that regulate TLR pathways in the placenta and cord blood.
Natalie Aboustate¹, Vicki Clifton², Michael Stark¹,³, Nicolette Hodyl¹,³
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Institute, Mothers and Babies Theme, University of Queensland, South Brisbane, QLD, Australia,
³ Neonatal Medicine, Women's and Children's Hospital, North Adelaide, SA, Australia

P2.82  Confocal imaging of clarified placenta terminal villi for evaluation of 3D intervillous branching
structure
George Merz¹, Wojciech Kaczmarshi¹, Valerie Schwenk¹, Alec Andoyan¹,², Philip Necaise¹,², Daphne
Landau¹, Carolyn Salafia¹,³, Ruchit Shah¹,³
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USA, ³ New York Methodist Hospital, Brooklyn NY, USA

P2.83  Transfer study of Trastuzumab® across the human placenta barrier in an ex-vivo human placenta
perfusion model
Michael Gruber¹, Bettina Amtmann¹, Birgit Hirschmugl¹, Uwe Lang¹, Gernot Desoye¹, Christian
Wadsack¹,
¹ Medical University of Graz, Graz, Austria
KEYNOTE & PLENARY ABSTRACTS

OPENING KEYNOTE LECTURE

The Placenta is the Culprit in Programming Chronic Disease

Kent L. Thornburg, Samantha Louey, Melinda Pierce and Kevin Kolahi

Center for Developmental Health, Knight Cardiovascular Institute, School of Medicine, Oregon Health and Science University, Portland, Oregon, 97237

For the first time in US history, a sustained decrease in life expectancy is predicted. The abrupt change in the century long upward trajectory in life expectancy is related to increases in the prevalence of obesity, diabetes and uncontrolled hypertension, all of which have been increasing since the ‘90s. These sudden increases can only be explained by environmental factors, the most powerful of which is maternal diet. Children in the US are now the 3rd generation of people who have consumed mostly processed foods to satisfy their energy requirements. Processed foods are generally high in calories and low in nutrients. It is now well known that babies born at the extremes of birth weights are especially prone to developing chronic disease later in life. Because the placenta is the gatekeeper for all nutrients entering the fetus and because the vascular bed of the placenta sets the loading conditions of the embryo/fetal heart, it plays a central role in determining risk for later disease. The size and shape of the placenta interact with maternal phenotypic features to determine risk of coronary heart disease, heart failure and sudden death. Lung and colorectal cancers are predicted by placental size and shape; ovarian. The number of placental cotyledons predicts blood pressure in children and umbilical cord length is associated with risk for rheumatic heart disease. Placental size, shape and efficiency in a population vary over time for unknown reasons. One can fairly speculate that population level dietary changes over time are the drivers of placentation; animal data are sparse but support the idea. Sex-specific features of placental function have been shown in humans and animals but the role of diet in altering functional changes is unknown. Understanding the interactions between maternal diet, stress, phenotype and placental form and function should be a high priority for scientists and funding agencies.

Reflections of Preterm Birth

John Challis, Grace Yang, Stephen Lye, Alan Bocking, Felice Petraglia

The University of Western Australia, University of Toronto, University of Siena

• Preterm birth (PTB) has a high prevalence and cost, with substantial regional, ethnic and gender differences as well as altered underlying etiology at different gestational ages
• Understanding these differences may help understanding core mechanisms of PTB
• Inflammatory activation drives much of early PTB
• Malnutrition, hypoxemia are major stressors that upregulate fetal HPA function and result in PTB, early or later in gestation
• Placental 11b HSD-2 is a crucial mediator of placental cortisol transfer and action, PTB and fetal programming, and is reduced with infection, hypoxemia and undernutrition
• In women, placental CRH affects placental and membrane endocrine and MMP generation and may contribute to and serve as a predictive biomarker for some PTB
• There is extensive Interaction between the stress and inflammatory axes in causing PTB
• The heterogeneous nature of the PTB syndrome requires precision (personalised) approaches to diagnosis and treatment
**NIH LECTURE**

**Role of mitochondria and oxidative stress in aging and age-related skeletal muscle atrophy**

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Aging is a multifactorial process that is associated with numerous physiologic declines involving multiple pathways. Oxidative stress and its potential for damaging cellular components has been implicated as a major causal effector in the aging process. Mitochondrial are a primary source of reactive oxygen species (ROS) and thus mitochondria and mitochondrial dysfunction have been proposed to be key players in the role of oxidative stress in aging and age-related disease. My laboratory is studying the role of mitochondrial dysfunction and oxidative stress in the initiation and progression of age related loss of muscle mass and function (sarcopenia). To study the role of chronic oxidative stress and mitochondria dysfunction in vivo, we used a mouse model that lacks the antioxidant enzyme CuZnSOD (Sod1). Sod1−/− mice are characterized by high levels of oxidative stress/damage and an acceleration of sarcopenia. Muscle atrophy in the Sod1−/− mice is accompanied by a progressive decline in mitochondrial bio-energetic function and an elevation of mitochondrial generation of reactive oxygen species. Aged Sod1−/− mice show a striking increase in muscle mitochondrial content near the neuromuscular junctions (NMJs) that is associated with disruption of postsynaptic endplates and a significant loss of contractile force in muscle from Sod1−/− mice. Dietary restriction is a powerful intervention that delays aging and extends lifespan in many species. In Sod1−/− mice, dietary restriction is associated with lower mitochondrial ROS generation, decreased oxidative damage, a reduction in age-related muscle atrophy and a significant increase in lifespan in the Sod1−/− mice compared to Sod1−/− mice fed ad libitum. The age-associated disruption in NMJs is also partially protected in Sod1−/− CR mice. These data suggest that mitochondrial function and dysregulation of oxidative stress can alter skeletal muscle mass and function though disruption of the neuromuscular junction. Our current hypothesis is that pre-synaptic deficits initiate motor neuron dysfunction that triggers the induction of ROS by muscle mitochondria that in turn feeds back upon the synapse and motor neurons leading to NMJ disruption and denervation resulting in sarcopenia. To test this we are studying whether alterations in the redox status of both neuronal and muscle tissue are required for loss of muscle mass/strength by comparing the effect of oxidative stress and mitochondrial dysfunction targeted specifically to muscle or neuronal tissue. These experiments will increase our understanding of the role of mitochondrial oxidative stress in the initiation and progression of sarcopenia and point to new pathways for intervention to delay or reduce the impact of sarcopenia in the elderly.

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**IFPA AWARD LECTURE**

**Phylogenomic origins and evolution of the mammalian placenta**

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The placenta has had the most dynamic evolutionary history of all mammalian organs. It has undergone massive shifts in anatomy, physiology, and the way in which uterine and fetal tissue interact with one another during pregnancy. The human placenta is arguably the best studied amongst mammals, yet much about its function during pregnancy is not understood. The purpose of this presentation is to outline the evolutionary history of the placenta throughout human evolutionary history, and to point out major gaps in the current state of knowledge. I also propose novel theoretical, experimental, and computational approaches that are likely to provide insight into the normal process of placentation and the role the placenta plays in the great obstetrical syndromes.
Impact of placental growth factor and preeclampsia on brain development, cognition, and behaviour

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Objective: In many preeclamptic (PE) pregnancies, maternal plasma is low in the placentally-produced angiokine “placental growth factor” (PGF). Offspring of PE (PE-F1) compared to uncomplicated pregnancies have higher risks for hypertension, cognitive impairment, and stroke. Mechanisms explaining this are poorly described. Pgf−/− mice have aberrant decidual and placental vascular branching and most experience stroke after unilateral common carotid artery occlusion. We hypothesized that PGF deficiency, which often manifests in PE, diminishes brain vascular development, leading to impaired cognition and elevated postpartum stroke risk.

Methods: Pgf−/− and Pgf+/+ adult mouse brain vasculature and structural anatomy were examined by micro-computed tomography and magnetic resonance imaging (MRI). Cognition and behaviour were assessed in these mice by standard paradigms that tested depression, spatial learning, short and long term memory, activity and anxiety. Ten child pairs aged 8-10 born to PE or uncomplicated pregnancies were analysed for cognitive functions (psychometrics, eye tracking) and brain structure through MRI.

Results: Pgf−/− brain vasculature was deficient from midgestation and abnormally patterned compared to Pgf+/+. Cognitive and behavioural tests revealed numerous, sexually dimorphic impairments in Pgf−/− mice. In children, specific functional tests differed between PE-F1s and matched controls. Preliminary child vascular and anatomic analyses suggested differences but did not reach statistical significance in this pilot study.

Conclusion: This work has uncovered a previously unknown link between PGF expression, brain vascular development, and cognitive functions in mice. Our data in children suggest that experience of gestation in a PE pregnancy may also depress brain vascular development with the involvement of PGF yet to be defined. These initial data suggest expansion of our approaches to children born to pregnancies complicated by PE, IUGR and diabetes will have great merit in defining critical transgenerational impacts of common gestational pathologies.

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IFPA SENIOR AWARD LECTURE

Fetal membranes: a focus for the future or a legacy from the past?

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The fetal membranes of amniotes are allantois, amnion, chorion and yolk sac. The manner of their development and contribution to placentation varies greatly across mammals. This is true also of the cavities they enclose, which are the allantoic sac, amniotic sac and exocoelom. In human development, there is an allantoic stalk, but no allantoic sac; the yolk sac is a short-lived structure and the exocoelom is obliterated in the second trimester by expansion of the amnion. The mouse is commonly used as a model of human placentation. Here, too, there is no allantoic sac and the exocoelom does not persist to term. In contrast, the inverted vascular yolk sac supports the embryo until the chorioallantoic placenta is formed and it acts as an accessory placenta through term. It has a string of important functions yet frequently is discarded or ignored either through ignorance or because it has no obvious equivalent in human pregnancy. In ruminants, which are important to veterinary research and as models in fetal physiology, there is yet another pattern that includes transient yolk sac placentation and a persistent allantoic sac. Embryologists of the late 19C, like Hubrecht and Selenka, uncovered the role of fetal membranes in early development and placentation. They feature prominently in the mid-20C accounts of Amoroso and Mossman. This legacy seems largely to have slipped from the collective consciousness of 21C placentologists. Yet the mouse yolk sac may harbour clues to overall placental function and human yolk sac play a critical role in embryonic development. Medical as well as veterinary research might be well served by a closer focus on fetal membranes.
KEYNOTE LECTURE

Aging and the placenta

Roger Smith

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Aging may be defined as a deterioration in function that occurs over time. Different species age at dramatically different rates. Aging is strongly related to reproduction as a decline in reproductive fitness potently impacts on the ability of an individual to transmit genetic material. Individual tissues also age at different rates leading to death from a variety of age related conditions. Energy is required to maintain the highly ordered structure of living tissues against the force of entropy. Generation of energy within the cell is linked to oxidative damage to DNA, RNA, lipids and proteins. Protection against oxidative damage also incurs energetic costs for the cell and the organism. The balance between oxidative damage and antioxidant processes is likely optimised in each organism to maximise reproductive potential.

Mammals have an unusual method of reproduction that includes provisioning of the developing conceptus via a placenta during pregnancy. When delivery of the fetus occurs the placenta is discarded. Therefore for optimal reproductive potential the balance of investment in placental oxidative defences is likely linked to the length of gestation in that species. For humans gestational length is between 37 completed weeks and 41 weeks for more than 90% of pregnancies. In this setting investment to protect the placenta beyond 41 weeks would be energetically wasteful of a mother’s limited resources and therefore have a negative effect on likelihood of survival of the offspring that is dependent on maternal care following delivery. These data suggest that the rapid increase in still birth rates per 1000 continuing pregnancies that occurs following 40 weeks of gestation may be linked to aging of the placenta. Our laboratory has been testing this hypothesis. We find substantial evidence of increased biochemical and histological evidence of aging in placentas from post-dated deliveries.

Uterine – Placental Transitions for Birth and Fetal Development

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Parturition is the process through which the gestational tissues progressively mature prior to the events of active labour that culminate in birth. Changes to the local and systemic immune environment with gradual erosion of the immune tolerance and anti-inflammatory state of earlier pregnancy precede the overt inflammatory events characteristic of labour and birth. The clinical problem of preterm birth invokes variations of these events in the uterus and placenta. I will review our conceptual framework of the birth cascade and how the transformation of gestational tissues from pregnancy to delivery relies upon the processes of positive feedback, synergy and amplification. Stress models demonstrating transgenerational effects utilizing epigenetic mechanisms that alter pregnancy outcomes will be described. The involvement of the placenta in transmission of transgenerational signals and as a window on fetal brain development will be noted.
P2.73

Supraphysiological gestational weight gain worsens gestational diabetes-reduced adenosine transport by reducing human equilibrative nucleosides transporter 1 expression without altering its distribution in human umbilical vein endothelial cells.

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Excessive gestational weight gain (eGWG) is a condition where mothers with pre-pregnancy normal body mass index develop supraphysiological weight gain during pregnancy ending with obesity (≥30 kg/m²) (spGWG). Even when spGWG and GD associate reduced equilibrative nucleosides transporter 1 (hENT1)-mediated adenosine transport, a phenomenon leading to altered placental vascular reactivity, nothing is known regarding the potential effect of spGWG on GDM- alterations in the human fetoplacental endothelial cells. Thus, the aim of this study was to determine whether spGWG worsens the GDM-reduced hENT1-mediated adenosine transport in human umbilical vein endothelial cells (HUVECs). Methods: ADO transport in the absence or presence of 1 or 10 µmol/L nitrobenzylthioinosine (NBPT) or 10 µmol/L chlorpromazine (CPZ). Results: ADO transport via hENT1 is reduced in the presence of insulin (~60%) and insulin (~50%) with or without spGWG. Conclusion: Reduced hENT1-mediated adenosine transport results from lower hENT1 protein availability at the plasma membrane in spGWG, but increased internalization in GDM. In cells from GDM+spGWG the reduced adenosine transport is likely due to lower hENT1 expression rather than a redistribution of this protein in HUVECs.

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P2.74

Insulin requires A2B adenosine receptors activation to restore fetoplacental human endothelial function in late-onset preeclampsia

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Preeclampsia (PE) associates with reduced placental vasodilatation, endothelial nitric oxide synthase (eNOS) activity and L-arginine transport in human fetoplacental circulation. Adenosine and insulin cause vasodilation of the human umbilical vein involving adenosine receptors (ARs) activation in uncomplicated pregnancies. However, under pathological conditions such as PE, plasma adenosine level increases in umbilical vein and also the A2B adenosine receptors (A2B AR) expression in human umbilical vein endothelial cells (HUVECs). Elevated placenta adenosine level not only contributes to the pathogenesis of PE, but also A2B AR could have a role in the placental development. Objectives. The aim of the study was to determine the role of A2B AR in the endothelial L-arginine/NO signalling pathway and vascular reactivity in response to insulin in HUVECs from late onset preeclampsia (LOPE). Methods. Vascular reactivity to insulin (0.1-1000 nmol/L, 5 min) was measured in KCl-precontracted human umbilical vein rings (wire myography) from normal and LOPE pregnancies in the absence or presence of adenosine (1 nmol/L) and/or the A2B AR antagonist (MRS-1754, 30 nmol/L). The protein level of total eNOS and L-arginine transport were determined by Western blot and high performance liquid chromatography (HPLC), respectively, in the absence or presence of insulin (1 nmol/L) and/or MRS-1754 in HUVECs from normal and LOPE pregnancies. L-Arginine transport (100 µmol/L, 3 µCi/mL L-[13C]arginine, 1 min, 37°C) was measured in the absence or presence of insulin and/or A2B AR agonist (NECA, 1 µmol/mL) and MRS-1754 in HUVECs. Results. LOPE associates with reduced insulin-mediated umbilical vein ring relaxation compared with normal pregnancies, which was improved by adenosine, an effect abolished in the presence of adenosine + MRS-1754. LOPE increased total eNOS expression and activity compared with normal pregnancies, and the A2B AR antagonist blocked these effects of LOPE. LOPE increased hCAT-1-mediated L-arginine transport in HUVECs, an effect unaltered by the A2B AR agonist alone, but blocked by the A2B AR antagonist in the presence of insulin. Conclusion. HUVECs from LOPE exhibit impairment of A2B AR-mediated L-arginine/NO signalling pathway. A2B AR activation in response to insulin is required to restore the relaxation and endothelial function in LOPE pregnancies. Support: FONDECYT 1150377, 1150344, CONICYT 3140516, 3130583, Chile. RS, TS, and LS hold CONICYT-PhD fellowships. RS and LS hold Faculty of Medicine PUC-fellowships.
Proteomic identification of placental proteins associated with subsequent allergic disease in childhood
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Allergic disease has risen to epidemic proportions during recent years. It has become evident that prenatal events play a critical role in determining disease susceptibility via environmental influences on placental function and fetal programming. We hypothesize that childhood susceptibility to allergy is increased through significant alterations in placental function that exert a programming effect on the fetal immune system.

OBJECTIVES:
We aim to identify the placental proteins associated with childhood allergy using placental tissue from two populations of women whose children have different risks of allergic disease susceptibility.

METHODS:
Placental tissue will be examined using a proteomic approach that involves quantitative label-free comparative MS and data analysis is performed using Mascot database and MaxQuant software. Placental tissue from children with no allergy were compared to children with allergic diseases (male n=8 and female n=8).

RESULTS:
Three candidate proteins were identified in placental samples associated with subsequent allergic disease in all children that include Human Biglycan (ratio of >2 relative to non-allergic samples), Human Amine oxidase [flavin-containing] A and Human Amine oxidase [flavin-containing] B (ratio <0.5 fold change relative to non-allergic samples). Moreover, there were 19 proteins significantly altered in placenta of allergic males and 21 proteins altered in placenta of allergic female relative to non-allergic children. Many of these proteins could exert a programming effect on the fetal immune system including Human Ig heavy chain V-1 region HG, Human Haptoglobin, Human Haptoglobin-related protein, Human Complement C3, Human Apolipoprotein B-100 and Human Endoplasmic reticulum aminopeptidase 1.

CONCLUSION:
The current findings suggest protein expression varies in utero in children who subsequently develop allergy and the altered expression of these proteins vary in a sex specific manner.

Placental mitochondrial methylation and exposure to airborne particulate matter in the early life environment: An ENVIRONAGE birth cohort study
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BACKGROUND: Most research to date has focused on epigenetic modifications in the nuclear genome, with little attention devoted to mitochondrial DNA (mtDNA). Placental mtDNA content has been shown to be respond to environmental exposures that induce oxidative stress including airborne particulate matter (PM). Damaged or non-functioning mitochondria are specifically degraded through mitophagy, exemplified by a lower mtDNA content, and could be primed by epigenetic modifications in the mtDNA.

OBJECTIVE: We studied placental mtDNA methylation in the context of the early life exposure.

METHODS: We investigated placental tissue from 381 mother-newborn pairs that were enrolled in the ENVIRONAGE birth cohort. We determined mtDNA methylation by bisulfite-pyrosequencing in two regions, i.e., the D-loop control region and 12S ribosomal RNA (MT-RNR1) and measured mtDNA content by qPCR. PM2.5 exposure was calculated for each participant’s home address using a dispersion model.

RESULTS: An interquartile range (IQR) increment in PM2.5 exposure over the entire pregnancy was positively associated with mtDNA methylation (MT-RNR1: +0.91%, p = 0.01 and D-loop: +0.21%, p = 0.05) and inversely associated with mtDNA content (relative change of -15.60%, p = 0.001) in placental tissue. mtDNA methylation was estimated to mediate 54%, p = 0.01 (MT-RNR1) and 27%, p = 0.06 (D-loop) of the inverse association between PM2.5 exposure and mtDNA content.

CONCLUSION: This study provides new insight into the mechanisms of altered mitochondrial function in the early life environment. Epigenetic modifications in the mitochondrial genome, especially in the MT-RNR1 region, substantially mediate the association between PM2.5 exposure during gestation and placental mtDNA content which could reflect signs of mitophagy and mitochondrial death.
P2.77

Anti-hypertensive Medications methyldopa, labetalol, hydralazine, and clonidine reversed TNF-α inhibited NOS expression in endothelial and trophoblast cellular networks in-vitro

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Objectives: Anti-hypertensive medications used to control hypertension in pregnancy have been shown to alter placental cytokines. Some medications also improve trophoblast and endothelial cellular interaction in-vitro. This study investigated whether selected anti-hypertensive medications can modulate endothelial nitric oxide synthase (eNOS) and inducible nitric oxide synthase (iNOS) expression in the interaction between trophoblast and endothelial cells.

Methods: Human uterine myometrial microvascular endothelial cells (UtMVECs) were pre-incubated with (or without) low dose TNF-α (0.5 ng/ml) or TNF-α plus sFlt-1 (100 ng/ml). Red fluorescent-labelled endothelial cells were then cultured on matrigel. After endothelial cellular networks appeared, green fluorescent-labelled HTR-8/SVneo trophoblast cells were co-cultured in the presence of clinically relevant doses of methyldopa, labetalol, hydralazine or clonidine for 24 hours. Cells were then retrieved from matrigel to extract mRNA. Medication effects on eNOS and iNOS expression were examined by quantitative PCR.

RESULTS: Methyldopa, labetalol, hydralazine and clonidine reversed the inhibitory effect of TNF-α on eNOS mRNA expression. After pre-incubating endothelial cells with TNF-α plus sFlt-1, all the medications lost their effects on eNOS mRNA expression. In the absence of TNF-α, there was no change in eNOS expression by anti-hypertensive medications. The mRNA expression of iNOS was not affected by TNF-α or any medication selected.

CONCLUSION: Our data suggest some pregnancy-specific anti-hypertensive medications may improve vascular relaxation via increasing eNOS expression. Increased sFlt-1 seen in preeclampsia may negate this effect. This indicates an adverse effect of sFlt-1 in the treatment of hypertension in pregnancy by inhibiting NOS related vascular relaxation.

P2.78

Hexosamine signaling pathway gene expression is increased in women with gestational diabetes mellitus (GDM)

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Background

The hexosamine signaling pathway (HSP) leads to the posttranslational addition of O-linked N-acetylglucosamine (O-GlcNAc) to proteins, altering their fate and function. Fructose-6-phosphate is funneled from the glycolytic pathway into the HSP by glutamine:fructose-6-phosphate aminotransferase (GFAT1). O-GlcNAc transferase (OGT1) adds O-GlcNAc and the O-GlcNacase OGA removes it. GFAT1 acts as a nutrient sensor and its activity is dependent on glucose and amino acid metabolism. In type 2 diabetes mellitus, GFAT1 mRNA levels and activity are increased in skeletal muscle. Higher O-GlcNAc levels are associated with the development of insulin resistance. This study aims to analyze placental expression of important enzymes in the HSP in GDM.

Methods

mRNA was extracted from placentas from 10 women with and 30 women without GDM matched for BMI, gestational age at delivery and birth weight. Expression of GFAT1, OGT1 and OGA was assessed by QPCR. GFAT localization was investigated with immunohistochemistry. Non-parametric methods were used to compare expression between the groups.

Results

Placental mRNA expression of GFAT1 was higher in women with GDM (2.16 (1.21-6.78) median (IQR) AU) than in women without (0.76 (0.48-2.25), P<0.05). OGT1 expression also was higher in women with GDM (2.53 (0.89-8.18)) vs. 0.49 (0.16-2.87), P<0.05. There was no difference in the expression of OGA. The expression of these genes was not correlated with maternal BMI or infant birth weight. GFAT is widely expressed in the placenta.

Conclusion

Maternal GDM is associated with an increase in the placental expression of two key enzymes in the HSP. The direction of change is suggestive of a funneling of glucose toward the HSP and increased O-GlcNAc cycling. These changes are not associated with changes in infant birth weight.
P2.79

Changes in exosome concentration and exosomal endothelin-1 throughout late gestation and early postpartum in dairy cows
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Background: Placental exosomes have recently been recognised as playing a role in maternal changes to support fetal development during human pregnancy; however, the exosome profile in dairy cows during gestation has not been established. The aim of this study was to determine the gestational-age release profile and bioactivity of exosomes in bovine plasma.

Methods: Plasma samples (n=4 cows sampled five times during gestation) were obtained from pregnant Holstein-Friesian cows at days 133, 182, 238 and 282 of gestation, as well as at days 7-13 postpartum. Extracellular vesicles were isolated from plasma by differential centrifugation (from 20 000 x g to 100,000 x g), exosomes were enriched by layering on top of a discontinuous iodixanol gradient (OptiPrep™) and centrifuged at 100,000 g for 20 h. Twelve fractions were obtained and separated into low (1.088 to 1.10 g/ml), exosomes (1.12-1.18 g/ml) and high (1.22 to 1.29 g/ml) density. Fractions were further characterised by size distribution (NTA, NanoSight NS500), presence of enrich exosomal markers (TSG101 and CD63) by Western blot analysis, morphology (electron microscopy) and protein content (mass spectrometry). Finally, the protein abundance of endothelin-1 in exosomes was determined by Western blot.

Results: Enriched exosome fractions were identified as cup-shape vesicles with diameters around 100 nm and positive for TSG101 and CD63 markers. A significant effect of gestational age on plasma exosome number was identified (p = 0.027). In addition, a significant difference was identified between cows (p=0.035). Interestingly, endothelin-1 protein was highly expressed in exosomes compared with low and high fractions across gestation. Exosomal endothelin-1 protein abundance increased progressively with stage of gestation and regression analysis indicated that gestational age accounted for 60% of the observed variation.

Conclusions: In dairy cows, the concentration of exosomes in plasma changes with gestational age. Endothelin-1 is mainly transported into exosomes rather than other extracellular vesicles. While the role of exosomes in dairy cow reproduction remains to be established, this work verifies that they are measurable and linked in a meaningful way to their reproductive development.

P2.80

Selective increase of exosome release from bovine endometrial stromal cells under hypoxic conditions
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Background: Invasion and migration of placental cells in the maternal tissues are controlled by the endometrium in bovine pregnancy. Cell-to-cell communication between endometrial cells is critical for implantation. The aim of this study was to establish the effect of oxygen tension on the release and bioactivity of exosomes from bovine endometrial stromal cells.

Methods: Bovine endometrial stromal cells isolated from the intercaruncular areas (i.e. ICAR*) were cultured under 8% and 1% O2 for 48 hours (n=18). ICAR proliferation and migration rates under hypoxic conditions were established using a real-time, live-cell imaging system (Incucyte®3D). Extracellular vesicles (EVs) were isolated from ICAR-conditioned media by differential centrifugation (i.e. 100,000 x g, n=3 independent isolations) and exosomes were enriched by layering on top of a discontinuous iodixanol gradient (OptiPrep™), which were further centrifuged at 100,000 x g for 20 h. Twelve fractions were obtained and characterised by size distribution (NTA, NanoSight NS500), presence of enrich exosomal markers (TSG101 and CD63) by Western blot and morphology (electron microscopy). The effect of hypoxia on exosome release from ICAR was determined by quantifying the immunoreactive exosomal marker, CD63.

Results: Hypoxia (i.e. 1% O2) significantly decreased (p<0.05) ICAR proliferation and migration by ~60% and ~18%, respectively compared with values observed at 8% O2. EVs protein concentration was lower in 1% compared to 8% O2 (168 ± 124 vs. 73.2 ± 18 µg protein / 106 cells). However, the number of vesicles positive for CD63 in the EVs pellet was significantly higher (1.5-fold) under an atmosphere of 1% O2 compared to 8% O2. Hypoxia induced exosome release by ~1.36 fold compared to 8% O2. Interestingly, the number of exosome vesicles per µg of protein was not significantly affected by oxygen tension (i.e. 3.1 x 106 ± 2.5 x 106 and 1.2 x 107 ± 6.6 x 107 at 8% and 1%, respectively).

Conclusions: The data obtained in this study established that hypoxia selectively increase the release of exosomes from ICAR cells. While the role of ICAR-derived exosomes in vivo remains to be established, their release and potential interactions with trophoblast cells could be an adaptive response during implantation.

* ICAR were generously provided Dr Michel A Fortier (Université Laval, Ste-Foy, Québec, Canada)

P2.81
**Differential expression of microRNAs that regulate TLR pathways in the placenta and cord blood.**

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Preterm delivery accounts for 75% of neonatal deaths, with half related to inflammation. This may be due to an immature and dysregulated innate immune response. Exposures that commonly accompany preterm birth, including maternal smoking and antenatal steroids, may exacerbate the immaturity of the preterm infant’s innate immune response. Placenta and cord blood from preterm neonates exhibit decreased Toll-like Receptor (TLR) and pro-inflammatory cytokine expression compared to term. TLR activation is regulated by microRNAs (miRs) which repress the translation of inflammatory mediators. This study aims to examine the expression of miRs involved in regulating TLR activation in preterm placenta and cord blood.

Placenta and cord blood were collected following term (≥37 weeks gestation, n=27) and preterm delivery (<37 weeks, n=20). The expression of let7e, miR155, miR146a, and miR106a were characterised by qRT-PCR relative to RNU48 in placenta and cord blood at baseline and following stimulation with lipopolysaccharide (LPS; TLR4 agonist) and peptidoglycan (PGN; TLR2 agonist). Data were analysed according to gestational age, betamethasone exposure (n=10) and smoking (n=9).

Placental miR expression did not vary according to gestational age or betamethasone exposure. Maternal smoking was associated with decreased placental miR146a (p=0.032) and miR155 (p=0.067) expression, and decreased cord blood miR155 (p=0.05). Following in-vitro stimulation with LPS, decreased miR155 (p=0.0001) and let7e (p=0.043) expression were observed in preterm cord blood compared to term cord blood. Preterm cord blood also decreased miR155 expression following stimulation with PGN (p=0.041).

These results demonstrate differential expression of miRs that regulate TLR signalling in term and preterm placenta. The decrease in placental miR155 and miR146a may contribute to an increased sensitivity to inflammation during the preterm period, potentially contributing to the pathophysiological process of preterm birth. Following TLR stimulation, reduced expression of let7e and miR155 in preterm cord blood potentially contributes to a pro-inflammatory innate immune system bias.

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**Confocal imaging of clarified placenta terminal villi for evaluation of 3D intervillous branching structure**

George Merz\(^1\), Wojciech Kaczmarski\(^2\), Valerie Schwenk\(^1\), Alee Andoyan\(^1,2\), Philip Necaise\(^1,2\), Daphne Landau\(^1\), Carolyn Salafia\(^1,3\), Ruchit Shah\(^1,3\)

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There is accumulated evidence that stressors (e.g. maternal obesity, smoking, diabetes, and hypertension) can have lasting effects on the health of the developing fetus, delivered infant, and adult. These stressors can impact the placenta vascular network as reduced chorionic surface vessels and branch points. However, little is known about their impact at the “micro” level of the vascular tree i.e. the terminal villi. As the seat of nutrient, gas and waste exchange between maternal and fetal blood they are likely targets for these stressors.

To date, 3D villous structure has been assessed by scanning electron microscopy or serial sectioning, which limit analysis to targets for these stressors.

**Objective:** To use a combination of tissue clarification and single photon confocal microscopy to overcome the ~50um depth limit of fluorescence recovery inherent with single photon confocal microscopy which precludes the identification of higher order network structure

**Methods:** Pieces of villus tree (~5-55um) from midway between chorionic and basal plates were soaked overnight in 7% acrylamide in PBS, with bis-acrylamide and polymerized. The embedded tissue was passively clarified in 0.2% NaBorate and 4 % SDS for 7-14 days, washed in PBS overnight and stained with acridine orange or eosin-Y. Stacks of the stained tissue were acquired by confocal microscopy, processed in Imaris and rendered with Amira (Visualization Services Group, Burlington,MA)

**Results:** Clarification allowed imaging through ~ 600-800 um, a more than 10-fold improvement in assessable depth of field, enabling us to acquire image stacks and render their 3D structure to visualize the distal villous architecture.

Figure 1: 3D rendering of clarified villus tree sample

**Conclusion:** With the successful clearing of the terminal villi we now have a platform on which to develop strategies for the analysis of multilevel villus branching networks that are currently unobtainable.
Transfer study of Trastuzumab® across the human placenta barrier in an ex-vivo human placenta perfusion model
Michael Gruber¹, Bettina Amtmann¹, Birgit Hirschmugl¹, Uwe Lang¹, Gernot Desoye¹, Christian Wadsack¹.
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Background: The increase in gestational age leads to enhanced probabilities of occurring breast cancer during pregnancy. Therefore exposure of targeted therapeutics, like Trastuzumab®, a monoclonal IgG1 antibody which blocks the human epidermal growth factor receptor 2 protein, to the fetus is a rare but increasing problem. In case studies the development of reversible oligohydramnios and renal problems in new-borns could be observed after Trastuzumab® administration during gestation. Transport of IgG antibodies across the placenta is described in the literature. There is no reported experience with the use of the agent in pregnancy and possible effects on the fetus.

Objectives: In this study transport of Trastuzumab® across the human term placenta was examined.

Methods: The dual ex vivo human placental perfusion model was used to analyse the transfer of Trastuzumab® across the placental barrier (n=3). Artificial maternal and fetal circulation of a term placenta cotyledon by using cannulas was established directly after delivery. Closed maternal and fetal circulation was used as experimental approach. Nutrition and oxygen supply was online monitored to keep the tissue vital over several hours. A therapeutic reasonable antibody concentration (50µg/ml) was used; quantification was performed by ELISA. The presence of Her2/Neu in placental tissue was determined by immunohistochemically staining.

Results: Transport of Trastuzumab® from maternal to fetal circulation could not be observed during 90min perfusion of the tissue. A significantly 30% decrease of Trastuzumab® concentration was detectable in maternal perfusates. A positive staining of the Her2/Neu receptor within the syncytiotrophoblast was shown.

Conclusions: Results indicating that Trastuzumab® does not cross the human placenta although Her2/Neu receptor staining was detectable at the maternal side of the placenta suggesting binding or even placental uptake of the drug. Secondary effects on placental function and/or impact on fetal development can’t be ruled out, and drug should be used with caution.
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