IFPA 2016

Placenta: Back to the Basics

Sept. 13–16, 2016
Portland, Oregon
Oregon Convention Center
IFPA 2016

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Department of Pathology
Department of Obstetrics and Gynecology
Bob and Charlee Moore Institute for Nutrition & Wellness
Oregon National Primate Research Center
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Dear colleagues,

On behalf of the local organizing committee, Oregon Health & Science University (OHSU) and the Placenta Association of the Americas (PAA) it gives me great pleasure to welcome you to Portland, Oregon for the 2016 International Federation of Placenta Associations (IFPA) meeting. Research in the last five years has highlighted for us the critical role the placenta plays in health across generations, not only regulating fetal growth and development but also subsequent development of disease in the offspring and also in the mother who carried that placenta. In the U.S., this has been recognized in the Human Placenta Project, an unprecedented effort focused on understanding the many aspects of placental function in relation to these vital roles. Under the theme of “Placenta: Back to the Basics” we have assembled a topical program that highlights cutting edge techniques and advances from other disciplines that can be brought to bear on our field to increase our basic understanding of placental function. Our opening keynote speakers will perfectly illustrate this and also highlight our research strengths at OHSU. The three symposia address contemporary issues including the application of “omics” and systems biology to the placenta, trophoblast specification/progenitors and viral infection and the placenta. The latter symposia will include some exciting new data on ZIKV and the placenta. In addition we have two poster sessions and 12 workshops planned where the chairs have been urged to pose controversial questions and encourage participation by attendees. New this year for IFPA has been the organization of two satellite meetings on placental pathology and multiscale imaging techniques, both of which have attracted strong attendance.

IFPA has always prided itself on development of new investigators, the lifeblood of our discipline. This year with the assistance of Elsevier, the publishers of Placenta and Trophoblast Research, we have been able to provide travel awards in addition to those traditionally supported by Y.W. (Charlie) Loke and by the National Institutes of Health conference grant. Also new this year are additional poster awards, the IFPA Early Career Research Award for the best oral presentation by a junior faculty member and the Andrée Gruslin Award which recognizes the achievements of a mid-career female investigator.

Finally, I would like to thank our sponsors, the members of our local organizing committee and the staff of the OHSU Bob and Charlee Moore Institute for Nutrition & Wellness for their generous support and unflagging help in putting on this meeting. Please enjoy the meeting, soak up the science, develop new collaborations and take some time to explore the many delights of Portland and the wonderful scenery of Oregon.
Dear Friends and Colleagues in IFPA,

Welcome to another exciting meeting of the International Federation of Placenta Associations, IFPA 2016 in Portland, Oregon. This city is home to an array of researchers interested in the placenta and they have combined, under the leadership of Dr. Leslie Myatt, to produce an exceptional scientific schedule for our annual international conference.

Many of the conference topics address recent innovations that will provide us with new tools and concepts to help us pursue placental research. The “Back to Basics” theme of the meeting highlights the results of novel collaborations with other disciplines including bioengineering, biophysics and bioinformatics. The products of these collaborations are leading us into new avenues of investigation in fields such as placental modeling, imaging and multidimensional analysis.

I, along with all of our long-standing IFPA members, want to give an especially warm welcome to the early career researchers (ECRs), many of whom will never have been to an IFPA meeting. One of IFPA's primary goals as a non-profit educational organization is to support young investigators as the future of our research field. We are dedicated to ensuring your continued attendance and immersion in placental and perinatal biology. That is why we will be making over 60 travel awards for ECRs. We would like to thank Dr. Y.W. (Charlie) Loke, Professor Emeritus at Cambridge University for his continued generosity in funding the Y.W. Loke Travel Awards, which support up to 40 ECRs from around the globe. We also thank the U.S. National Institutes of Health for their support of 10 U.S.-based ECRs. Our publishers Elsevier have also generously increased their support of IFPA enabling us to provide a further 16 travel awards. We are also devising ways to help those who have just become independent investigators as well as our mid-career investigators, one of whom will receive the prestigious Andrée Gruslin Award, presented for the first time this year with the generous assistance of Elsevier. We look to your advice on ways to extend our support.

Remember that your attendance at this meeting makes you a member of IFPA. There are no dues, no annual sign-up. As a growing organization, IFPA needs your input on organization and future directions. It is important that you participate and I invite you to attend our Annual General Meeting (Friday, September 16, at 12:30 pm) to help us to guide IFPA forward.

I know you will appreciate this meeting and the surrounding city. Portland is unique — it is beautiful, environmentally conscious, creative and has excellent breweries, wineries, restaurants and cafes. The organizers have made it easy to get around, so explore this unique community. Enjoy the science, the place and above all, the people.
## PROGRAM AT A GLANCE

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<th>SEPT. 12</th>
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<td>New Investigator Session 1</td>
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<td>Andrée Gruslin Award Lecture</td>
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<td>Lunch IFPA AGM, Final Poster Judging</td>
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## Registration Hours

Located in the Portland Ballroom Lobby

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<td>Tues, Sept. 1</td>
<td>8 a.m. – 5 p.m.</td>
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<td>Weds, Sept. 14</td>
<td>7:30 a.m. – 6 p.m.</td>
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<td>Thurs, Sept. 15</td>
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## Speaker Ready Room Hours

Located in E145

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<td>Tues, Sept. 13</td>
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<td>Fri, Sept. 16</td>
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## Committee Lists

### Local Organizing Committee
- Bernadette Battilega
- Robert Brace, Ph.D.
- Shawn Chavez, Ph.D.
- Ceci Cheung, Ph.D.
- Antonio Frias, M.D.
- Peta Grigsby, Ph.D.
- Liana Haywood, MPH
- Alina Maloyan, Ph.D.
- Terry Morgan, M.D., Ph.D.
- Leslie Myatt, Ph.D.
- Lisa Rhuman
- Victoria Roberts, Ph.D.
- Kent Thornburg, Ph.D.
- Amy Valent, D.O.

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- Berthold Huppertz, Ph.D.
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- Thomas Jansson, M.D., Ph.D.
- Alicia Jawerbaum, Ph.D.
- Naihiro Kanayama, M.D., Ph.D.
- Yoshiki Kudo, M.D.
- Peter Mark, Ph.D.
- Padma Murthi, Ph.D.
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- Claire Roberts, Ph.D.
- Shigeru Saito, M.D.
- Carlos Salomon, Ph.D.
- Annette Staff, M.D., Ph.D.
- Cathy Vaillancourt, Ph.D. | Treasurer of IFPA
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Estela Bevilacqua
Chair

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CHAIR OF POSTER JUDGING AND AWARDS COMMITTEE

Theresa Powell, Ph.D.
Department of Pediatrics
University of Colorado, Anschutz Medical Campus
Dr. Lindner is currently a Professor of Medicine at Oregon Health & Science University where he is the Director of Cardiovascular Imaging and holds the M. Lowell Edwards Professorship of Cardiology. He received his medical degree and residency training in internal medicine at the University of Texas Southwestern Medical School, and received his cardiovascular training at the University of Virginia. Dr. Lindner has expertise in the fields of cardiovascular imaging and microvascular physiology; and is currently the principal investigator on several R01 grants from the National Institutes of Health and a grant from the NASA National Space and Biomedical Research Institute. His research laboratory has pioneered the use of contrast ultrasound for non-invasive molecular imaging of disease and the evaluation of microvascular function/dysfunction. Specific areas of research include: a) application of molecular imaging techniques for early detection of atherosclerosis and evaluation of new therapies for atherosclerosis; (b) molecular imaging of angiogenesis and stem cell therapy; (c) molecular imaging for early diagnosis of myocardial ischemic injury and inflammation; (d) microvascular dysfunction and endothelial abnormalities in atherosclerosis, insulin resistance, and sickle cell disease; (e) development of new methods for detecting and treating peripheral arterial disease, and (f) novel methods for site-targeted gene and drug delivery with ultrasound. Dr. Lindner is the Vice President for the American Society of Echocardiography and also serves as an Associate Editor for the Journal of the American Society of Echocardiography. He is on the Board of Directors and serves as the Vice-chair for the Exam Writing Committee for the National Board of Echocardiography.

Dr. Gray, a physicist and an engineer by training, serves as director for the Center for Spatial Systems Biomedicine (OCSSB) and associate director for biophysical oncology for the Knight Cancer Institute at OHSU. Dr. Gray’s research program applies experimental and mathematical tools to elucidate mechanisms by which genomic, transcriptional and proteomic abnormalities occur in selected cancers, including how these abnormalities alter the multiscale architecture of cancers and their microenvironments, cancer pathophysiology and how these changes contribute to cancer progression and response to therapy. He brings substantial experience in development and application of advanced measurement technologies. Past contributions include development of cytometric techniques for cell and genome analysis including high speed chromosome sorting, BrdUrd/DNA analysis of cell proliferation, fluorescence in situ hybridization (FISH), comparative genomic hybridization (CGH) and End Sequence Profiling (ESP). More recently, Dr. Gray has elucidated mechanisms of cancer progression and developed systems biology approaches for identification of molecular markers that predict and determine response to therapeutic treatment. Currently, Dr. Gray is developing aspects of the OCSSB that he founded and directs, to elucidate how genomic and epigenomic events influence the multiscale (Ångströms to millimeters) structures that enable function in cells and tissues. Dr. Gray has published over 350 peer reviewed papers, 100 reviews and commentaries, co-edited six books and has been awarded more than 80 US Patents. He currently serves on the NIH NCI Frederick National Laboratory for Cancer Research Advisory Board, and is US Councilor on the Board of the Radiation Effects Research Foundation (RERF) of Japan.
Francesco J. DeMayo received his BS in General Studies from Cornell University, Ithaca N.Y. and his M.S. and Ph.D in Physiology from Michigan State University, East Lansing MI. He then conducted his postdoctoral training at Baylor College of Medicine, Houston TX in the laboratory of Dr. David Bullock where he investigated the regulation of steroid control of the rabbit uteroglobin utilizing transgenic mice. He then joined the faculty at Baylor College of Medicine where he rose to the ranks of Professor and held the Cullen-Duncan-McAshan Endowed Chair in Cancer Research. He also served as Director of the Genetically Engineered Mouse Core. There he investigated the role of progesterone in the regulation of uterine biology developing novel mouse models to ablate or express genes in the uterus. In 2015 he moved to the NIEHS as Senior Principal Investigator and Deputy Chief of the Reproductive and Developmental Biology Laboratory where he continues his work investigating the molecular mechanisms governing female reproduction and pregnancy.

Dr. Nathan Price is Professor & Associate Director of the Institute for Systems Biology (ISB) in Seattle. He is co-PI with Lee Hood of ISB’s 100K Person Wellness Project, and is a Co-Founder and member of the Board of Directors of Arivale, a scientific wellness company. He has won numerous awards for his scientific work, including a Howard Temin Pathway to Independence Award from NIH, an NSF CAREER award, a young investigator award from the Roy J. Carver Charitable Trust, and he was named as one of the inaugural “Tomorrow’s PIs” by Genome Technology and, most recently, as a Camille Dreyfus Teacher-Scholar. Dr. Price serves on editorial boards for many leading scientific journals including Science Translational Medicine and Cell Systems.
Tom Metz’s early career began in the study of diabetes mellitus, both types 1 and 2, and focused on the impact of chronic, cumulative chemical modification of tissue proteins by the products of browning (also known as Maillard) reactions. These non-enzymatic protein modifications are implicated in the development of diabetic complications, such as nephropathy and retinopathy. After completing his Ph.D. studies, Dr. Metz began post-doctoral studies at Pacific Northwest National Laboratory (PNNL) where he trained in advanced MS-based proteomics analyses, and developed methods for MS-based metabolomics and lipidomics analyses. Currently, he is the technical lead for metabolomics at PNNL and manages a team of 18 scientists that focuses on applications of high throughput proteomics, metabolomics, and lipidomics methods to various biological questions. At PNNL, he has worked to develop state-of-the-art, high throughput MS-based metabolomics and lipidomics capabilities that are comparable to the existing world-class MS-based proteomics capabilities. Currently, Dr. Metz is PI of the Proteomics Laboratory for The Environmental Determinants of Diabetes in the Young consortium, PI of the Metabolomics Core for the Undiagnosed Diseases Network, and Co-PI of a Proteomics, Metabolomics, and Lipidomics Core for a NIAID Systems Biology for Infectious Disease Research Center.

Andrew Adey, Ph.D., is Assistant Professor of Molecular and Medical Genetics in the Oregon Health & Science University School of Medicine. Dr. Adey started out in biotechnology development at the University of Texas where he researched alternative applications of microarrays in the lab of Andrew D. Ellington, Ph.D. He later served as interim director of the UT microarray core facility and then helped set up the UT genome sequencing and analysis facility in the early days of next generation sequencing. He then completed his doctoral studies in the Molecular and Cellular Biology Program at the University of Washington in the lab of Jay Shendure, M.D., Ph.D. in the Genome Sciences Department. Previous research highlights include pioneering a novel transposase-based method for rapid, low-input DNA sequencing library construction, which he extended to the genome-wide analysis of DNA methylation. He also applied long-range sequencing methods to produce the first haplotype resolved genome and epigenome of an aneuploid cell line, HeLa, where he investigated the role of haplotype and copy number on the epigenetic and transcriptional landscape. Dr. Adey plans to continue his focus on the development and implementation of novel strategies to investigate the epigenome with high precision. This includes single cell approaches to disambiguate epigenetic and transcriptional heterogeneity within populations of cells, which is typically obscured by bulk preparation methods. This work will provide insight into the dynamic regulatory landscape of cells and may reveal functional and targetable subpopulations in the context of disease intervention.
Dr. David Weinberg is a Program Officer in the Eunice Kennedy Shriver National Institute of Child Health and Human Development and the Project Lead for NICHD’s Human Placenta Project. David earned his Ph.D. at the Johns Hopkins University and spent almost 20 years at the pharmaceutical company Merck, where he led projects focused on drug discovery for the treatment of obesity and developed expertise in translational research. In 2009 David moved to NIH as a Scientific Review Officer where he managed the review of applications on female reproduction and contraception. In 2014, he became the project lead for the HPP, whose goals are to develop a deep understanding of human placenta development and function, accelerate the capability for safe, real-time assessment of these processes across pregnancy, utilize these methods to distinguish normal from abnormal pregnancy trajectories, and ultimately facilitate development of interventions that may lead to better pregnancy outcomes.

Dr. Drewlo is a trained molecular cell biologist with a background in biotechnology and placental pathology, working at the Department of Obstetrics and Gynecology at Wayne State University School of Medicine as an Associate Professor. He received his Ph.D. in 2006 in Germany with the well-known placentologist Dr. Peter Kaufmann. Dr. Drewlo performed his post-doctoral, translational research training at the Lunenfeld Research Institute Toronto, Canada. During this time, he published over twenty papers and accepted in 2013 a faculty position at Wayne State University. The goal of is research is to close the gap between diagnosis of placental dysfunction and treatment. He has published more than forty-six peer reviewed papers and received the Basil O’Connor Starter Scholar Research Award (2014) and five year NIH R01 funding (2015) to investigate human trophoblast differentiation, and the potential of novel intervention strategies for preeclampsia. Dr. Drewlo has interest in the early detection of fetal and placental disorders along with deciphering the underlying molecular mechanisms of abnormal placentation.
Dr. Hubert Schorle is Professor for Developmental Pathology at University of Bonn Medical School, Bonn, Germany. Dr Schorle studied Biology (Diploma) at Bayerische Julius Maximilians Universität, Würzburg, Germany from 1989-1991 and then received his Dr.rer.nat (Doctor of Natural Sciences) from the Institute for Virology, Universität Würzburg, Germany in 1991. From 1993-1996 he undertook a postdoctoral fellowship with Dr. R. Jaenisch at the Whitehead Institute for Biomedical Research/M.I.T., Cambridge, USA before becoming a Junior Research Group Leader at the Institute for Toxicology and Genetics, Forschungszentrum Karlsruhe from 1996-2001. He subsequently moved to the University of Bonn in 2001 as head of the Transgenic Facility of the Institute of Pathology and in 2004 was appointed Professor of Developmental Biology. Dr Schorle is interested in several aspects of basic biomedical research, including trophoblast stem cells and development of the placenta, germ cells and germ cell tumors and applying transgenic approaches to address the genetics and epigenetics of these systems.

The focus of my research is molecular mechanisms of trophoblast lineage specification, proliferation, and differentiation as related to placental development and disease. As a student of the medical scientist training program, I was trained in the laboratories of Dr. Carol Otey and Ann Sutherland, and applied cell biological techniques to the discovery of a novel actin-binding protein, which also plays a unique role in trophoblast differentiation. Later, as a research fellow in the laboratory of Dr. David Milstone at the Center for Excellence in Vascular Biology at Brigham and Women’s Hospital, I expanded my training to mouse placental biology, including derivation of trophoblast stem (TS) cells from mouse blastocysts, while studying the role of the nuclear receptor PPARg, in trophoblast differentiation. In order to become better acquainted with methods in human placental research, I also attended the week-long “Human Placenta Workshop” (a laboratory course directed by Dr. Anne Croy) at Queen’s University in Kingston, Ontario, in October 2007. On the clinical side, I am a board-certified anatomic pathologist with subspecialty training in gynecologic and perinatal pathology. Since obtaining an independent position at UC San Diego, I have established a strong research program focusing on human placental development and disease. I have developed optimized protocols for derivation of trophoblast from human pluripotent stem cells and identified a key pathway regulating this process. We are currently expanding on this work, probing in detail the human trophoblast stem cell niche and establishing pluripotent stem cell-based models for normal and diseased human placental development.
Dr. Soumen Paul completed Ph.D. in molecular biology from the University of Calcutta, India and postdoctoral training in molecular pharmacology at the University of Wisconsin, Madison. In 2007, Dr. Paul joined the University of Kansas Medical Center (KUMC) as an Assistant Professor. In 2012, he was promoted to an Associate Professor with tenure. Dr. Paul is also the Director of the graduate program within the Department of Pathology and Laboratory Medicine at KUMC. Dr. Paul’s research is focused on understanding molecular mechanisms that control cellular identity during mammalian development. A major research effort in Dr. Paul’s laboratory is to delineate transcriptional mechanisms that regulate self-renewal, differentiation, and function of trophoblast stem/progenitors cells. Dr. Paul’s research has provided fundamental insights into the transcriptional mechanism that control development of trophoblast lineage in early mammalian embryo. In recent years, he has focused on defining conserved transcriptional mechanisms that control development of human trophoblast lineage and to assess how alterations of those mechanisms contribute to pathological pregnancies.

Dr. Daniel Streblow received his B.S. in Pharmacology/Toxicology from the University of Wisconsin-Madison in 1992. He graduated from University of Wisconsin-Madison with a Ph.D. in Viral Pathogenesis in 1997. Dr. Streblow received a NIH post-doctoral fellowship and worked with Dr. Jay A. Nelson in the Department of Molecular Microbiology and Immunology at Oregon Health & Science University. He is currently an Associate Scientist at the Vaccine and Gene Therapy Institute and Research Assistant Professor at the Department of Molecular Microbiology and Immunology.
Shannon Bainbridge is an assistant professor in the Interdisciplinary School of Health Sciences at the University of Ottawa (Ottawa, Canada), with cross appointment to the Department of Cellular and Molecular Medicine and affiliate investigator status at the Ottawa Hospital Research Institute (OHRI). She obtained her Ph.D. in 2001 from Queen’s University (Kingston, ON) with a dissertation that focused on the effects of maternal smoking on placental development and function. Professor Bainbridge went on to complete two post-doctoral fellowships, the first at Magee-Womens Research Institute (Pittsburgh, PA) and the second at the The Lunenfeld-Tanenbaum Research Institute at Mount Sinai Hospital (Toronto, ON). In both fellowships her research examined how different biological and genetic influences affect early placental development in the context of both healthy and pathological pregnancies.

Dr. Bainbridge’s current research program focuses on placental dysfunction, specifically as it relates to two common and debilitating complications of pregnancy, preeclampsia and intrauterine fetal growth restriction. The primary aims of her research program are to identify distinct subclasses of placental disease underlying these complications using a combination of molecular profiling and detailed placental histopathology. Work in her laboratory further aims to translate these findings into subclass-specific biomarker panels, used to screen and identify these different subclasses of disease, as well as etiology-driven therapeutic interventions. Dr. Bainbridge’s research program is funded by the Canadian Institute of Health Research, the Canadian Foundation for Innovation, Molly Towell Perinatal Research Foundation and the Preeclampsia Foundation.

Dr. Miller received his undergraduate and graduate training at Dartmouth College and Dartmouth Medical School with Professors William Berndt, Gilbert Mudge, Virgil Ferm and William Layton in Pharmacology/Toxicology/Teratology. He also did post graduate training at Jefferson Medical College with Professors Robert Brent and Tom Koszalka in Developmental Biology/Teratology. In 1974, he joined the Departments of Obstetrics/Gynecology at the University of Rochester under Professor Henry A. Thiede and of Pharmacology/Toxicology under Professor Louis Lasagna as an Assistant Professor. He is currently Professor of Obs/Gyn, of Environmental Medicine and of Pathology and Clinical Laboratory Medicine.

In 1978, he began his career as an NIH Principal Investigator. He also has received an NIH Fulbright Distinguished Professorship, an NIH Senior International Fogarty Fellowship, the Teratology Society Distinguished Service Award, Recipient of the Bock Prize in Developmental Biology and Child Health, named a Fellow of the Academy of Toxicology Sciences and a member of the TERIS Advisory Board. At Rochester, he has been Associate Chair for Research in Obs/Gyn, Director of the Division of Research and Scientific Director of the NIH Women’s Reproductive Health Research Scholars program. In 1980, Dr. Miller assumed responsibility of the Rochester Trophoblast Conference Meetings with Dr. Thiede. He continued in this capacity until 2000, when the RTC transitioned into the Placenta Association of the Americas. He was also the founding Secretary of IFPA.
Antonio Frias is an Assistant Scientist in the Division of Diabetes, Obesity, & Metabolism and the DRDS. He is an Associate Professor and Maternal Fetal Medicine (MFM) subspecialist in the Department of Obstetrics & Gynecology, Director of the Diabetes and Pregnancy Program at OHSU and Director of the Maternal Fetal Medicine Fellowship. He completed medical school at the Mayo Clinic (1998), residency training in Obstetrics and Gynecology at the University of Utah (2002), and fellowship training in MFM at the University of Utah (2005). He joined the ONPRC as a Women’s Reproductive Health Research Fellow in 2007. Dr. Frias also serves on the OHSU Institutional Review Board. He is a reviewer for OHSU clinical research grants funded by the Center for Women’s Health Circle of Giving and the Moore Institute for Nutrition & Wellness.

Dr. Permar is a physician scientist focusing on the prevention and treatment of neonatal viral infections. She leads a research laboratory investigating immune protection against vertical transmission of neonatal viral pathogens, namely HIV and cytomegalovirus (CMV), using human cohorts and nonhuman primate models. Dr. Permar has made important contributions to the development of vaccines for prevention of vertical HIV transmission, defining both innate and adaptive immune responses that are associated with protection against infant HIV acquisition. Moreover, Dr. Permar is leading the development of HIV vaccine strategies in maternal/infant nonhuman primate models and clinical vaccine trials in infants. Dr. Permar has also contributed to understanding the immunology of perinatal CMV transmission and the pathogenesis of postnatal infection in preterm infants. Dr. Permar developed the nonhuman primate model of congenital CMV infection and uses this model for defining the immune correlates of protection against CMV transmission and vaccine development.

Dr. Permar has a Ph.D. in Microbiology/Immunology from Johns Hopkins Bloomberg School of Public Health in Baltimore, an M.D. from Harvard Medical School and completed her clinical training in pediatric infectious diseases at Children’s Hospital in Boston. She has received several prestigious early-stage investigator awards, including the Presidential Early Career Award in Science and Engineering (PECASE) and the Society for Pediatrics Research (SPR) Young Investigator Award.
Dr. Grigsby received her Ph.D., at the Monash University and has more than 10 years’ experience working with animal models of preterm labour (including sheep, rodents, and non-human primates). Dr. Grigsby is an Early Stage Investigator whose independent research program focuses on the development of new approaches to prevent prematurity and its adverse fetal and neonatal consequences.

Dr. Grigsby has developed an interdisciplinary, multi-institutional collaborative research program at the ONPRC, and in 2008 she was awarded the first NICHD K99/R00 Pathway to Independence Career Development Award at the ONPRC. She completed the K99 portion of this award under the mentorship of Dr. Miles J. Novy and in June 2009 transitioned to the Independent Phase of this award. She is currently appointed as an Professor in the Division of Reproductive & Developmental Sciences at ONPRC and holds a joint appointment as an Assistant Research Professor in the Department of Obstetrics and Gynecology, Division of Maternal-fetal Medicine at Oregon Health & Science University.

Dr. Grigsby’s current research program incorporates studies directed at the fundamental mechanisms of parturition, with an emphasis on therapeutic interventions for preterm labour associated with reproductive tract infections (i.e., Ureaplasma spp.), and for the prevention of subsequent fetal and neonatal sequelae, including lung and brain injury.
Dr. Claudia Göhner originally studied pharmaceutical biotechnology at the University of Applied Sciences Jena, Germany. She finished her studies with an engineering degree in 2009 as well as a Master of Sciences in 2012. During her master thesis, Claudia started her work in placenta research and reproductive immunology in the Placenta lab of the Department of Obstetrics at the University Hospital Jena, Germany. Here, she focused on the establishment of a quantification method of syncytiotrophoblast extracellular vesicles for early preeclampsia prediction. Her master thesis was awarded with the STIFT award for application-oriented study thesis of the Thuringian Foundation for Technology, Innovation and Science (Stiftung für Technologie, Innovation und Forschung Thüringen) and she received the best presentation award at the annual meeting of the Science Group for Molecular Biology of the German Society of Gynaecologic Endocrinology and Reproductive Medicine. Afterwards, Claudia continued her research within her PhD project about the functionality of syncytiotrophoblast extracellular vesicles in a cooperative project between the Placenta lab of the Department of Obstetrics at the University Hospital Jena, Germany and the Department of Obstetrics and Gynaecology of the University Medical Center Groningen, The Netherlands. Here, she focussed on the immunologic and pro-coagulant function of syncytiotrophoblast microvesicles and exosomes. Next to that, Claudia led the ex vivo placenta perfusion group of the Placenta lab for one year. During her PhD, Claudia was awarded several travel grants of the ProChance program of the Friedrich Schiller University, Jena, Germany and the International Federation of Placenta Associations as well as (project) grants of the Abel Tasman Talent program of the University of Groningen, The Netherlands and the Fonds Gezond Geboren, The Netherlands. Additionally, she received the New Investigator award of the German Society of Perinatal Medicine in 2013 and the Elsevier Trophoblast Research New Investigator Award of the International Federation of Placenta Associations in 2015. Claudia finished her Ph.D. in April 2016.
# Y.W. LOKE NEW INVESTIGATOR TRAVEL AWARDS

Supported by the generous endowment of Y.W. (Charlie) Loke, Emeritus Professor of Reproductive Immunology at the University of Cambridge and member of the IFPA and EPG. It offsets travel expenses for approximately 40 young investigators per meeting.

<table>
<thead>
<tr>
<th>NAME</th>
<th>COUNTRY</th>
<th>INSTITUTION</th>
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<tbody>
<tr>
<td>Natalie Aboustate</td>
<td>Australia</td>
<td>University of Adelaide</td>
</tr>
<tr>
<td>Pallavi Arora</td>
<td>India</td>
<td>All India Institute of Medical Sciences</td>
</tr>
<tr>
<td>Christian Castillo</td>
<td>Chile</td>
<td>University of Chile, Santiago</td>
</tr>
<tr>
<td>Wittaya Chaiwangyen</td>
<td>Germany</td>
<td>University of Jena</td>
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<tr>
<td>Natalie Cureton</td>
<td>UK</td>
<td>University of Manchester</td>
</tr>
<tr>
<td>Omar Elfeky</td>
<td>Australia</td>
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</tr>
<tr>
<td>Leonardo Ermini</td>
<td>Canada</td>
<td>The Hospital for Sick Children, Toronto</td>
</tr>
<tr>
<td>Phelipe Favaron</td>
<td>Brazil</td>
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<tr>
<td>Karolin Frohlich</td>
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<tr>
<td>Lobke Gierman</td>
<td>Norway</td>
<td>Norwegian University of Technology</td>
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<tr>
<td>Catherine Gilmore</td>
<td>UK</td>
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<tr>
<td>Eva Haeussner</td>
<td>Germany</td>
<td>Ludwig-Maximillians University</td>
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<td>Hirotaki Hamada</td>
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<tr>
<td>Xiao Huang</td>
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<td>University of Bern</td>
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<tr>
<td>Hanna Huebner</td>
<td>Germany</td>
<td>Friedrich-Alexander University, Erlangen</td>
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<tr>
<td>Mai Inagaki</td>
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<tr>
<td>Shrey Kohli</td>
<td>Germany</td>
<td>Otto-von-Guericke University Magdeburg</td>
</tr>
<tr>
<td>Yuan-Yuan Liu</td>
<td>China</td>
<td>Guangzhou Medical University</td>
</tr>
<tr>
<td><strong>NAME</strong></td>
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<tr>
<td>Jiangwen Lu</td>
<td>China</td>
<td>Shanghai Jiao Tong University</td>
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<tr>
<td>Alejandro Majali-Martinez</td>
<td>Austria</td>
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<td>UK</td>
<td>Brunel University</td>
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<td>Uzma Nadeem</td>
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<td>Fen Ning</td>
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<td>Wendy Phoswa</td>
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<td>Katie Powell</td>
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<tr>
<td>E. Magda Price</td>
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<tr>
<td>Patricia Romagnoli</td>
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<td>Isidora Rovic</td>
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<tr>
<td>Tamara Saez</td>
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<td>University of Groningen</td>
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<tr>
<td>Julian Sallais</td>
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<tr>
<td>Volodymyr Shablii</td>
<td>Ukraine</td>
<td>Institute of Cell Therapy Kiev</td>
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<td>Gordon Stevenson</td>
<td>Australia</td>
<td>Texas Tech University Medical Center</td>
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<td>Mancy Tong</td>
<td>New Zealand</td>
<td>University of Auckland</td>
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<td>Rebecca Wilson</td>
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<tr>
<td>Samantha Wilson</td>
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<tr>
<td>Karin Windsperger</td>
<td>Austria</td>
<td>Medical University of Vienna</td>
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<tr>
<td>Frances Wong</td>
<td>Canada</td>
<td>University of Toronto</td>
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## TRAVEL AWARDS

**ELSEVIER TRAVEL AWARDS**

Generously supported by Elsevier Ltd., Publishers of Placenta and Trophoblast Research, to allow new investigators in any aspect of placental research to attend the annual IFPA meeting.

<table>
<thead>
<tr>
<th>NAME</th>
<th>COUNTRY</th>
<th>INSTITUTION</th>
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<tbody>
<tr>
<td>Renee Albers</td>
<td>USA</td>
<td>Wright State University</td>
</tr>
<tr>
<td>Samantha Benton</td>
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<td>University of Ottawa</td>
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<tr>
<td>Cassidy Blundell</td>
<td>USA</td>
<td>University of Pennsylvania</td>
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<tr>
<td>Sonia da Silva-Arnold</td>
<td>USA</td>
<td>Hackensack University Medical Center</td>
</tr>
<tr>
<td>Francesca Diaz-Perez</td>
<td>Austria</td>
<td>Medical University of Graz</td>
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<tr>
<td>Vernon Ebegboni</td>
<td>UK</td>
<td>Nottingham Trent University</td>
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<tr>
<td>Roberta Hannibal</td>
<td>USA</td>
<td>Stanford University</td>
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<tr>
<td>Naoyuki Iwahashi</td>
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<tr>
<td>Junya Kojima</td>
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<td>Katherine Leavey</td>
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<tr>
<td>Li-Yang Ma</td>
<td>China</td>
<td>Chinese Academy of Sciences, Beijing</td>
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<td>Diana Morales-Prieto</td>
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<td>Pinki Nandi</td>
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<td>University of Western Ontario</td>
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<tr>
<td>Francesca Soncin</td>
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<td>University of California San Diego</td>
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<td>Yu Takahashi</td>
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<td>Maja Weber</td>
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<td>University of Jena</td>
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</table>
**NATIONAL INSTITUTES OF HEALTH NEW INVESTIGATOR TRAVEL AWARDS**

Supported by a R13 Conference grant awarded to the International Federation of Placenta Associations by the Eunice Kennedy Shriver National Institute of Child Health and Human Development to enable U.S.-based new investigators in any aspect of placental research to attend the annual IFPA meeting.

<table>
<thead>
<tr>
<th>NAME</th>
<th>INSTITUTION</th>
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<tbody>
<tr>
<td>Jennifer Adibi</td>
<td>University of Pittsburgh</td>
</tr>
<tr>
<td>Maira Carillo</td>
<td>Texas Tech University</td>
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<tr>
<td>Damayanti Chakraborty</td>
<td>University of Kansas Medical Center</td>
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<tr>
<td>Stephanie Chassen</td>
<td>University of Colorado Anschutz Medical Campus</td>
</tr>
<tr>
<td>Caitlin Dunn-Fletcher</td>
<td>Cincinnati Children's Hospital Medical Center</td>
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<tr>
<td>Elizabeth Enninga</td>
<td>Mayo Clinic</td>
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<td>Rebecca Jessel</td>
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<td>Che-Ying Kuo</td>
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<tr>
<td>Sze Ting Kwan</td>
<td>Cornell University</td>
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<tr>
<td>Parisa Mirbod</td>
<td>Clarkson University</td>
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<tr>
<td>Sean Nguyen</td>
<td>Michigan State University</td>
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<tr>
<td>Alison Paquette</td>
<td>Institute of Systems Biology, Seattle</td>
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<tr>
<td>Calais Prince</td>
<td>University of Texas Health Science Center San Antonio</td>
</tr>
<tr>
<td>Abhijeet Sharma</td>
<td>Weil Cornell Medical School</td>
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<tr>
<td>Bryce Wolfe</td>
<td>University of Wisconsin Madison</td>
</tr>
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# Detailed Program

<table>
<thead>
<tr>
<th><strong>Tuesday Sept. 13, 2016</strong></th>
<th><strong>Time</strong></th>
<th><strong>Room</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>IFPA Executive Committee</strong></td>
<td>8:30 a.m.</td>
<td>E147–148</td>
</tr>
<tr>
<td><strong>Executive Committee Lunch (invitation only)</strong></td>
<td>12:30 p.m.</td>
<td>E147–148</td>
</tr>
<tr>
<td><strong>Opening Address</strong></td>
<td>2 p.m.</td>
<td>Portland Ballroom 251–258</td>
</tr>
<tr>
<td>Leslie Myatt, Ph.D.</td>
<td></td>
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<tr>
<td>IFPA 2016 chair</td>
<td></td>
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<tr>
<td>Nick Illsley, Ph.D.</td>
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<tr>
<td>IFPA president</td>
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<tr>
<td><strong>Keynote Speakers</strong></td>
<td>2:30 p.m.</td>
<td>Portland Ballroom 251–258</td>
</tr>
<tr>
<td>Moderator</td>
<td></td>
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<tr>
<td>Kent Thornburg, Ph.D.</td>
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<tr>
<td><strong>Key.01</strong></td>
<td></td>
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<tr>
<td>Advanced ultrasound imaging: insights from perfusion and molecular imaging</td>
<td></td>
<td></td>
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<tr>
<td>Jonathan R. Lindner, M.D., Oregon Health &amp; Science University, Portland OR, USA</td>
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<tr>
<td>Exploring the architectures of life at multiple scales</td>
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<tr>
<td>Joe Gray, Ph.D., Oregon Health &amp; Science University, Portland OR, USA</td>
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<tr>
<td><strong>Refreshments</strong></td>
<td>4 p.m.</td>
<td>Portland Ballroom Foyer</td>
</tr>
<tr>
<td><strong>National Institutes of Health Lecturer</strong></td>
<td>4:30 p.m.</td>
<td>Portland Ballroom 251–258</td>
</tr>
<tr>
<td>Moderators</td>
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<tr>
<td>Stacy Zamudio, Ph.D., Martin Knöfler, Ph.D.</td>
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<tr>
<td>Investigating the molecular mechanisms regulated by progesterone governing the ability of the uterus to support pregnancy</td>
<td></td>
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<tr>
<td>Francesco DeMayo, Ph.D., National Institute of Environmental Health Science, USA</td>
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</tbody>
</table>
### TUESDAY SEPT. 13, 2016

<table>
<thead>
<tr>
<th>OPENING RECEPTION</th>
<th>TIME</th>
<th>ROOM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Presentation of New Investigator Loke Awards, NIH Awards and Elsevier Awards</td>
<td>6:30 – 8:30 p.m.</td>
<td>Doubletree Hotel</td>
</tr>
</tbody>
</table>

Graham Burton, Ph.D., Joan Anuels, Leslie Myatt, Ph.D.

### WEDNESDAY SEPT. 14, 2016

<table>
<thead>
<tr>
<th>AWARDS COMMITTEE</th>
<th>TIME</th>
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<tbody>
<tr>
<td></td>
<td>7:30 a.m.</td>
<td>E147-148</td>
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### NEW INVESTIGATOR/*JUNIOR FACULTY SESSION 1

<table>
<thead>
<tr>
<th>Moderators</th>
<th>TIME</th>
<th>ROOM</th>
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</thead>
<tbody>
<tr>
<td>Alicia Jawerbaum, Ph.D., Charles Graham, Ph.D.</td>
<td>8:30 a.m.</td>
<td>Portland Ballroom 251–258</td>
</tr>
</tbody>
</table>

#### Ni1.01

**Transcriptomic profiling of sncRNA in placenta by RNAseq**

E. Magda Price, B.S., University of British Columbia, Canada

#### Ni1.02

**Impaired gene-expression and epigenetic regulation of Retinoic Acid Receptor Responder 1 in preeclampsia and choriocarcinoma**

Hanna Huebner, B.S., Friedrich-Alexander University Erlangen-Nuremberg, Germany

#### Ni1.03

**Utero-placental cell interactions revealed by single cell transcriptomics**

*Mihaela Pavlicev, Ph.D., Cincinnati Children’s Hospital Medical Center, Cincinnati OH, USA

#### Ni1.04

**Dynamically Regulated Trophoblast Subpopulations Isolated from Early Human Placentas**

Frances Wong, B.Sc., University of Toronto, Canada

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**NOTE TO DELEGATES:** The code listed before the title of the presentation (e.g. KEY.01) in this program relates 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 Abstracts section of this booklet.
**Symposium 1**

Bioinformatics/systems biology applied to the placenta

**Moderators**
Yoel Sadovsky, M.D., Padma Murthi, Ph.D.

<table>
<thead>
<tr>
<th>Time</th>
<th>Room</th>
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<tbody>
<tr>
<td>10:30 a.m.</td>
<td>Portland Ballroom 251–258</td>
</tr>
</tbody>
</table>

<p>| Session    | Title                                         | Speaker                                                  | Institution and Location                        |
|------------|-----------------------------------------------|-----------------------------------------------------------|
| <strong>N1.05</strong>  | First trimester infection with <em>Listeria monocytogenes</em> is characterized by decidual and placental inflammation in pregnant cynomolgus macaques | K. Bryce Wolfe, B.S., University of Wisconsin – Madison WI, USA |
| <strong>N1.06</strong>  | Integrating multi-dimensional genomics data to build regulatory maps of placental development | <em>Geetu Tuteja, Ph.D.</em>, Iowa State University, Ames IA, USA |
| <strong>O1.01</strong>  | Genome scale analysis of mRNA and miRNA regulation in preterm birth | Nathan Price, Ph.D., Institute of Systems Biology, Seattle WA, USA |
| <strong>O1.02</strong>  | Mass Spectrometry-based Metabolomics: State-of-the-Art and the Future | Thomas Metz, Ph.D., Pacific Northwest National Laboratories, Richland WA, USA |
| <strong>O1.03</strong>  | Chromatin remodeling during formation of the syncytiotrophoblast | Andrew Adey, Ph.D., Oregon Health &amp; Science University, Portland OR, USA |</p>
<table>
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<tr>
<th><strong>WEDNESDAY SEPT. 14, 2016</strong></th>
<th><strong>TIME</strong></th>
<th><strong>ROOM</strong></th>
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</thead>
<tbody>
<tr>
<td>LUNCHTIME SESSION</td>
<td>12:30 p.m.</td>
<td>Portland Ballroom 251–258</td>
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</table>

**HPP**

**The Human Placenta Project: Current Progress and Future Directions**

David Weinberg, Ph.D., Eunice Kennedy Shriver National Institute of Child Health and Human Development, Bethesda MD, USA

**GABOR THAN AWARD PRESENTATION AND LECTURE**

1:30 p.m. Portland Ballroom 251–258

Moderators
Thomas Jansson, M.D., Ph.D.
Claire Roberts, Ph.D.

**THAN**

**Quo Vadis, trophoblast? Exploring the new ways of an old cell lineage**

Sascha Drewlo, Ph.D., Wayne State University, Detroit MI, USA

**POSTER SESSION 1**

2:30 p.m. Portland Ballroom Foyer

**REFRESHMENTS**

4 p.m. Portland Ballroom Foyer

**WORKSHOPS 1–4 (parallel sessions)**

4:30 – 6:30 p.m.

1. **Genomic communication**
   Yoel Sadovsky, M.D.
   Portland Ballroom 251–258

2. **New innovations in placental imaging**
   Antonio Frias, M.D., Victoria Roberts, Ph.D., John Sled, Ph.D.
   Portland Ballroom 252

3. **Linkage between placenta and development of other organs**
   Peta Grigsby, Ph.D.
   Portland Ballroom 253

4. **Decidua-trophoblast interactions**
   Gendie Lash, Ph.D.
   Portland Ballroom 254
<table>
<thead>
<tr>
<th>TIME</th>
<th>ROOM</th>
<th>SESSION 2</th>
<th>Moderators</th>
</tr>
</thead>
<tbody>
<tr>
<td>7:30 a.m.</td>
<td>E147-148</td>
<td>*<em>NEW INVESTIGATOR/<em>JUNIOR FACULTY SESSION 2</em></em></td>
<td>Cathy Vaillancourt Ph.D., Anthony Perkins Ph.D.</td>
</tr>
<tr>
<td>8:30 a.m.</td>
<td>Portland Ballroom 251–258</td>
<td>NI2.01</td>
<td>Glycolytic utilization and capacity of human cytotrophoblasts are higher than syncytiotrophoblasts</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Amy Valent, D.O., Oregon Health &amp; Science University, Portland OR, USA</td>
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<tr>
<td></td>
<td></td>
<td>NI2.02</td>
<td>Disruption of prolyl hydroxylase domain protein-2 (PHD2) activity impairs TGF-dependent sphingolipid metabolism in murine placenta</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Julien Sallais, M.Sc., Lunenfeld-Tanenbaum Research Institute, Toronto, Canada</td>
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<td></td>
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<td>NI2.03</td>
<td>Distinct molecular mechanisms regulate mammalian placental glucose and leucine transporter induced transport in response to varying degrees of maternal calorie restriction</td>
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<td>*Amit Ganguly, Ph.D., David Geffen School of Medicine at UCLA, Los Angeles CA, USA</td>
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<td></td>
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<td>NI2.04</td>
<td>Manipulation of TRKB activation alters cellular respiration in syncytiotrophoblasts</td>
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<td></td>
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<td>Calais Prince, B.S., University of Texas Health Science Center, San Antonio TX, USA</td>
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<td>NI2.05</td>
<td>Targeted nanoparticle delivery of a novel nitric oxide donor increased fetal weight in a mouse model of fetal growth restriction</td>
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<td>Natalie Cureton, BS.c., The University of Manchester, UK</td>
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<td>NI2.06</td>
<td>Vascular endothelial growth factor A administration rescues fetoplacental endothelial cell defects seen in severe fetal growth restriction</td>
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<td></td>
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<td>*Emily Su, M.D., University of Colorado School of Medicine, Denver CO, USA</td>
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<tr>
<td>10 a.m.</td>
<td>Portland Ballroom Foyer</td>
<td>REFRESHMENTS</td>
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</tbody>
</table>
**SYMPOSIUM 2**  
Trophoblast specification/progenitors

**Moderators:**  
Shawn Chavez, Ph.D., Louise Laurent M.D., Ph.D.

**TIME** | **ROOM**
---|---
10:30 a.m. | Portland Ballroom 251–258

**O2.01**

Trophoblast stem cells from murine fibroblasts – can the mouse serve as blueprint for the human situation?  
Hubert Schorle, Ph.D., University of Bonn, Germany

**O2.02**

Human trophoblast stem cells: real or not real?  
*Emily Su, M.D., University of Colorado School of Medicine, Denver CO, USA

**O2.03**

Controlling multi-stage trophoblast development: orchestration of progenitor and differentiated cell fate by transcription factors  
Soumen Paul, Ph.D., University of Kansas Medical Center, Kansas City KS, USA

**LUNCHTIME SESSION**  
Regional Placenta Associations Meetings

**TIME**

12:30 p.m.

**ROOM**

<p>| Regional Placenta Associations Meetings |<br />
|---|---|
| PAA | Portland Ballroom 251–258 |
| EPG | Portland Ballroom 252 |
| JPG | Portland Ballroom 253 |
| ANZPRA | Portland Ballroom 254 |</p>
<table>
<thead>
<tr>
<th><strong>THURSDAY SEPT. 15, 2016</strong></th>
<th><strong>TIME</strong></th>
<th><strong>ROOM</strong></th>
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<tbody>
<tr>
<td>ELSEVIER TROPHOBLAST RESEARCH AWARD</td>
<td>1:30 p.m.</td>
<td>Portland Ballroom 251–258</td>
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<tr>
<td>PRESENTATION AND LECTURE</td>
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<td>Moderators</td>
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<tr>
<td>Murray Mitchell, D.Phil., Alina Maloyan, Ph.D.</td>
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</table>

**ELSEVIER**

**Immune-modulatory effects of syncytiotrophoblast extracellular vesicles during normal and preeclamptic pregnancy**
Claudia Göhner, Ph.D., Jena University Hospital, Jena Germany and University Medical Center Groningen, The Netherlands

**POSTER SESSION 2**

2:30 p.m.
Portland Ballroom Foyer

**REFRESHMENTS**

4 p.m.
Portland Ballroom Foyer

**WORKSHOPS 5-8 (parallel sessions)**

4:30 p.m.
Portland Ballroom 251–258

5. **Inflammation – What is it and how does it affect the placenta?**
   Kent Thornburg, Ph.D., Murray Mitchell, D. Phil.

6. **Sexual Dimorphism in placental function**
   Alina Maloyan, Ph.D., Leslie Myatt, Ph.D., FRCOG
   Portland Ballroom 252

7. **Key mechanisms and novel insights into trophoblast implantation and invasion**
   Martin Knöfler, Ph.D., Alexander Beristain, Ph.D.
   Portland Ballroom 253

8. **Bioinformatics and omics applied to the placenta**
   Lucia Carbone, Ph.D.
   Portland Ballroom 254
<table>
<thead>
<tr>
<th>TIME</th>
<th>ROOM</th>
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<tbody>
<tr>
<td>7:30 a.m.</td>
<td>E147-148</td>
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<tr>
<td>8:30 a.m.</td>
<td>Portland Ballroom 251–258</td>
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**AWARDS COMMITTEE**

**NEW INVESTIGATOR//**JUNIOR FACULTY SESSION 3

**Moderators**
Vicki Clifton Ph.D., Yoshiki Kudo M.D.

<table>
<thead>
<tr>
<th>ID</th>
<th>Title</th>
<th>Presenter</th>
<th>Institution</th>
<th>Location</th>
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</thead>
<tbody>
<tr>
<td>N13.01</td>
<td>3D printed, bioengineered placenta model incorporating decellularized placental extracellular matrix and primary trophoblasts</td>
<td>Che-Ying Kuo, M.S., University of Maryland College Park, Children's National Medical Center, Washington DC, USA</td>
<td>Portland Ballroom 251–258</td>
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<tr>
<td>N13.02</td>
<td>A micro-engineered model of the human placental barrier</td>
<td>Cassidy Blundell, B.S., University of Pennsylvania, Philadelphia PA, USA</td>
<td>Portland Ballroom 251–258</td>
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<tr>
<td>N13.03</td>
<td>Characterization of exosomal miRNAs present in plasma from women with gestational diabetes mellitus</td>
<td>*Carlos Salomon, Ph.D., The University of Queensland, Brisbane, Australia</td>
<td>Portland Ballroom 251–258</td>
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<tr>
<td>N13.04</td>
<td>In vivo localization and vascular effects of nano-vesicles derived from normal first trimester human placentae</td>
<td>Mancy Tong, B.Sc., The University of Auckland, Auckland, New Zealand</td>
<td>Portland Ballroom 251–258</td>
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<tr>
<td>N13.05</td>
<td>A vascularised in vitro placental barrier model using primary human trophoblasts</td>
<td>Catherine Gilmore, B.Sc., University of Bristol, Bristol, UK</td>
<td>Portland Ballroom 251–258</td>
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<tr>
<td>N13.06</td>
<td>Placental metabolic flexibility is affected by maternal obesity</td>
<td>*Alina Maloyan, Ph.D., Oregon Health &amp; Science University, Portland OR, USA</td>
<td>Portland Ballroom 251–258</td>
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<td><strong>FRIDAY SEPT. 16, 2016</strong></td>
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<td><strong>REFRESHMENTS</strong></td>
<td>10 a.m.</td>
<td>Portland Ballroom Foyer</td>
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<tr>
<td><strong>ANDRÉE GRUSLIN AWARD PRESENTATION AND LECTURE</strong></td>
<td>10:30 a.m.</td>
<td>Portland Ballroom 251-258</td>
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<td>Moderators</td>
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<tr>
<td>Isabella Caniggia M.D., Ph.D.,</td>
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<td>Ceci Cheung, Ph.D.</td>
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<tr>
<td><strong>AGA</strong></td>
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<tr>
<td>Understanding the molecular underpinnings of distinct subclasses of preeclampsia</td>
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<td>Shannon Bainbridge, Ph.D, University of Ottawa, Canada</td>
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<tr>
<td><strong>IFPA SENIOR AWARD PRESENTATION AND LECTURE</strong></td>
<td>11:30 a.m.</td>
<td>Portland Ballroom 251-258</td>
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<td>Moderator</td>
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<td>Nick Illsley, D. Phil</td>
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<tr>
<td><strong>IFPA</strong></td>
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<tr>
<td>Placenta toxicology - susceptibility, assessment and contributions to reproductive and fetal/neonatal outcome</td>
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<tr>
<td>Richard K. Miller, Ph.D., University of Rochester, Rochester NY, USA</td>
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<tr>
<td><strong>LUNCHTIME SESSION</strong></td>
<td>12:30 p.m.</td>
<td>Portland Ballroom 251-258</td>
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<tr>
<td>IFPA Annual General Meeting</td>
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<tr>
<td>Final Poster Judging</td>
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<td><strong>FRIDAY SEPT. 16, 2016</strong></td>
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<td>SYMPOSIUM 3</td>
<td>1:30 p.m.</td>
<td>Portland Ballroom 251–258</td>
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<tr>
<td>Viral Infection and the Placenta</td>
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<td><strong>Moderators</strong></td>
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<tr>
<td>Larry Chamley, Ph.D., Peggy Petroff, Ph.D</td>
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<tr>
<td>O3.01</td>
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<tr>
<td>Immune protection against placental cytomegalovirus transmission in non-human primate models</td>
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<td>Sallie Permar, M.D., Ph.D., Duke University, Durham NC, USA</td>
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<tr>
<td>O3.02</td>
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<tr>
<td>Role of polymicrobial infection on viral-induced teratogenic effects</td>
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<td>Gil G. Mor, M.D., Ph.D., Yale University, New Haven CT, USA</td>
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<tr>
<td>Effect of Zika Virus during primate pregnancy</td>
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<td>Peta Grigsby, Ph.D., Antonio Frias, M.D., Daniel Streblow, Ph.D., Oregon Health &amp; Science University, Portland OR, USA</td>
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<tr>
<td>REFRESHMENTS</td>
<td>4 p.m.</td>
<td>Portland Ballroom Foyer</td>
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<td>WORKSHOPS 9–12 (parallel sessions)</td>
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<tr>
<td>9. Trophoblast biology and pathology</td>
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<tr>
<td>Shawn Chavez, Ph.D., Julie Baker, Ph.D.</td>
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<td>10. Transport NextGen: cool new stuff</td>
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<td>Nick Illsley, D.Phil.</td>
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<td>11. Immune cells at the maternal-fetal interface</td>
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<td>Peggy Petroff, Ph.D., Ted Golos, Ph.D.</td>
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<td>12. Trophoblast cell lines: their characteristics and limitations</td>
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<td>Graham Burton, Ph.D., Udo Markert, M.D.</td>
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<tr>
<td>GALA DINNER AND PRIZES</td>
<td>8 – midnight</td>
<td>Doubletree Hotel Lloyd Ballroom</td>
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## BUSINESS MEETING SCHEDULE

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<tr>
<th>TIME</th>
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<tr>
<td><strong>TUESDAY SEPT. 13, 2016</strong></td>
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<tr>
<td>8 a.m. – 12:30 p.m.</td>
<td>IFPA EXECUTIVE MEETING <em>(by invitation)</em></td>
<td>E147–148</td>
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<tr>
<td>12:30 – 1:30 p.m.</td>
<td>IFPA EXECUTIVE LUNCH <em>(by invitation)</em></td>
<td>E147–148</td>
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<td><strong>WEDNESDAY SEPT. 14, 2016</strong></td>
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<td>7:30 – 8:30 a.m.</td>
<td>AWARDS COMMITTEE</td>
<td>E147–148</td>
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<td><strong>THURSDAY SEPT. 15, 2016</strong></td>
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<tr>
<td>7:30 – 8:30 a.m.</td>
<td>PLACENTA/TROPHOBLAST RESEARCH</td>
<td>E147–148</td>
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<td>EDITORIAL BOARD MEETING</td>
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<tr>
<td>12:30 – 1:30 p.m.</td>
<td>REGIONAL PLACENTA ASSOCIATION MEETINGS</td>
<td>PAA, EPG, JPG, ANZPRA</td>
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<td><strong>FRIDAY SEPT. 16, 2016</strong></td>
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<tr>
<td>7:30 – 8:30 a.m.</td>
<td>AWARDS COMMITTEE</td>
<td>E147–148</td>
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<tr>
<td>12:30 – 1:30 p.m.</td>
<td>ANNUAL GENERAL MEETING</td>
<td>Portland Ballroom 251–258</td>
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<td>FINAL POSTER JUDGING</td>
<td>Portland Ballroom Foyer</td>
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WORKSHOP 1
Genomic communication

ORGANIZER
Yoel Sadovsky, M.D.
Magee Womens Research Institute, Pittsburgh PA, USA

While circulating RNAs, either bound by plasma membranes or packaged within extracellular vesicles (EVs), may transmit information to researchers about tissue function, disease, and organismal wellness, recent data indicate that these messages play a key role in local and distant cell communication. Using a series of targeted and provocative exchanges, this workshop will center on the transfer of RNAs within EVs and their entry into target cells, the use of minimally invasive, circulating RNA biomarkers for disease monitoring, and the integration of these data into longitudinal assessment of pregnancy health, projecting a futuristic view of “scientific wellness”.

SPEAKERS
Larry Chamley, Ph.D.
University of Auckland, New Zealand

Peter Kurre, Ph.D.
Oregon Health & Science University, Portland OR, USA

Nathan Price, Ph.D.
Institute for Systems Biology, Seattle WA, USA

Yoel Sadovsky, M.D.
Magee-Womens Research Institute, Pittsburgh PA, USA

Alison Paquette, Ph.D.
Institute for Systems Biology, Seattle WA, USA

Carlos Salomon, Ph.D.
University of Queensland, Australia
WORKSHOPS

WORKSHOP 2
New innovations in placental imaging; focus on practical aspects of in vivo data acquisition and analysis

ORGANIZERS

Antonio Frias, M.D., Victoria Roberts, Ph.D.
Oregon Health & Science University, Portland OR, USA

John Sled, Ph.D.
University of Toronto, Canada

SPEAKERS

Matthias Schabel, Ph.D.
Oregon Health & Science University, Portland OR, USA

Sally Collins, M.D.
University of Oxford, UK

John Sled, Ph.D.
University of Toronto, Canada

Dinesh Shah, M.D.
University of Wisconsin, Madison WI, USA
The intimate linkage between placental size and function and overall fetal growth and development has been recognized for a long time. Similarly the linkage between placental function and development of certain organs, e.g. heart, liver, pancreas, kidney has been increasingly recognized. More recently the mechanisms operating at the physical and molecular levels linking placental development and function to development and function of fetal organs are beginning to be elucidated, particularly in relation to fetal programming of adult disease. In addition development of other fetal organs, e.g. brain are being shown to be regulated by placental factors such as serotonin. This workshop will feature a series of presentations describing the mechanistic linkages between placental function and development of fetal organs in the normal and pathologic settings, the role for these mechanisms in fetal programming and potential therapeutic approaches.

SPEAKERS

Helen Jones, Ph.D.
Cincinnati Children's Hospital Medical Center, USA

Carolyn Salafia, M.D.
Placental Analytics, NY, USA

Cathy Vaillancourt, Ph.D.
INRS-Institut Armand Frappier, Canada
WORKSHOP 4
Decidua- trophoblast Interactions

ORGANIZER

Gendie Lash, Ph.D.
Guangzhou Institute of Pediatrics, Guangzhou Women and Children's Medical Center, Guangzhou, China

Invasion of the uterine decidua and inner third of the myometrium by extravillous trophoblast cells (EVT) and the resultant remodeling of the spiral arteries are key to the establishment of a healthy successful pregnancy. During these processes EVT interact with different decidual cell types including decidual stromal cells, uterine natural killer cells, uterine macrophages, different T cell populations, vascular smooth muscle cells, endothelial cells and myometrial stromal cells. These interactions have the potential to alter the phenotype, and therefore function, of either interacting cell type. The aim of this workshop is to explore some of these interactions and discuss how they contribute to the establishment of a healthy pregnancy.

SPEAKERS

Potential mechanisms of communication between decidual cell populations and trophoblast
Peggy Petroff, Ph.D.
Michigan State University, Flint MI, USA

Consequences of decidual leucocyte and EVT communication
Gendie Lash, Ph.D.
Guangzhou Institute of Pediatrics, China

Multinuclear trophoblast giant cells: evidence of ongoing terminal differentiation of extravillous trophoblast to term
Christina Duzyj, Ph.D.
Rutgers University, New Brunswick NJ, USA

EVT genotype/phenotype disconnect in abnormally invasive placenta: necessity for decidual interactions in over invasion
Sonia DaSilva-Arnold, Ph.D.,
Hackensack University Medical Center, Hackensack NJ, USA
Inflammation or mediators of inflammation play important roles in many physiological and pathological mechanisms in reproductive biology. The relationships between pathogen-driven inflammation vs. non-infectious inflammation or more simply, “hot” versus “cold” inflammation are complex. The different intracellular pathways that are activated in chronic versus acute processes are important and not understood even though they should result in different approaches to therapy. In this workshop we aim to approach the issues head on in this critical area of reproductive biology.

SPEAKERS

Kent Thornburg, Ph.D.
Oregon Health & Science University, Portland OR, USA

Murray Mitchell, D.Phil.
University of Queensland, Brisbane Australia

David Olson, Ph.D.
University of Alberta, Canada

Pepper Schedin, Ph.D.
Oregon Health & Science University, Portland OR, USA
WORKSHOPS

WORKSHOP 6
Sexual dimorphism in placental function

ORGANIZERS

Leslie Myatt, Ph.D.
Oregon Health & Science University, Portland OR, USA

Alina Maloyan, Ph.D.
Oregon Health & Science University, Portland OR, USA

Sexual dimorphism in pregnancy outcomes has been described for many years with the male fetus being at increased risk for adverse outcomes particularly those related to prematurity. There is now increasing awareness of sexual dimorphism at the level of placental function with well described differences in gene expression and in inflammatory responses being seen. However the molecular basis for sexual dimorphism in placental function is poorly understood. The aim of this workshop is to discuss findings from recent studies and to outline potential mechanisms underlying the sexual dimorphism

SPEAKERS

Sexual dimorphism in placenta and pregnancy outcomes
Leslie Myatt, Ph.D.
Oregon Health & Science University, Portland OR, USA

Mechanisms operating at the Genome Level
Wendy P. Robinson, Ph.D.
UBC Dept. of Medical Genetics, Child & Family Research Institute, University of British Columbia, Canada

Teasing out the complexity of sex differences in placental GR
Vicki Clifton, Ph.D.
Mater Research and Translational Research Institute, Brisbane, Australia

The battleground of viruses, plastics and hormones: unified thinking on sex-specific placental mediation of teratogenic and endocrine disrupting effects
Jennifer Adibi, M.P.H., Sc.D.
Department of Epidemiology, Department of Obstetrics, Gynecology and Reproductive Sciences, University of Pittsburgh, Pittsburgh PA, USA
Trophoblast differentiation along the invasive pathway is fundamental to early implantation, placental development and establishment of the fetal-maternal interface. Multiple gene pathways and heterogeneous cellular interactions have been described in directing and controlling trophoblast invasion. The focus of this workshop aims to address well accepted as well as controversial paradigms central to the intrinsic control of trophoblast invasion. Novel cellular mechanisms with respect to implantation, development of an early invasive trophoblast lineage, and new insights into the versatile functions of invasive trophoblasts will be discussed. The importance of key transcription factors, the cell cycle, and aging as well as the role of polyploidy and ADAM proteases in differentiating extravillous trophoblast populations will be a primary focus. Moreover, the role of external environmental stimuli, such as hypoxia and mechanisms regulating uterine leukocyte cross-talk will be explored.

SPEAKERS

Introduction
M. Knöfler / A. Beristain

Development of the invasive trophoblast phenotype in the early stages of implantation
John D. Aplin, Ph.D.
Institute of Human Development, University of Manchester, UK

Plasticity in development of the invasive trophoblast lineage
Michael J. Soares, Ph.D.
Institute of Health and Regenerative Medicine, University of Kansas, USA

Defining the versatile functions of invasive human trophoblasts
Jürgen Pollheimer, Ph.D.
Department of Obstetrics and Gynecology, Medical University of Vienna, Austria

ADAM proteases in trophoblast differentiation: Establishing protective mechanisms in hypoxia?
Alexander Beristain, Ph.D.
Department of Obstetrics and Gynecology, University of British Columbia, Canada

Transcription factors and SNPs affecting trophoblast-leukocyte interactions
Caroline Dunk, Ph.D.
Research Centre for Women’s and Infants’ Health, The Samuel Lunenfeld Research Institute, Toronto, Canada

General discussion and concluding remarks
M. Knöfler / A. Beristain
WORKSHOP 8
Bioinformatics and omics applied to the placenta

ORGANIZER

Lucia Carbone, Ph.D.
Oregon Health & Science University, Portland OR, USA

Omic technologies and associated bioinformatics tools have greatly and rapidly evolved in the last few years. These advances have significantly impacted the placenta field as such datasets offer the potential for identifying characteristic attributes reflecting placenta health and/or possible abnormalities. One of the ultimate goals of performing omics analyses is the identification of biomarkers that inform about the status of the placenta, the mother and the baby. However, the bioinformatics analysis and integration of such datasets is faced with many issues. First, references for placenta transcriptomes, metabolomics, and epigenomes are missing, hindering the interpretation and integration of omics data. Moreover, the range of variability within the population is still unknown; hence a baseline to evaluate adverse profiles is missing. During this workshop scientists involved in the analyses of different types of omics data (e.g. epigenomes, microRNAomes and metabolomes), will elaborate on the current methods used to obtain and analyze omics data and strategies used to deal with the issues raised above. During this workshop we aim to raise and start addressing some of the provocative questions that are raised in the placenta field when omics data are generated and analyzed.

SPEAKERS

Geetu Tuteja, Ph.D.
Iowa State University, Ames IA, USA

Priyadarshini Pantham, Ph.D.
University of Illinois, Urbana-Champaign IL, USA

Katie Powell, Ph.D.
University of Sydney, Australia

Diana Morales-Prieto, Ph.D.
University of Jena, Germany

Samantha Wilson, BS.c.
University of British Columbia, Canada
WORKSHOP 9
Trophoblast biology and pathology

ORGANIZERS

Shawn Chavez, Ph.D.
Oregon Health & Science University, Portland OR, USA

Julie Baker, Ph.D.
Stanford University, Stanford CA, USA

Normal placental development is largely dependent upon the differentiation and invasion of the trophoblast, which originates from the trophectoderm of the blastocyst prior to embryo implantation. Given that aberrant trophoblast development is a common phenomenon observed in pregnancy complications such as preterm labor, preeclampsia, and intrauterine growth retardation, much research emphasis has been placed on the genetic, epigenetic, and chromosomal aspects regulating trophoblast function. Recent technological advances in genome-wide DNA methylation analysis, next generation sequencing (NGS), and live-cell imaging, as well as the use of human pluripotent stem cells to assess trophoblast regulation, has provided considerable insight into normal placental development and the pathophysiology of these pregnancy-related diseases.

The overall objectives of this workshop are to discuss the NGS, imaging, and other emerging approaches for assessing trophoblast competency at the single-cell and/or whole-genome level. We will also discuss the key trophoblast regulators, including endogenous retroviruses, and intracellular signaling pathways mediating trophoblast fate that have been identified as important for normal placental function.

SPEAKERS

Louise Laurent, M.D., Ph.D.
University of California, San Diego CA, USA

Balaji Rao, Ph.D.
North Carolina State University, Raleigh NC, USA

Julie Baker, Ph.D.
Stanford University, Stanford CA, USA

Shawn Chavez, Ph.D.
Oregon Health & Science University, Portland OR, USA
WORKSHOPS

WORKSHOP 10
Transport NextGen: cool new stuff

ORGANIZER

Nick Illsley, Ph.D.
Hackensack University Medical Center, Hackensack NJ, USA

This workshop will look at several new technologies that are becoming available for research into placental transport and will provide new opportunities for investigation. These will be presented as 30 min talks including time for questions and discussion.

SPEAKERS

Prospects for non-invasive measurement of placental metabolic and transport processes with Hyperpolarized MRI
Charles McKenzie, Ph.D.
Department of Medical Biophysics, University of Western Ontario, Canada

Primary trophoblast Transwell model mimicking the function of chorionic villi
Christiane Albrecht, Ph.D.
Institute of Biochemistry & Molecular Medicine, University of Bern, Switzerland

Engineering diffusion of chemoattractants in bioprinted placental tissue models to measure migration
Che-Ying Kuo, Ph.D.
Department of Bioengineering, University of Maryland, USA

Leveraging organ-on-a-chip technology to study the human placental barrier
Cassidy Blundell, Ph.D.
School of Engineering and Applied Science, University of Pennsylvania, Philadelphia PA, USA
Immune cells at the maternal-fetal interface can serve multiple purposes. On one hand, they play an important role in placental development and uterine remodeling; on the other, they participate in defense against pathogens and prevent vertical transmission. Yet a third effect is the ‘bystander’ damage that occurs as a result of inflammation when infection or other stressors are present. In this workshop we will discuss these seeming Janus-faced roles of immune and other cells, and immune-mediated processes that occur in normal pregnancy outcomes.

Questions to be addressed will include:

- When does the balance shift from normal to abnormal immune function?
- Who are the players in adverse, immune-mediated placental dysfunction – resident, in-fluxing, and/or peripheral immune cells? Are the immune cells that are critical in normal placental development and function the same as those involved in host defense against pathogens?
- What are the different roles of resident, influxing and peripheral immune cells in normal placental development and function, and in the host response to infection?
- Can the decidua and/or chorionic villi safely accommodate an inflammatory response to infection?
- What is the role of the trophoblast in the host response to infection?

Speakers

- Gendie Lash, Ph.D.
  Guangzhou Women and Children’s Medical Center, Guangdong, China

- Caroline Dunk, Ph.D.
  Lumenfeld-Tanenbaum Research Institute, Mount Sinai Hospital, Toronto, Canada

- Ann-Charlotte Iversen, Ph.D.
  Norwegian University of Science and Technology, Trondheim, Norway

- Ulrike Kemmerling, Ph.D.
  University of Chile, Santiago, Chile

- Jennifer Stencel-Baerenwald, Ph.D.
  University of Washington, Seattle WA, USA

- Ted Golos, Ph.D.
  University of Wisconsin – Madison WI, USA
WORKSHOP 12

Trophoblast cell lines: their characteristics and limitations.

ORGANIZERS

Graham J. Burton, Ph.D.
University of Cambridge

Udo Markert, M.D.
University of Jena, Germany

In the absence of human trophoblast stem cells various trophoblast-like cell lines have been developed, some choriocarcinoma and some transformed. These are widely used in placental research, but how accurately do they reflect the real tissue? As analyses become more sophisticated the limitations of the different cell lines are becoming increasingly evident. This workshop will review evidence from a number of different approaches, including karyotype, gene expression, epigenetics and HLA antigens. The principal aim is to generate a panel of markers that can be used to characterize the various cell lines, and to consider what supporting data should be included in any publication in Placenta reporting data based on their usage.

SPEAKERS

Maja Weber, Ph.D.
University of Jena, Germany

Cheryl Lee, Ph.D.
University of Cambridge, UK

Diana Morales Prieto, Ph.D.
University of Jena, Germany

Padma Murthi, Ph.D.
Monash University, Australia

Jürgen Pollheimer, Ph.D.
University of Vienna, Austria

Georges Daoud, Ph.D.
American University of Beirut, Lebanon
POSTER SESSION 1

WEDNESDAY SEPT. 14, 2016

ANGIOGENESIS/ VASCULATURE

P1.1  Involvement of inverted formin-2 (INF2) in trophoblast invasion and birth timing
Katherine Bezold Lamm\textsuperscript{1,2}, Emily Holloway\textsuperscript{1}, Jude McElroy\textsuperscript{1}, Helen Jones\textsuperscript{1}, Louis Muglia\textsuperscript{1}, \textit{Cincinnati Children’s Hospital Medical Center, Cincinnati, OH, USA, University of Cincinnati, Cincinnati, OH, USA, Vanderbilt University, Nashville, TN, USA}

P1.2  Angiogenic factors are associated with placental weight and birth weight in Bangladeshi pregnancies
Alison Gernand\textsuperscript{1}, Abdullah Mahmud\textsuperscript{2}, Eszter Papp\textsuperscript{3}, Joy Shi\textsuperscript{3}, Daniel Roth\textsuperscript{3}, \textit{The Pennsylvania State University, University Park, PA, USA, International Center for Diarrhoeal Disease Research, Bangladesh, Dhaka, Bangladesh, Hospital for Sick Children, Toronto, ON, Canada}

P1.3  Role of miR-141 in angiogenesis and communication between trophoblast and endothelial cells via extracellular vesicles with emphasis on preeclampsia
Ruby N. Gutiérrez-Samudio\textsuperscript{1}, Yvonne Heimann\textsuperscript{1}, Udo R. Markert\textsuperscript{1}, Diana M. Morales-Prieto\textsuperscript{1}, \textit{University Hospital Jena, Jena, Thuringia, Germany}

P1.4  Exosomes derived maternal and umbilical cord blood differentially regulated endothelial cell migration through exosomal miRNAs
Linyan Jia\textsuperscript{1}, Julei Yao\textsuperscript{1}, Hao Ying\textsuperscript{1}, Tao Duan\textsuperscript{1}, Kai Wang\textsuperscript{1}, Shanghai First Maternity and Infant Hospital, Shanghai, China

P1.5  The role of stanniocalcin-1 in spiral artery remodelling
Arwa Mazen\textsuperscript{1}, Sandra Ashton\textsuperscript{1}, Judith Cartwright\textsuperscript{1}, Guy Whitley\textsuperscript{1}, \textit{St George's University of London, London, UK}

P1.6  The human placenta: function and morphological assessments using ultrasound and magnetic resonance elastography
Richard K Miller\textsuperscript{1}, Stephan McAleavey\textsuperscript{2}, Juvenal Ormachea\textsuperscript{2}, Eva Pressman\textsuperscript{1}, Loralei Thornburg\textsuperscript{1}, David Dombroski\textsuperscript{1}, Jianhui Zhong\textsuperscript{1}, Marvin Doyley\textsuperscript{2}, Ronald W Wood\textsuperscript{1}, Jonathan Carroll-Nellenback\textsuperscript{2}, Ollivier Hyrien\textsuperscript{1}, Philip Katzman\textsuperscript{1}, Christopher Stodgell\textsuperscript{1}, Kam Szlachetka\textsuperscript{1}, Henry Wang\textsuperscript{1}, Kevin Parker\textsuperscript{1}, \textit{University of Rochester School of Medicine and Dentistry, Rochester, New York, USA, University of Rochester Hajim School of Engineering, Rochester, New York, USA}

P1.7  Arterial chorionic surface vessel branch point density and autism risk.
Simon Morgan\textsuperscript{1}, Dimitri Vvedensky\textsuperscript{1}, Ruchit Shah\textsuperscript{2}, Theresa Girardi\textsuperscript{2}, Craig Newschaffer\textsuperscript{2}, Philip Katzman\textsuperscript{4,5}, Richard Miller\textsuperscript{4,5}, John Moye\textsuperscript{5,6}, Carolyn Salafia\textsuperscript{2,3}, \textit{Imperial College, London, UK, Placental Analytics, LLC, New Rochelle, NY, USA, Drexel University, Philadelphia, PA, USA, University of Rochester, Rochester, NY, USA, Placental Consortium, Bethesda, MD, USA, NICHD, Bethesda, MD, USA}
P1.8 Familial high ASD risk differs from a low risk cohort by longer segment lengths and potential reduced branching in early but not later branch generations.
David Schubert1, Alexander Leonard1, Jonathan Lee2, Simon Morgan1,2, Dimitri Vvedensky1, Craig Newschaffer4, Ruchit Shah2, Richard Miller3,6, Philip Katzman3,6, John Moye1, Carolyn Salafia2,6, 1Imperial College, London, UK, 2Placental Analytics LLC, New Rochelle, NY, USA, 3University of Rochester, Rochester, NY, USA, 4National Institute of Child Health and Development, Bethesda, MD, USA, 5Drexel University, Philadelphia, PA, USA, 6Placental Consortium, Bethesda, MD, USA

P1.9 Whole chorionic surface vessel feature analysis with the Boruta method, and autism risk.
Jen–Mei Chang1, Ya-Mei Chang2, Ruxu Han1, Hui Zeng1, Ruchit Shah3, Craig Newschaffer4, Richard Miller5,6, Philip Katzman1,2, John Moye1, Carolyn Salafia3, 1California State University, Long Beach, CA, USA, 2Tamkang University, Taiwan, Taiwan, 3Placental Analytics, LLC, New Rochelle, NY, USA, 4Drexel University, Philadelphia, OPA, USA, 5University of Rochester, Rochester, NY, USA, 6Placental Consortium, Bethesda, MD, USA, 7NICHD, Bethesda, MD, USA

P1.10 A priori specified relationships among arterial chorionic surface vessel networks (PCSVNs) and autism risk.
Ruchit Shah1, Theresa Girardi1, Jen-mei Chang2, Jennifer Straughen3, Craig Newschaffer4, Dawn Misra4, Philip Katzman6,7, Richard Miller6,7, John Moye7, Carolyn Salafia1,2, 1Placental Analytics, LLC, New Rochelle, NYU, USA, 2California State, Long Beach, Long Beach, CA, USA, 3Henry Ford Health Systems, Detroit, MI, USA, 4Wayne State University, Detroit, MI, USA, 5Drexel University, Philadelphia, PA, USA, 6University of Rochester, Rochester, NY, USA, 7Placental Consortium, Bethesda, MD, USA, 8NICHD, Bethesda, MD, USA

P1.11 Effects of placental terminal villous capillarisation on vascular wall shear stress and placental angiogenesis
Win Min Tun1,2, Joanna James2, Alys Clark1, 1Auckland Bioengineering Institute, University of Auckland, Auckland, New Zealand, 2Faculty of Medical and Health Sciences, University of Auckland, Auckland, New Zealand

P1.12 Trophoblasts stimulate the release of MMP10 by endothelial cells.
Nora Lagzouli1, Ben Sayer1, Sandra Ashton1, Judith Cartwright1, Guy Whitley1, 1St George’s University of London, London, UK

P1.13 Three dimensional modelling of haemodynamics in a rat feto-placental arterial network.
Tim Crough1, Lachlan Kelsey1, Barry Doyle1, Caitlin Wyrwoll1, 1The University of Western Australia, Perth, Australia

BIOINFORMATICS

P1.14 Characterization of global human microRNA expression patterns across fetal plasma, placenta, and maternal plasma
Alison Paquette1, Tianjiao Chu2, Xiagong Wu1, Kai Wang1, Cory Funk1, Yoel Sadovsky2, Nathan Price6, 1Institute for Systems Biology, Seattle, Washington, USA, 2Magee Womens Research Institute, Pittsburgh, Pennsylvania, USA
CELL SIGNALING

P1.15 Unravelling IGF-I signalling in villous trophoblast
Magda Karolczak-Bayatti¹, James Horn¹, Lynda Harris¹, Melissa Westwood¹, John Aplin¹, ¹University of Manchester, Manchester, UK

P1.16 Autocrine effect of BMP4 in trophoblast cells
Francesca Soncin¹, Anna Wakeland¹, Kanaga Arul-Nambi-Rajan¹, Matteo Moretto-Zita¹, Katharine Nelson¹, Mana Parast¹, ¹University of California - San Diego, San Diego, California, USA

P1.17 The crucial role of AKAP95 in the induction of COX-2 by cortisol in human amnion fibroblasts
Jiangwen Lu¹, Wangsheng Wang¹, Kang Sun¹, ¹Center for Reproductive Medicine, Renji Hospital, School of Medicine, Shanghai Jiao Tong University, Shanghai, China

COMPARATIVE/ANIMAL MODEL

P1.18 AMPK alpha placental-specific knockdown results in altered labyrinthine and trophoblast giant cell development.
Renee Albers¹, Melissa Kaufman¹, Chanel Keoni¹, Guarav Kaushik², Bryony Natale², David Natale², Thomas Brown¹, ¹Wright State University, Dayton, Ohio, USA, ²University of California San Diego, San Diego, California, USA

P1.19 Transplacental tranfer of nanoparticles inhaled due to maternal exposure to filtered diesel engine exhaust during pregnancy in a rabbit model
Josiane Aioun¹-², Anne Tarrade¹-², Sarah Valentino¹-², Marie-Christine Aubrière¹-², Delphine Rousseau-Ralliard¹-², Michèle Dahirel¹-², Eve Mourier¹-², Christophe Richard¹-², Sylvaine Camous¹-², Marie-Sylvie Lallemand¹-², Marine Guinot¹-², John A. Boere³, Paul H. Fokkens³, Rémy Slama³, Flemming R. Cassee³, Pascale Chavatte-Palmer¹-², ¹UMR 1198 Biologie du Développement et Reproduction, INRA, ENVA, Université Paris Saclay DR, INRA, ENVA, Université Paris-Saclay-Saclayology du, Jouy-en-Josas, France, ²PremUp Foundation, Paris, France, ³National Institute for Public Health and the Environment, Center for Sustainability, Environmemnt and Health, BA Bilthoven, The Netherlands, ³Inserm and University Grenoble Alpes, U823, IAB Reserch Center Team of Environmental Epidemiology Applied to Reproduction and Respiratory Health, Grenoble, France

P1.20 Mechanotransduction drives morphogenesis to develop folding at the uterine-placental interface of pigs
Greg Johnson¹, Heewon Seo², Fuller Bazer³, Robert Burghardt¹, Kayla Bayless³, ¹Department of Veterinary Integrative Biosciences, Texas A&M University, TX, USA, ²Department of Animal Science, Texas A&M University, TX, USA, ³Department of Molecular & Cellular Medicine, Texas A&M System Health Science Center, TX, USA

P1.21 A Novel mouse model of Hypoplastic Left Heart Syndrome recapitulates placental and fetal growth anomalies found in human cases
Kathryn Owens¹, Weston Troja¹, Helen Jones¹, ¹cincinnati Children's Hospital Medical Center, Cincinnati, Ohio, USA
**POSTER SESSIONS**

**P1.22** The impact of antenatal uric acid serum levels on murine neonatal development  
Camilla Marini1,2,4, Benjamin P. Lüscher1,3, Philipp Schneider1, Christiane Albrecht1, Matthias A. Hediger3, Daniel V. Surbek1,2, Marc U. Baumann1,2,1, Department of Obstetrics and Gynaecology, University Hospital of Bern, Bern, Switzerland, Switzerland, 2Department of Clinical Research, University of Bern, Bern, Switzerland, Switzerland, 3Institute of Biochemistry and Molecular Medicine, University of Bern, Bern, Switzerland, Switzerland, 4Graduate School for cellular and Biomedical Sciences (GCB), University of Bern, Bern, Switzerland, Switzerland

**P1.23** Metabolic hormone dynamics of the marmoset monkey pregnancy  
Julienne Rutherford1, Suzette Tardif2, Corinna Ross2, Laren Riesche1, Victoria deMartelly1, Aubrey Sills4, Layne Colon Donna1, Toni Ziegler2, University of Illinois at Chicago, Chicago, IL, USA, 2Texas Biomedical Research Institute, San Antonio, TX, USA, 3University of Wisconsin, Madison, WI, USA, 4Barshop Institute, University of Texas Health Science Center San Antonio, San Antonio, TX, USA, 5Texas A&M University San Antonio, San Antonio, TX, USA

**P1.24** Zinc deficiency alters placental development and maternal hemodynamics contributing to fetal growth restriction in mice  
Rebecca Wilson1,2, Shalem Leemaqz1,2, Zona Goh1,2, Dale McAninch1,2, Tanja Jankovic-Karasoulos1,2, Tina Bianco-Miott1,2, Claire Roberts1,2, Robinson Research Institute, Adelaide, SA, Australia, 3University of Adelaide, Adelaide, SA, Australia

**GENE EXPRESSION**

**P1.25** Loss of function of the Polycomb repressor complex (PRC1.1) protein, BCOR, in the placenta results in disruption of trophectoderm derived cell fates, IUGR and death  
Vivian Bardwell1, Connie Corcoran1, Micah Gearhart1, Teng Zhang1, University of Minnesota, Minneapolis, MN, USA

**P1.26** MicroRNA-519d in trophoblastic cell functions and intercellular communication with immune cell via extracellular vesicles  
Wittaya Chaiwangyen1,2, Diana M. Morales-Prieto1, Ruby N. Gutierrez-Samudio1, Stephanie Ospina-Prieto1, Stella M. Photini1, Ekkehard Schleussner1, Udo R. Markert1, Placenta Lab, Department of Obstetrics, University Hospital Jena, Jena, Germany, 2Department of Biochemistry, School of Medical Sciences, University of Phayao, Phayao, Thailand

**P1.27** PIM Kinases in Trophoblast cells  
Stella M. Photini1, Wittaya Chaiwangyen1,2, Boodor Al-kawlani1, Diana M. Morales-Prieto1, Ekkehard Schleussner1, Udo R. Markert1, Placenta-Lab, Department of Obstetrics, University hospital Jena, Germany, Jena, Germany, 2Department of Biochemistry, School of Medical Sciences, University of Phayao, Phayao, Thailand

**P1.28** Deletion of retroviral element THE1B in humanized mice abolishes placental expression of human corticotropin-releasing hormone  
Caitlin Dunn-Fletcher1, Lisa Muglia1, Louis Muglia1, Cincinnati Children’s Hospital Medical Center, Cincinnati, Ohio, USA
P1.29  Alterations in epithelial sodium channels (ENaC α, β, γ, δ) and its regulator pendrin in response to sodium chloride in BeWo and JEG3 cell lines
Rahel Klossner¹, Paula Williams², Nicole Eisele¹, Markus Mohaupt¹, Hiten Mistry², ¹University of Bern, Bern, Switzerland, ²University of Nottingham, Nottingham, UK

P1.30  Placental expression of epithelial sodium channels and NEDD4-2 throughout gestation in human pregnancy
Paula Williams¹, Lesia Kurlak¹, Jorn Mohaupt¹, Guy Whitley², Judith Cartwright³, Markus Mohaupt¹, Hiten Mistry¹, ¹University of Nottingham, Nottingham, UK, ²St. George’s University of London, London, UK, ³University of Bern, Berne, UK

P1.31  Expression and functional analysis of C19MC microRNAs in normal and preeclamptic pregnancies
Daniel Fabig¹, Ivan Dimitrov¹, Udo R. Markert¹, Diana M. Morales-Prieto¹, ¹University Hospital Jena, Jena, Thuringia, Germany

P1.32  Discerning the link in endogenous retroviral type-K (ERV-K) expression between pre-implantation embryo development and placentation in primates.
Jimi Rosenkrantz¹,3, Brittany Daughtry²,3, Shawn Chavez³,4, ¹Department of Molecular & Medical Genetics; Graduate Program in Molecular & Cellular Biosciences; Oregon Health & Science University (OHSU) School of Medicine, Portland, Oregon 97239, USA, ²Department of Cell, Developmental & Cancer Biology; Graduate Program in Molecular & Cellular Biosciences; Oregon Health & Science University (OHSU) School of Medicine, Portland, Oregon 97239, USA, ³Division of Reproductive & Developmental Sciences; Oregon National Primate Research Center (ONPRC), Beaverton, Oregon 97006, USA, 4Departments of Obstetrics & Gynecology and Physiology & Pharmacology, OHSU School of Medicine, Portland, Oregon 97239, USA

P1.33  Regulation of SATB homeobox 1 gene expression in trophoblast stem cells
Wei Yu¹, Geetu Tuteja², Yan Hong¹, Anamika Ratri¹, Shaon Borosha¹, Michael Wolfe¹, Mohammad Rumi¹, ¹University of Kansas Medical Center, Kansas City, KS, USA, ²Iowa State University, Ames, IA, USA

P1.34  Interaction of SATB homeobox proteins in trophoblast-specific gene regulation
Wei Yu¹, Anamika Ratri¹, Yan Hong¹, Shaon Borosha¹, Michael Wolfe¹, Mohammad Rumi¹, ¹University of Kansas Medical Center, Kansas City, KS, USA

P1.35  Placental-specific extracellular vesicle sorting by high-resolution flow cytometry
Terry Morgan¹, Philip Streeter¹, Kevin Judge², ¹OHSU, Portland, OR, USA, ²BD Biosciences, San Jose, CA, USA

P1.36  Shear wave absolute vibro-elastography of the placenta
Jeffrey Abeysekera², Manyou Ma², Mehran Pesteie², Jefferson Terry¹, Denise Pugash¹, Jennifer Hutcheon¹, Chantal Mayer¹, Septimiu Salcudean², Lutz Lampe², Robert Rohling², ¹Children’s & Women’s Health Centre of BC, Vancouver, BC, Canada, ²University of British Columbia, Vancouver, BC, Canada
POSTER SESSIONS

P1.37 Magnetic resonance imaging and ultrasound fusion imaging for the evaluation of placental invasion
Hiroaki Aoki1, Osamu Samura1, Taisuke Sato1, Akiko Konishi1, Momoko Inoue1, Yuki Ito1, Taizan Kamide1, Tomohiro Tanemoto1, Satomi Kitai1, Aikou Okamoto1, ‘The Jikei University school of medicine, Tokyo, Japan

P1.38 High resolution three dimensional imaging of placental villi using serial block face scanning electron microscopy
Eleni Palaiologou1, Wendy Chiu1, Rodolfo Ribeiro de Souza1, Patricia Goggin2, Emma Lofthouse1, Jane Cleal1, Anton Page1, Rohan Lewis1, ‘University of Southampton, Faculty of Medicine, Southampton, UK, ‘University of Southampton, Biomedical Imaging Unit, Southampton, UK

P1.39 Evaluation of the thermal map of human placenta as the first step to in vitro application of infrared thermometry
Jonathan Lugo1, Natalia Schlabritz-Loutsevitch1, Alan John1, James Maher1, ‘Texas Tech University: Permian Basin, Odessa, TX, USA

P1.40 Magnetic Resonance Imaging of Utero-Placental Vascular Flow and Tissue Perfusion in Pregnant Rhesus Macaques
Jacob Macdonald1, Sydney Skopos1, Kevin Johnson1, Kai Ludwig1, Sean Fein1, Kevin Reeder1, Dinesh Shah1, Oliver Wieben1, Thaddeus Golos1, ‘University of Wisconsin - Madison, Madison, Wisconsin, USA

P1.41 An Image Processing Technique for the Visualization and Quantification of Blood Flow Entering the Placenta using 3D power Doppler Ultrasound (PD-US)

P1.42 Obesity affects KIR expression in decidual natural killer cells (dNK)
Barbara Castellana1,2, Sofie Perdu1, Yoona Kim1, Alex G. Beristain1,2, ‘The Child & Family Research Institute, Vancouver/British Columbia, Canada, ‘Department of Obstetrics & Gynecology, The University of British Columbia, Vancouver/British Columbia, Canada

IMMUNOLOGY

P1.43 Trophoblast turnover in human placental chorionic villi explants induced by Trypanosoma cruzi is mediated by Toll like receptor-2 activation.
Christian Castillo1, Lorena Muñoz1, Ileana Carrillo1, Daniel Droguett1, Ana Liempi1, Javier Astudillo1, Juan Diego Maya1, Norbel Galanti1, Ulrike Kemmerling1, ‘Faculty Of Medicine, University of Chile, Santiago, Chile, Chile

P1.44 The ex vivo infection of human placental chorionic villi explants with Trypanosoma cruzi and Toxoplasma gondii is mediated by different Toll-like receptors
Lorena Muñoz1, Christian Castillo1, Ileana Carrillo1, Andrea Salinas1, Ana Liempi1, Daniel Droguett1, Juan Diego Maya1, Norbel Galanti1, Ulrike Kemmerling1, ‘Faculty Of Medicine, University of Chile, Santiago, Chile, Chile
P1.45 Shared immunoregulatory properties of galectin-9 and PD-L1 in pregnant women and patients with stage IV melanoma
Elizabeth Ann Enninga1, Wendy Nevala1, Douglas Creedon1, Thomas Flotte1, Haidong Dong1, Svetomir Markovic1, 1Mayo Clinic, Rochester, MN, USA, 2North Memorial Medical Center, Robbinsdale, MN, USA

P1.46 Placental Exosomes as Modulators of The Maternal Immune System During Pregnancy
Sean Nguyen1, Jacob Greenberg1, Margaret Petroff1, 1Michigan State University, East Lansing, MI, USA

P1.47 Isolation and characterization of leukocytes from the placental bed following normal term Cesarean section delivery.
M.G. Petroff1, S.K. Kshirsagar1, G. Grzesiak1, K. Behan1, T. Fortes1, A.M. Filler1, D. Martin1, 1Michigan State University, East Lansing, Michigan, USA

P1.48 Immunohistochemical analysis of CD138-positive Plasma cells in the endometrium
Maja Weber1, Bettina Toth2, Isabel Santillan1, Ruben Kuon1, Ekkehard Schleußer1, 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

INFECTION AND INFLAMMATION

P1.49 MicroRNAs regulating Toll Like Receptor inflammatory pathways in preterm and term cord blood and placenta.
Natalie Aboustate1, Vicki Clifton3, Michael Stark1,2, Nicolette Hodyl1,2, 1Robinson Research Institute, University of Adelaide, Adelaide, South Australia, Australia, 2Neonatal Medicine, Women’s and Children’s Hospital, Adelaide, South Australia, Australia, 3Mater Research Institute, Brisbane, Queensland, Australia

P1.50 Changes in the Human Placental Inflammasome Across Gestation
Paige Cooper1, Stephanie Teal2, Thomas Jansson1, Theresa Powell1, 1Department of Obstetrics & Gynecology, Division of Reproductive Science @ University of Colorado Anschutz Medical Campus, Aurora, CO, USA, 2Department of Obstetrics & Gynecology, Division of Family Planning @ University of Colorado Anschutz Medical Campus, Aurora, CO, USA, 3Department of Pediatrics, Division of Neonatology @ University of Colorado Anschutz Medical Campus, Aurora, CO, Aurora, CO, USA

P1.51 NLRP3 inflammasome expression and activation at the maternal-fetal interface in preeclamptic and healthy pregnancies
Lobke Gierman1, Gabriela Silva1,2, Guro Stødle1,2, Line Tangerås1,2, Liv Cecilie Thomsen1,3, Bente Skei1, Karin Collett1, Anne-Lise Beversmark2, Marie Aune1, Line Bjørge1,4, Ann-Charlotte Iversen1, 1Centre of Molecular Inflammation Research and Department of Cancer Research and Molecular Medicine, Norwegian University of Science and Technology, Trondheim, Norway, 2St. Olavs Hospital, Trondheim University Hospital, Trondheim, Norway, 3Haukeland University Hospital, Bergen, Norway, 4University of Bergen, Bergen, Norway


P1.52 Placental growth factor enhances toll-like receptor-dependent inflammatory responses in human mononuclear phagocytes
Laura Newell1, Shernan Holtan2, Jane Yates3, Leonardo Pereira4, Elizabeth Ann Enninga5, Douglas Creedon6, Svetomir Markovic6, Jeffrey Tyner1,2, Irina Burd6, Grover Bagby1,2, 1Knight Cancer Institute, Hematology and Medical Oncology, Oregon Health & Science University, Portland, OR, USA, 2Division of Hematology, Oncology and Transplant, University of Minnesota, Minneapolis, MN, USA, 3Northwest Veterans Affairs Cancer Research Center, Portland, OR, USA, 4Division of Maternal-Fetal Medicine, Department of Obstetrics and Gynecology, Oregon Health & Science University, Portland, OR, USA, 5Department of Medicine, Division of Hematology, Mayo Clinic Graduate School of Medicine, Rochester, MN, USA, 6North Memorial Medical Center, Department of Obstetrics and Gynecology, Robbinsdale, MN, USA, 7Department of Cell, Development, and Cancer Biology, Oregon Health & Science University, Portland, OR, USA, 8Integrated Research Center for Fetal Medicine, Department of Gynecology and Obstetrics, Johns Hopkins University School of Medicine, Baltimore, MD, USA

P1.53 The periodontal bacterium Porphyromonas gingivalis impairs spiral artery remodelling and induces acute atherosis in a rat model of periodontitis.
Mary Brown1, Priscilla Phillips1, Ann Progulske-Fox2, Leticia Reyes4,2, 1University of Florida, Department of Infectious Disease and Pathology, Gainesville, FL, USA, 2University of Florida, Department of Oral Biology, Gainesville, FL, USA, 3A.T. Still University of Health Sciences, Department of Microbiology & Immunology, Kirksville, MO, USA, 4University of Wisconsin - Madison, Department of Pathobiological Sciences, Madison, WI, USA

P1.54 Intrapartum factors and autism risk
Carolyn Salafia1, Jennifer Straughen1, Gabriela Perez-Avilan1, Victoria Onbreyt1, Beata Dygulska1, Sandford Lederman1, Pramod Narula1, 1New York Methodist Hospital, Brooklyn, NY, USA, 2Placental Analytics, LLC, New Rochelle, NY, USA, 3Henry Ford Hospital, Detroit, MI, USA

P1.55 Mechanistic study of human placental infection by Listeria monocytogenes
Joanna Marshall1, John Robinson1, William Ackerman1, Catalin Buhimschi1, Yoel Sadovsky2, Stephanie Seveau1, 1The Ohio State University, Columbus, Ohio, USA, 2Magee-Womens Research Institute, Pittsburgh, Pennsylvania, USA

P1.56 Focal segmental lymphoplasmacytic villitis of cytomegalovirus infection: The most ominous diffuse pattern of placental injury
Jerzy Stanek1, Tricia Smith1, Jenny Coffman1, 1Cincinnati Children’s Hospital Medical Center, Cincinnati, Ohio, USA

P1.57 Prenatal placental inflammatory exposures as measured by routine placental histopathology in a community cohort of ASD
Jennifer Straughen1, Carolyn Salafia2, Gabriela Perez-Avilan2,3, Victoria Onbreyt1, Ruchit Shah1, Alana Devine-Dunn1, Beata Dygulska1, Sandford Lederman1, Pramod Narula1, Dawn Misra4, 1Henry Ford Hospital, Detroit, MI, USA, 2New York Methodist Hospital, Brooklyn, NY, USA, 3Placental Analytics LLC, New Rochelle, NY, USA, 4Wayne State University, Detroit, MI, USA
Expression of NLRP3 inflammasome-related molecules in human trophoblasts
Kazuhiro Tamura¹, Wakana Ohneda¹, Mikihiro Yoshie¹, Gen Ishikawa², Akihito Nakai², Toshiyuki Takeshita², Hirotaka Nishi³, Keiichi Isaka¹, Naoko Kuwabara¹, Eiichi Tachikawa¹, ¹Department of Endocrine and Neural Pharmacology, Tokyo University of Pharmacy and Life Sciences, Tokyo, Japan, ²Department of Obstetrics and Gynecology, Nippon Medical School, Tokyo, Japan, ³Department of Obstetrics and Gynecology, Tokyo Medical University, Tokyo, Japan

Progesterone suppresses the lipopolysaccharide-induced inflammatory response in mononuclear cells isolated from human term placenta.
Eduardo Preciado-Martinez¹,², Guido Garcia-Ruiz¹,², Pilar Flores-Espinosa¹, Luisa Bermejo-Martinez¹, Gabriela Sedano-González¹, Araceli Mejia-Salvador¹, Guadalupe Estrada-Gutierrez², Guadalupe Razo-Aguilera¹, Martha Granados-Cepeda¹, Veronica Zaga-Clavellina¹, ¹Instituto Nacional de Perinatología Isidro Espinosa de los Reyes, Mexico City, Mexico, ²Facultad de Estudios Superiores Cuautitlan, Universidad Nacional Autonoma de Mexico, Mexico City, Mexico

Impaired trophoblast mitochondrial function following maternal nutrient restriction (MNR) in the baboon is alleviated by activation of mechanistic Target of Rapamycin (mTOR) signaling
Fredrick Joseph Rosario¹, Cun Li³, Leslie Myatt¹, Theresa Powell¹,², Peter W Nathanielsz¹, Thomas Jansson¹, ¹Department of OB/GYN, Anschutz Medical Campus, Aurora, Colorado, USA, ²Department of Pediatrics, Anschutz Medical Campus, Aurora, Colorado, USA, ³Southwest National Primate Research Center and Department of Animal Science, University of Wyoming, Laramie, WY, USA, ⁴Department of Obstetrics and Gynecology, Oregon Health and Science University, Portland, Oregon, USA

Identification of biomarkers to predict pregnancy complications: A metabolomics approach
Katie Powell¹,², Anthony Carrozzi²,³, Vitomir Tasevski¹,³, Anthony Ashton¹,³, Anthony Dona²,³, ¹Division of Perinatal Research, Kolling Institute, Northern Sydney Local Health District, St Leonards, NSW, Australia, ²Department of Cardiology, Kolling Institute, Northern Sydney Local Health District, St Leonards, NSW, Australia, ³Sydney Medical School Northern, University of Sydney, Sydney, NSW, Australia, ⁴Pathology North, NSW Health Pathology, Royal North Shore Hospital, St Leonards, NSW, Australia

Mechanistic target of rapamycin regulation of the trophoblast secretome.
Fredrick Joseph Rosario¹, Pardo Sam¹, Dominik Reinhold¹, Theresa Powell¹,², Sue Weintraub¹, Thomas Jansson⁴, ¹Department of OB/GYN, Anschutz Medical Campus, Aurora, Colorado, USA, ²Department of Pediatrics, Anschutz Medical Campus, Aurora, Colorado, USA, ³Department of Biostatistics & Informatics, Anschutz Medical Campus, Aurora, Colorado, USA, ⁴Department of Biochemistry, University of Texas Health Science Center, San Antonio, Texas, USA

Global re-wiring of molecular networks in placenta accreta
Roberta Hannibal¹, Janet Song¹, Kelly McGowan¹, Ann Fokkins¹, Amy Heerema-McKenney¹, Deirdre Lyell¹, Julie Baker¹, ¹Stanford University School of Medicine, Stanford, CA, USA
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<td>Jessica Hebert¹,², Terry Morgan¹, Oregon Health and Science University, Portland, OR, USA, Portland State University, Portland, OR, USA</td>
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<td>Michelle O’Brien¹, Dora Baczyk¹, John Kingdom¹,², Lunenfeld-Tanenbaum Research Institute, Toronto, Canada, Department of Obstetrics &amp; Gynaecology, University of Toronto, Toronto, Canada</td>
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<th>P1.67</th>
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<td>Isidora Rovic¹, Andrea Jurisicova¹,², Katherine Szelag¹, University of Toronto, Toronto, Ontario, Canada, Department of Obstetrics and Gynecology, Mount Sinai Hospital, Toronto, Ontario, Canada</td>
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<td>Qinglan Xia¹, Simon Morgan², Carolyn Salaña³, University of California, Davis, Davis, CA, USA, Imperial College, London, UK, Placental Analytics, LLC, New Rochelle, NY, USA</td>
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<th>P1.69</th>
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<td>Gitta Turowski¹, Line Sletner², Branka Yli³, Anne K. Jenum³, Borghild Roald¹,², Department of Pathology, Oslo University Hospital, Oslo, Norway, Department of Child and Adolescents Medicine, Akershus University Hospital, Lørenskog, Norway, Department of Obstetrics, Women Child clinic, Oslo University Hospital, Oslo, Norway, Institute of Health and Society, Department of General Practice, Faculty of Medicine, University of Oslo, Oslo, Norway, Institute of Clinical Medicine, Faculty of Medicine, University of Oslo, Oslo, Norway</td>
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<th>P1.70</th>
<th>Exposure to manganese and lead disrupt the human placental serotonergic system</th>
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<td>Marc Fraser¹,², Mélanie Viau¹, Joey St-Pierre¹,², Julie Lafond¹, Donna Mergler²,³, Céline Surette²,³, Cathy Vaillancourt¹,², INRS-Institut Armand Frappier, Laval, QC, Canada, CNIBIOSE, Montreal, QC, Canada, UQAM, Montreal, QC, Canada, Université de Moncton, Moncton, NB, Canada</td>
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<td>Xuan Shao¹, Huifen Lu¹, Dong Li², Ran Huo², Ming Liu¹, Yanlei Liu¹, Xuejiang Guo², Guangming Cao³, Yuxia Li¹, Jiahao Sha², Yan-ling Wang¹, State Key Laboratory of Stem Cell and Reproductive Biology, Institute of Zoology, Chinese Academy of Sciences, Beijing, China, State Key Laboratory of Reproductive Medicine, Nanjing Medical University, Nanjing, China</td>
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P1.72 THC suppresses human amniotic epithelial cell migration via the inhibition of MMP2 and MMP9.
Julei Yao1, Xinwen Chang1, Qizhi He1, Kai Wang1, Tao Duan1, 1Shanghai first maternity and infant hospital, Shanghai, China

P1.73 Evaluation of placental function using Ultrasound elastography
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P1.76 Balance of Lin28a and Lin28b in bovine trophoblast giant cells formation
Rodrigo da Silva Nunes Barreto1, Juliano Coelho da Silveira1, Lilian de Jesus Oliveira1, Maria Angelica Miglino1, Flavio Vieira Meirelles1, 1School of Veterinary Medicine and Animal Sciences, University of São Paulo, São Paulo, Brazil

P1.77 Evidence of Caveolin-1/Caveolae-mediated migration during early stages of placentation
Alejandra Reca1, Julieta Reppetti1, Cristina Ibarra1, Nora Martinez1, Alicia Damiano1,2, 1IFIBIO CONICET-UBA, Buenos Aires, Argentina, 2Facultad de Farmacia y Bioquimica-UBA, Buenos Aires, Argentina

P1.78 Impact of mono-2-ethylhexyl phthalate (MEHP) exposure on PPARγ expression and activity during human cytotrophoblast differentiation
Severine DEGRELLE1,2, Audrey CHISSEY1,2, Hussein SHOAITO1,2, Sophie GIL1,2, Thierry FOURNIER1,2, 1INSERM, UMR-S1139, Faculté des Sciences Pharmaceutiques et Biologiques, Paris F-75006, France, 2Université Paris Descartes, Sorbonne Paris Cité, Paris F-75006, France

P1.79 Evidence that extravillous trophoblast fusion into multinuclear trophoblast giant cells involves a mesenchymal-epithelial transition
Christina Duzyj1, Debra Heller2, Ciaran Mannion3, Christopher Koenig3, Stacy Zamudio4, Nicholas Illsley4, 1Department of Obstetrics and Gynecology, Rutgers- Robert Wood Johnson Medical School, New Brunswick, NJ, USA, 2Department of Pathology, Rutgers- New Jersey Medical School, Newark, NJ, USA, 3Department of Pathology, Hackensack University Medical Center, Hackensack, NJ, USA, 4Department of Obstetrics and Gynecology, Hackensack University Medical Center, Hackensack, NJ, USA
P1.80  The cytoprotective effects of bioflavonoids against oxidative stress during trophoblast cell invasion
Vernon Justice Ebegboni¹, Shweta Sunil Wadhare¹, John M. Dickenson¹, Shiva Sivasubramaniam¹,
¹Nottingham Trent University, Nottingham, UK

P1.81  Placental expression and role of lysyl oxidases (LOX) in the differentiation of human trophoblasts: a LOX Story?
Nadine Segond¹,², Audrey Chissey¹,², Jean Guibourdenche¹,², Alicia Grosso¹,², Vassilis Tsatsaris¹,², Sophie Gil¹,², Thierry Fournier¹,², U1139, Inserm, 75006 Paris, France, ²UMR-S 1139, University Paris Descartes, 75006 Paris, France

P1.82  Effects of chemotherapeutics on trophoblast cells in 2D, 3D and placental explant culture
Julia Heger¹, Karolin Fröhlich¹, Vanessa Scholz¹, Lisa Uhl¹, Ralf Mrowka², Udo R. Markert¹, ¹Placenta Lab, Jena, Germany, ²KIMIII Department of Experimental Nephrology, Jena, Germany

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Sampada Kallol¹, Xiao Huang¹,², Michael Lüthi¹,², Marc Baumann¹,², Daniel Surbek¹,², Edgar Ontsouka¹,², Christiane Albrecht¹,², ¹Institute of Biochemistry and Molecular Medicine, University of Bern, Bern, Switzerland, ²Swiss National Centre of Competence in Research (NCCR) TransCure, University of Bern, Bern, Switzerland, ³Department of Obstetrics and Gynaecology, University of Bern, Bern, Switzerland

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Kathryn Owens¹, Helen Jones³, ¹Cincinnati Children’s Hospital Medical Center, Cincinnati, Ohio, USA

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P1.86  Endothelin-1 (ET-1) down-regulates membrane type metalloproteinase (MT-MMP) expression in human first trimester trophoblasts: a role in trophoblast invasion in the context of pre-eclampsia
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P1.87  Analyses of key regulators of the Hippo signalling pathway in first trimester cytotrophoblast
Gudrun Meinhardt¹, Sandra Haider¹, Victoria Kunihis¹, Jürgen Pollheimer¹, Martin Knöfler¹, ¹Department of Obstetrics and Gynecology, Reproductive Biology Unit, Medical University of Vienna, Vienna, Austria

P1.88  Nodal induces syncytiotrophoblast differentiation
Uzma Nadeem¹, Peifeng Yang¹, Chun Peng¹, ¹York University, Toronto, Ontario, Canada
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Fen Ning¹,², Danyang Chen¹,², Jieping Yang¹,², Huishu Liu¹,², Gendie Lash¹,², ¹Guangzhou Institute of Pediatrics, Guangzhou Women and Children's Medical Center, Guangzhou, Guangdong Province, China, ²Guangzhou Medical University, Guangzhou, Guangdong Province, China

P1.90 BMP signaling plays a biphasic role during trophoblast differentiation of human embryonic stem cells.
Prasenjit Sarkar¹, Adam Mischler¹, Balaji Rao¹, ¹North Carolina State University, Raleigh, NC, USA

P1.91 A quiet death; hypoxic stress forces diminished growth and potency and large, irreversible trophoblast stem cell differentiation
Dan Rappolee¹, ¹Wayne State University, Detroit Mi, USA

P1.92 miR-126 regulates placental development and glucose metabolism
Abhijeet Sharma¹, Heidi Stuhlmann¹, ¹Weill Cornell Medical College, New York, New York, USA

P1.93 Diamine oxidase in pregnancy – An old acquaintance exclusively released from an unexpected source
Philipp Velicky¹, Sophie Pils¹, Bernd Jilma¹, Martin Knöfler¹, Thomas McElrath¹,², David Cantonwine¹, Tamara Weiss¹, Thomas Böhm¹, Jürgen Pollheimer¹, ¹Medical University of Vienna, Vienna, Austria, ²Children's Cancer Research Institute, Vienna, Austria, ³Harvard Medical School, Boston, Massachusetts, USA, ⁴Brigham and Women's Hospital, Boston, Massachusetts, USA

P1.94 The role of exchange protein directly activated cAMP 2 (EPAC2) in cAMP-mediated trophoblast differentiation
Mikihiro Yoshie¹, Kazuhiro Tamura¹, Rinna Tamakoshi¹, Misaki Okada¹, Eriko Keno¹, Gen Ishikawa², Akihito Nakai², Toshiyuki Takeshita², Hirotaka Nishi³, Keiichi Isaka³, Naoko Kuwabara¹, Eiichi Tachikawa¹, ¹Department of Endocrine and Neural Pharmacology, Tokyo University of Pharmacy and Life Sciences, Tokyo, Japan, ²Department of Obstetrics and Gynecology, Nippon Medical School, Tokyo, Japan, ³Department of Obstetrics and Gynecology, Tokyo Medical University, Tokyo, Japan

P1.95 Trimotif family-like 1 (Triml1)/Triml2’s role in placental development and the potential link to preterm birth through loss-of-function mouse models
Xuzhe Zhang¹,², Helen Jones¹, Yueh-Chiang Hu², Lois Muglia¹, ¹Perinatal Institute, Cincinnati Children’s Hospital Medical Center, Cincinnati, Ohio, USA, ²Graduate Program in Molecular & Developmental Biology, Division of Developmental Biology, Cincinnati Children’s Hospital Medical Center, Cincinnati, Ohio, USA, ³Department of Surgery, Cincinnati Children’s Hospital Medical Center, Cincinnati, Ohio, USA, ⁴Division of Developmental Biology, Cincinnati Children’s Hospital Medical Center, Cincinnati, Ohio, USA

P1.96 Histone methyltransferase EZH2 is critical for HSD11B2 expression in human trophoblast cells
Rujuan Zuo¹, Xiaohui Liu¹, Yi Lu¹, Wenjiao Li¹, Kang Sun¹, ¹Center for Reproductive Medicine, Ren Ji Hospital, School of Medicine, Shanghai Jiaotong University, Shanghai, China
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FETAL GROWTH RESTRICTION

P1.97  Up-Regulation of Placental Fatty Acid Binding Proteins in a Baboon Model of Intrauterine Growth Restriction
Stephanie Chassen¹, Cun Li³, Thomas Jansson², Peter Nathanielsz³,⁴, Theresa Powell¹,², University of Colorado Section of Neonatology, Aurora, CO, USA, ³University of Colorado Dept of Obstetrics & Gynecology, Division of Reproductive Sciences, Aurora, CO, USA, ⁴University of Wyoming Dept of Animal Science, Laramie, WY, USA, ¹Southwest National Primate Research Center, San Antonio, TX, USA

P1.98  The effect of different placental glucocorticoid receptor isoforms on the expression of glucocorticoid regulated genes in placenta of small-for-gestational age pregnancies
Vicki Clifton¹, Zarqa Saif², Mater Research Institute-UQ, Brisbane, QLD, Australia

P1.99  Inflammation-induced fetal growth restriction is associated with increased placental HIF-1α accumulation
Tiziana Cotechini¹, Kevin P.L. Robb¹, Camille Allaire¹, Arissa Sperou¹, Charles H. Graham¹, Department of Biomedical and Molecular Sciences, Queen’s University, Kingston, Ontario, Canada

P1.100  Moderate exercise attenuates lipopolysaccharide-induced inflammation and associated maternal and fetal morbidities in pregnant rats
Tiziana Cotechini¹, Karina T. Kasawara¹,², Shannyn K. Macdonald-Goodfellow¹, Fernanda G. Surita¹, João L. Pinto e Silva², Chandrakant Tayade¹, Maha Othman¹, Terrence R.S. Ozolinš¹, Charles H. Graham¹, Department of Biomedical and Molecular Sciences, Queen’s University, Kingston, Ontario, Canada, ²Department of Obstetrics and Gynaecology, University of Campinas, Campinas, SP, Brazil

P1.101  Melatonin Supplementation During Pregnancy Increases Fetal Abdominal Circumference and Umbilical Artery Relaxation In A Mouse Model of Fetal Growth Restriction
Lewis Renshall¹, Mark Wareing¹, Elizabeth Cowley¹, Elizabeth Cottrell¹, Colin Sibley¹, Susan Greenwood¹, Mark Dilworth¹, University of Manchester, Manchester, UK

P1.102  Developmental programming of sex differences in nephrogenesis involves proximal tubule oxidative stress in murine fetal growth restriction model
Mayu Morita¹, Jessica Hebert¹, Terry Morgan¹, OHSU, Portland, OR, USA

P1.103  A novel tryptophan-based mechanism regulating the bonus of the placental vascualr bed is impaired in intrauterine growth restriction and preeclampsia
Pablo Zardoya-Laguardia¹, Astrid Blaschitz², Birgit Hirschmugl³, Ingrid Lang², Martin Gauster¹, Martin Häusler³, Mila Cervar-Zivkovic², Eva Karplš, Christian Wadsack³, Saša Frank¹, Peter Sedlmayr¹, Institute of Cell Biology, Histology and Embryology, Graz, Styria, Austria, Department of Obstetrics and Gynaecology, Graz, Styria, Austria, Institute of Pathology, Graz, Styria, Austria, Institute of Molecular Biology and Biochemistry, Graz, Styria, Austria
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<td>Inverted Formin-2: a novel gene essential for female blood pressure regulation, placental development, and fetal growth.</td>
<td>Katherine Bezold Lamm&lt;sup&gt;1,2&lt;/sup&gt;, Helen Jones&lt;sup&gt;1&lt;/sup&gt;, Louis Muglia&lt;sup&gt;1&lt;/sup&gt;, Cincinnati Children's Hospital Medical Center, Cincinnati, OH, USA, &lt;sup&gt;2&lt;/sup&gt;University of Cincinnati, Cincinnati, OH, USA</td>
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<td>P1.105</td>
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<td>Priyadarshini Pantham&lt;sup&gt;1&lt;/sup&gt;, Don Armstrong&lt;sup&gt;1&lt;/sup&gt;, Derek Wildman&lt;sup&gt;1&lt;/sup&gt;, University of Illinois at Urbana-Champaign, Champaign, IL, USA</td>
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A tight infection bottleneck undermined by inadequate early immune responses define the dynamics of decidual listeriosis
Gabrielle Rizzuto1, Elisa Tagliani1, Priyanka Manandhar1, Adrian Erlebacher1, Anna Bakardjiev1
1University of California, San Francisco (UCSF), San Francisco, CA, USA

A pathogenic role for high risk Human Papillomavirus in villitis of unknown etiology
Noelyn Anne Hung1, Amanda Fischer1, Sankalita Ray1, Janice Royds1, Ryuji Fukuzawa2, Celia Devenish3, Tania Slatter1
1University of Otago, Department of Pathology, Dunedin, New Zealand, 2University of Otago, Department of Women’s and Children’s Health, Dunedin, New Zealand, 3Tokyo Metropolitan Children’s Medical Center, Tokyo, Japan

Time dependent fetal raman spectroscopy (RS) fingerprints of placental hypoxia
Kushal Gandhi1, Paul Brownhill1,2, Gary Ventolini2, Moss Hampton2, James Maher3, Natalia Schlabritz-Loutsevitch1, 1Texas Tech University Health Sciences Center, Odessa, Texas, USA, 2University of Manchester, Manchester, UK

Human trophoblast survival at low oxygen during the first trimester requires MMP2 mediated shedding of HBEGF
Chandni V. Jain1,2, Brian A. Kilburn3, Michael Hertz2, D. Randall Armant2,3, 1Departments of Physiology, Wayne State University School of Medicine, Detroit, MI, USA, 2Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, USA, 3Anatomy and Cell Biology, Wayne State University School of Medicine, Detroit, MI, USA, 4Program in Reproductive and Adult Endocrinology, Eunice Kennedy Shriver National Institute of Child Health and Human Development, NIH, DHHS, Bethesda, MD, USA

Periconceptional alcohol exposure in the rat reduces maternal blood space volume at mid-gestation, which may lead to perturbed fetal growth.
Jacinta Kalisch-Smith1, David Simmons1, Marie Pantaleon1, Karen Moritz1, 1The University of Queensland, Brisbane, QLD, Australia

Transcriptomic analysis of trophoblast cells obtained non-invasively in the first trimester of ongoing pregnancies to investigate severe uteroplacental insufficiency
Chandni V. Jain1,2, Hamid Reza Kohan-Ghadr3, Rani Fritz2, Alan D. Bolnick2, Brian A. Kilburn3, Stephen A. Krawetz2,3, Sascha Drewlo2, D. Randall Armant2,4, 1Physiology, Wayne State University School of Medicine, Detroit, MI, USA, 2Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, USA, 3Centre for Molecular Medicine and Genetics, Wayne State University School of Medicine, Detroit, MI, USA, 4Anatomy and Cell Biology, Wayne State University School of Medicine, Detroit, MI, USA, 5Program in Reproductive and Adult Endocrinology, Eunice Kennedy Shriver National Institute of Child Health and Human Development, NIH, DHHS, Bethesda, MD, USA

SATB1 promotion of trophoblast stem cell renewal through regulation of threonine dehydrogenase
Kaiyu Kubota1, M. A. Karim Rumi1, Michael Soares1, 1University of Kansas Medical Center, Kansas City, USA
ANATOMY AND PATHOLOGY

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Heather Brockway¹, Helen Jones¹, Mihaela Pavlincev¹, Louis Muglia¹, ¹Cincinnati Childrens Hospital Medical Center, Cincinnati OH, USA

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Fabian Fahlbusch¹, Sebastian Herdl¹, Gudrun Volkert¹, Ines Marek¹, Carlos Menendez-Castro¹, Stephanie Nögel¹, Matthias Rübner², Hanna Hübner², Andrea Hartner¹, Wolfgang Rascher¹, ¹Dept. of Pediatrics and Adolescent Medicine, University of Erlangen-Nürnberg, Erlangen, Germany, ²Department of Gynaecology and Obstetrics, University of Erlangen-Nürnberg, Erlangen, Germany

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Karolin Fröhlich¹, Julia Heger¹, Andre Schmidt¹, Yvonne Heimann¹, Boodor Al-Kawlani¹, Amelie Lupp², Gitta Turowski³, Udo R. Markert¹, ¹Placenta Lab, Jena, Germany, ²Institute of Pharmacology and Toxicology, Jena, Germany, ³Department of Pathology, Olso, Norway

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Eva Haeussner¹, Christian Roth³, Christoph Schmitz¹, Tanja Rübelmann³, Franz Edler von Koch³, Wolfgang Wall¹, Hans-Georg Frank¹, ¹Department of Anatomy II, LMU Munich, Munich, Germany, ²Hospital Dritter Orden, Munich, Germany, ³Institute for Computational Mechanics, TUM Munich, Munich, Germany

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Eva Haeussner¹, Christoph Schmitz¹, Hans-Georg Frank¹, ¹Department of Anatomy II, LMU Munich, Munich, Germany

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Timothy Lyden¹, Stephanie Strohbeen¹, ¹University of Wisconsin-River Falls, River Falls, Wisconsin, USA

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Rojan Saghian¹, Joanna James³, Merryn Tawhai, Alys Rachel Clark¹, ¹Auckland Bioengineering Institute, University of Auckland, Auckland, New Zealand, ²Department of Obstetrics & Gynaecology, University of Auckland, Auckland, New Zealand

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Ruchit Shah¹, Carolyn Salafia¹,², Theresa Girardi¹, George Merz², ¹Placental Analytics, New Rochelle, NY, USA, ²Institute for Basic Research, Staten Island, NY, USA
## POSTER SESSIONS

### CELL CULTURE/CELL LINES

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<td>¹American University of Beirut, Beirut, Lebanon</td>
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<td>¹School of Veterinary Medicine and Animal Science, University of São Paulo, São Paulo, Brazil</td>
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<td>¹Department of Obstetrics &amp; Gynecology, Division of Reproductive Sciences, University of Colorado Anschutz Medical Campus, Aurora, Colorado, USA, ²Department of Endocrinology, Diabetes and Metabolism, University of Colorado Anschutz Medical Campus, Aurora, Colorado, USA, ³Department of Pediatrics, Section of Neonatology, University of Colorado Anschutz Medical Campus, Aurora, Colorado, USA</td>
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<td>¹Placenta Lab, Jena, Germany, ²Department of Obstetrics &amp; Gynecology, University of Erlangen, Erlangen, Germany, ³Department of Pediatrics and Adolescent Medicine, University of Erlangen, Erlangen, Germany</td>
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<td>¹University Hospital Jena, Jena, Germany</td>
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<td>¹Department of Gynaecology and Obstetrics, University Hospital Erlangen, Friedrich-Alexander University Erlangen-Nuremberg, Comprehensive Cancer Center Erlangen-EMN, Erlangen, Germany, ²Department of Pediatrics and Adolescent Medicine, University Hospital Erlangen, Friedrich-Alexander University Erlangen-Nuremberg, Comprehensive Cancer Center Erlangen-EMN, Erlangen, Germany, ³Institute of Human Genetics, University Hospital Erlangen, Friedrich-Alexander University Erlangen-Nuremberg, Comprehensive Cancer Center Erlangen-EMN, Erlangen, Germany</td>
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<td>Allerdien Visser¹, Omar Michel¹, Gunilla Kleiverda², Cees Oudejans¹, Marie van Dijk¹</td>
<td>¹VU University Medical Center, Amsterdam, The Netherlands, ²Flevoziekenhuis, Almere, The Netherlands</td>
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¹VU University Medical Center, Amsterdam, The Netherlands
²Flevoziekenhuis, Almere, The Netherlands
P2.16  **Karyotypes of trophoblastic cell lines**
Maja Weber1, Faezeh Vasheghani2,3, Claudia Göhner1,4, Thomas Liehr2, Ekkehard Schleussner1, Justine S. Fitzgerald1, Udo R. Markert1, Anja Weise2,1, Department of Obstetrics, University Hospital Jena; Placenta-Lab, Jena, Germany, 1Institute of Human Genetics, Jena University Hospital, Jena, Germany, 2Osteoarthritis Unit NotreDame Hospital, University of Montreal, Montreal, Canada, 3Department of Obstetrics and Gynaecology, University Medical Center Groningen, University of Groningen, Groningen, The Netherlands, 4Praxis Klinik am Anger, Kinderwunschzentrum Erfurt, Erfurt, Germany

**DIABETES/OBESITY**

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Juscieli Moreli1,2, Marina Paula-Silva1,2, Iracema Calderon3, Sandra Farsky4, Sonia Oliani1,2, Estela Bevilacqua5, 1Post-graduation in Structural and Functional Biology, Federal University of São Paulo - UNIFESP, Paulista School of Medicine, São Paulo, SP, Brazil, 2Department of Biology, Institute of Biosciences, Letters and Exact Sciences; São Paulo State University -UNESP, São José do Rio Preto, SP, Brazil, 3Graduate Program in Gynecology, Obstetrics and Mastology, Botucatu Medical School, São Paulo State University / UNESP, São Paulo, SP, Brazil, 4Department of Clinical and Toxicological Analyses, Faculty of Pharmaceutical Sciences, University of São Paulo / USP, São Paulo, SP, Brazil, 5Department of Cell and Developmental Biology, Institute of Biomedical Sciences, University of São Paulo - USP, São Paulo, SP, Brazil

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Maira Carrillo1, Cun Li2,3, Cezary Skobowiat4,5, Gary Ventolini1, Marcel Chuecos1, Patrick Joseph6, Edward Dick7, Gene Hubbard8, Peter Nathanielsz2,3, Natalia Schlabritz-Louisevitch1, 1Texas Tech University Health Sciences Center, Odessa, TX, USA, 2Texas Pregnancy and life Course Health Center, Texas Biomedical Research Institute San Antonio Texas, San Antonio, TX, USA, 3Department of Animal Science, University of Wyoming, Laramie, WY, USA, 4Department of Pharmacodynamics and Molecular Pharmacology, Collegium Medicum Nicolaus Copernicus University of Poland, Torun, Poland, Poland, 5Department of Dermatology, University of Alabama at Birmingham, Birmingham, AL, USA, 6Department of Pathology, University of Tennessee, Memphis, TN, USA, 7Texas Biomedical Research Institute, San Antonio, TX, USA, 8Department of Pathology, Unviersity of Texas Health Science Center at San Antonio, San Antonio, TX, USA

P2.19  **Matrix proteoglycan changes in human placenta after gestational diabetes.**
Francisca Diaz-Perez1, Ingrid Lang-Olip1, Gernot Desoye1, Ursula Hiden1, 1Medical University of Graz, Graz, Austria

P2.20  **The relationship between maternal body mass index and exosomal concentration across gestation**
Omar Elfeky1, Sherri Longo2, Gregory Rice1,2, Carlos Salomon1,2, 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, Queensland, Australia, 2Department of Obstetrics and Gynecology, Ochsner Baptist Hospital, New Orleans, Louisiana, USA
**POSTER SESSIONS**

**P2.21** Influence of gestational diabetes mellitus on transplacental iron transport
Jonas Zaugg\(^1,2\), Malgorzata Wegner\(^1,2\), Xiao Huang\(^1,2\), Edgar Ontsouka\(^1\), Marc Baumann\(^3\), Daniel Surbek\(^3\), Meike Körner\(^4\), Matthias A. Hediger\(^1,2\), Christiane Albrecht\(^1,2\), \(^1\)Institute of Biochemistry and Molecular Medicine, Bern, Switzerland, \(^2\)Swiss National Centre of Competence in Research (NCCR) TransCure, Bern, Switzerland, \(^3\)Department of Obstetrics and Gynaecology, Bern, Switzerland, \(^4\)Pathologie Länggasse, Bern, Switzerland

**P2.22** Activation of placental mTOR signalling in rats with GDM is prevented by dietary treatments with PUFAs in the previous generation.
Evangelina Capobianco\(^1\), Theresa Powell\(^2\), Daiana Fornes\(^1\), Thomas Jansson\(^2\), Alicia Jawerbaum\(^1\), \(^1\)Laboratory of Reproduction and Metabolism, CEFYBO. UBA. CONICET. School of Medicine, University of Buenos Aires., Buenos Aires, Argentina, \(^2\)Division of Reproductive Sciences, Department of OB/GYN, University of Colorado Denver Anschutz Medical Campus, Aurora, Colorado, USA

**P2.23** Decreased protein expression of folate transporters in early pregnancy placentas of obese women
Rebecca H. Jessel\(^2,1\), Frederick Rosario Joseph\(^1\), Stephanie B. Teal\(^1\), Thomas Jansson\(^1\), Theresa Powell\(^1,4\), \(^1\)University of Colorado, Department of Obstetrics and Gynecology, Division of Reproductive Sciences, Aurora, CO, USA, \(^2\)University of Colorado, Department of Obstetrics and Gynecology, Division of Maternal Fetal Medicine, Aurora, CO, USA, \(^3\)University of Colorado, Department of Obstetrics and Gynecology, Division of Family Planning, Aurora, CO, USA, \(^4\)University of Colorado, Department of Pediatrics, Neonatology Section, Aurora, CO, USA

**P2.24** Effect of high glucose exposure on endothelial function mediated by feto-placental endothelial exosomes.
Tamara Sáez\(^1,2\), Luis Sobrevia\(^2,3\), Marijke Faas\(^1\), \(^1\)University Medical Center Groningen. University of Groningen, Groningen, The Netherlands, \(^2\)Pontificia Universidad católica de Chile, Santiago, Chile, \(^3\)Centre for Clinical Research. \(^4\)University of Queensland, Queensland, Australia

**P2.25** Gestational diabetes and feto-placental endothelial dysfunction: role of exosomes from human umbilical vein endothelial cells on L-arginine/NO signalling pathway.
Tamara Sáez\(^1,2\), Rocio Salsoso\(^1\), Carlos Sanhueza\(^1\), Fabian Pardo\(^1\), Andrea Leiva\(^1\), Marijke Faas\(^1\), Luis Sobrevia\(^1,3\), \(^1\)Pontificia Universidad católica de Chile, Santiago, Chile, \(^2\)University Medical Centre Groningen. University of Groningen, Groningen, The Netherlands, \(^3\)Centre for Clinical Research. University of Queensland, Queensland, Australia

**P2.26** Placental predictors of childhood body size
Jennifer Straughen\(^1\), George Divine\(^1\), Gabriela Perez-Avilan\(^3\), Beata Dygulska\(^2\), Alana Devine Dunn\(^1\), Sam Ngu\(^1\), Xilu Ma\(^3\), Cassie Jourdan\(^2\), Karen Toubi\(^2\), Jennifer Grad\(^2\), Pramod Narula\(^3\), Sanford Lederman\(^3\), Carolyn Salafia\(^2,3\), \(^1\)Department of Public Health Sciences, Henry Ford Hospital, Detroit, MI, USA, \(^2\)Department of Pediatrics, New York Methodist Hospital, Brooklyn, NY, USA, \(^3\)Department of Obstetrics and Gynecology, New York Methodist Hospital, Brooklyn, NY, USA
**FETAL MEMBRANES**

**P2.27** Oxidative stress induced amnion cell derived exosomes produce inflammatory changes in myometrial cells: A feto-maternal signaling in human parturition
Ramkumar Menon\(^1\), Samantha Sheller\(^1\), George Saade\(^1\), Carlos Salomon\(^2\), \(^1\)Division of Maternal-Fetal Medicine & Perinatal Research, Department of Obstetrics & Gynecology, The University of Texas Medical Branch at Galveston, Galveston, Texas, USA, \(^2\)Exosome Biology Laboratory, Centre for Clinical Diagnostics, UQ Centre for Clinical Research, Faculty of Health Sciences, University of Queensland, Brisbane, QLD, Australia

**P2.28** Vesicular uptake of albumin in human amnion - implications in amniotic fluid regulation
Margarita Sharshiner\(^1\), Cecilia Cheung\(^1\), Robert Brace\(^1\), \(^1\)Oregan Health & Science University, Portland, OR, USA

**GENOMICS/EPIGENOMICS**

**P2.29** Placental MTHFR 677C>T genotypes in pregnancy pathologies
Giulia Del Gobbo\(^1\), E Magda Price\(^1,2\), Courtney Hanna\(^3,4\), Wendy Robinson\(^1,2\), \(^1\)University of British Columbia, Vancouver, BC, Canada, \(^2\)Child & Family Research Institute, Vancouver, BC, Canada, \(^3\)Babraham Institute, Cambridge, UK, \(^4\)University of Cambridge, Cambridge, UK

**P2.30** Aberrant inflammation associated with pregnancy complications in rats leads to maternal and fetal epigenetic alterations
Takafumi Ushida\(^1,2\), Shannyn K. Macdonald-Goodfellow\(^1\), M. Yat Tse\(^1\), Stephen C. Pang\(^1\), Louise M. Winn\(^1\), Charles H. Graham\(^1\), \(^1\)Queen’s University, Kingston, Ontario, Canada, \(^2\)Nagoya University, Nagoya, Japan

**P2.31** Incomplete reprogramming of germline DNA methylation in the human placenta
Hirotaka Hamada\(^1,4\), Hiroaki Okae\(^1\), Mikita Suyama\(^3\), Hiroyuki Sasaki\(^2\), Nobuo Yaegashi\(^4\), Takahiro Arima\(^1\), \(^1\)Department of Informative Genetics, Environment and Genome Research Center, Tohoku University Graduate School of Medicine, Sendai, Miyagi, Japan, \(^2\)Department of Molecular Genetics, Medical Institute of Bioregulation, Kyushu University, Fukuoka, Japan, \(^3\)Division of Bioinformatics, Medical Institute of Bioregulation, Kyushu University, Fukuoka, Japan, \(^4\)Departments of Obstetrics and Gynecology, Tohoku University Graduate School of Medicine, Sendai, Miyagi, Japan

**P2.32** Using DNA methylation signatures in placental tissue and cells to gain insight into chorioamnionitis
Chaini Konwar\(^1,2\), E. Magda Price\(^1,2\), Wendy P Robinson\(^1,2\), \(^1\)University of British Columbia, Vancouver, BC, Canada, \(^2\)Child and Family Research Institute, Vancouver, BC, Canada

**P2.33** Placental genome and transcriptome profile in unexplained recurrent pregnancy loss
Maris Laan\(^1\), Laura Kasak\(^1\), Siim Söber\(^1\), Kristiina Rull\(^1,2\), \(^1\)University of Tartu, Tartu, Estonia, \(^2\)Women’s Clinic, Tartu University Hospital, Tartu, Estonia

**P2.34** Expression of C19MC and C14MC miRNAs in pregnancy pathologies
Diana M. Morales-Prieto\(^1\), Daniel Fabig\(^1\), Ivan Dimitrov\(^1\), Ruby N. Gutiérrez-Samudio\(^1\), Tanja Groten\(^1\), Udo R. Markert\(^1\), \(^1\)University Hospital Jena, Jena, Thuringia, Germany
Placental telomere length decline with gestational age differs by sex and TERT, DNMT1, and DNMT3A DNA methylation.

Wendy Robinson¹,², Yao Liu¹, Samantha Wilson¹,², ¹University of British Columbia, Vancouver BC, Canada, ²Child & Family Research Institute, Vancouver BC, Canada

Unravelling the relationship between early and late-onset preeclampsia. What does the placental DNA methylation profile reveal?

Samantha L. Wilson¹,², Katherine Leavez³, Peter von Dadelszen¹,², Brian Cox³, Wendy P. Robinson¹,², ¹University of British Columbia, Vancouver, BC, Canada, ²Child and Family Research Institute, Vancouver, BC, Canada, ³University of Toronto, Toronto, ON, Canada

Bench to population: translating hCG biology to the sex-specific effects of endocrine disruptors on fetal development

Jennifer Adibi¹,², ¹University of Pittsburgh Graduate School of Public Health, Department of Epidemiology, Pittsburgh, PA, USA, ²University of Pittsburgh Department of Obstetrics, Gynecology and Reproductive Sciences, Pittsburgh, PA, USA

Maternal dexamethasone exposure in the mouse causes sex specific alterations in the pattern of glucocorticoid receptor isoform expression.

James Cuffe¹,², Zarqa Saif³, Karen Moritz², Vicki Clifton³, ¹School of Medical Sciences, Griffith University, Gold Coast, QLD, Australia, ²School of Biomedical Sciences, The University of Queensland, Brisbane, QLD, Australia, ³Mater Research Institute, The University of Queensland, Brisbane, QLD, Australia

The impact of cord blood leptin levels on growth in early infancy.

Emily McDonald¹,², Sangshin Park¹, Sunthorn Pond-Tor¹, Remigio Olveda¹, Luz Acosta¹, Veronica Tallo¹, Palmira Baltazar³, Jonathan Kurtis², Jennifer Friedman¹,², ¹Center for International Health Research, Rhode Island Hospital, Providence, RI, USA, ²Brown University, Providence, RI, USA, ³Research Institute of Tropical Medicine, Manila, The Philippines

A HIF-KDM3A-MMP12 regulatory pathway triggers adaptations at the maternal-fetal interface.

Damayanti Chakraborty¹, Wei Cui², Regan Scott³, Pramod Dhakal¹, Stephen Renaud³, Gracy Rosario⁶, Adam Krieg⁴,⁵, Mohammad Rumi¹,², Michael Soares¹,³, ¹Department of Pathology and Laboratory Medicine, University of Kansas Medical Center, Kansas City, Kansas, USA, ²Department of Veterinary & Animal Sciences, University of Massachusetts, Amherst, Massachusetts, USA, ³Department of Anatomy and Cell Biology, University of Western Ontario, London, Ontario, Canada, ⁴Department of Obstetrics and Gynecology, University of Kansas Medical Center, Kansas City, Kansas, USA, ⁵Institute for Reproductive Health and Regenerative Medicine, University of Kansas Medical Center, Kansas City, Kansas, USA, ⁶Global Pathways Institute, Mumbari, Maharashtra, India
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Sonia DaSilva-Arnold1, Christina Chong1, Stacy Zamudio1, Abdulla Al-Khan1, Nicholas Illsley1, 1Hackensack University Medical Center, Hackensack, NJ, USA

**P2.42** The tight junction protein occludin is regulated by RhoA and activin A in JEG3 trophoblast cells 
Sonia DaSilva-Arnold1, Stacy Zamudio1, Abdulla Al-Khan1, Nicholas Illsley1, 1Hackensack University Medical Center, Hackensack, NJ, USA

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fatemeh davari tanha1, ensieh tehraninejad1, mahbod kaveh1, zahra kaveh1, 1Tehran university of medical sciences, Tehran, Iran

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fatemeh davari tanha1, mahbod kaveh1, zahra kaveh1, ensieh tehraninejad1, 1Tehran university of medical sciences, Tehran, Iran

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Caroline Dunk1,4, Marie Van Dijk2, Ruhul Choudhury3, Lynda Harris3, Rebecca Lee Jones3, Stephen Lye1,4, 1Lunenfeld Tanenbaum Research Institute, Mount Sinai Hospital, Toronto, Canada, 2Department of Clinical Chemistry, VU University Medical Center, Amsterdam, The Netherlands, 3Maternal and Fetal Health Research Group, University of Manchester, Manchester, UK, 4Departments of Obstetrics and Gynecology and Physiology, University of Toronto, Toronto, Canada

**P2.46** A Revised Hypothesis for Placental Syncytialization in Sheep 
Heewon Seo1, Fuller Bazer1, Robert Burghardt1, Greg Johnson1, 1Texas A&M University, College Station, TX, USA

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Karin Windsperger1, Leila Saleh1, Sabine Dekan2, Martin Knößer1, Jürgen Pollheimer1, 1Department of Obstetrics and Gynecology, Reproductive Biology Unit, Medical University of Vienna, Vienna, 1090 Vienna, Austria, 2Clinical Institute for Pathology, Medical University of Vienna, Vienna, 1090 Vienna, Austria

**OXIDATIVE STRESS**

**P2.48** The antioxidant N-acetyl cysteine of oxidative stress on GBS-stimulated inflammatory pathways in human gestational membranes 
Rita Loch-Caruso1, Hae-Ryung Park1,2, Erica Boldenow1,3, 1University of Michigan, Ann Arbor, MI, USA, 2Harvard University, Boston, MA, USA, 3Seattle Children’s Hospital, Seattle, WA, USA
**P2.49** Trophoblast mitochondrial biogenesis and functionality is increased with selenium supplementation

Anthony Perkins¹, Alisha Khera¹, Olivia Holland¹, Jessica Vanderlelie¹, ¹Griffith University, Gold Coast, Queensland, Australia

**Preeclampsia**

**P2.50** Hypoxia and iron imbalance impairs JMJD6-mediated histone demethylation of VHL in preeclampsia

Sruthi Alahari¹², Alessandro Rolfo³, Isabella Caniggia¹², ¹Lunenfeld-Tanenbaum Research Institute, Toronto, Ontario, Canada, ²Departments of Physiology, and Obstetrics & Gynaecology, University of Toronto, Toronto, Ontario, Canada, ³Department of Surgical Sciences, University of Turin, Turin, Piedmont, Italy

**P2.51** Title: VEGF and sFlt-1 polymorphisms in Preeclampsia

Pallavi Arora¹, Renu Dhingra¹, Neerja Bhatla¹, Arundhati Sharma¹, ¹All India Institute of Medical Sciences, New Delhi, India, ²All India Institute of Medical Sciences, New Delhi, India

**P2.52** Growth arrest-specific 6 (Gas6)/AXL signaling induces preeclampsia

Camilo Mejia¹, Montana Wayment¹, Troy Monson¹, Paul Reynolds¹, Juan Arroyo¹, ¹Brigham Young University, Provo, Utah, USA

**P2.53** Can placental pathology identify women at high risk of cardiovascular disease following preeclampsia?

Samantha Benton¹, David Grynspan², Graeme Smith¹, Shannon Bainbridge¹, ¹University of Ottawa, Ottawa, Ontario, Canada, ²Children’s Hospital of Eastern Ontario, Ottawa, Ontario, Canada, ³Queen’s University, Kingston, Ontario, Canada

**P2.54** PIM 1 Kinase in preeclampsia

Stella M. Mary¹, Maja Weber¹, Wittaya Chaiwangyen¹², Ekkehard Schleussner¹, Udo R. Markert¹, ¹Placenta-Lab, Department of Obstetrics, University hospital Jena, Germany, Jena, Germany, ²Department of Biochemistry, School of Medical Sciences, University of Phayao, Phayao, Thailand

**P2.55** Nifedipine prevents endothelial cell activation in response to placental micro- and nano- vesicles released from 1st trimester placental explants that had been treated with preeclamptic sera

Qi Chen¹², Xirong Xiao³, Michelle Xiao³, Mancy Tong¹, Michelle Wise¹, Peter Stone¹, Larry Chamley¹, ¹The University of Auckland, Auckland, New Zealand, ²Fudan University, Shanghai, China

**P2.56** The expression of imprinted genes in the preeclamptic placenta

Julian Christians³, Nicole Tortora³, ³Simon Fraser University, Burnaby, BC, Canada

**P2.57** Decidua-driven Differentiation of Angiogenic Phenotype in Second Trimester Peripheral Blood Neutrophils from Healthy and Preeclamptic Women

Melissa Kwan¹, Caroline Dunk²³, Mark Kibschull², Lynda Harris⁴, Rebecca Lee Jones⁴, Stephen Lye¹², ¹Department of Physiology, University of Toronto, Toronto, Canada, ²Lunenfeld-Tanenbaum Research Institute, Mount Sinai Hospital, Toronto, Canada, ³Department of Obstetrics and Gynecology, Hadassah Hebrew University Medical Center, Jerusalem, Israel, ⁴Maternal and Fetal Health Research Group, University of Manchester, Manchester, UK, ⁵Department of Obstetrics and Gynecology, University of Toronto, Toronto, Canada
P2.58 Secretion of sFLT1 and sENG into the maternal circulation
Leonardo Ermini\(^1\), Jonathan Ausman\(^2\), Michael Litvack\(^1\), Michelle Letarte\(^1\), Martin Post\(^1\), Isabella Caniggia\(^2\), \(^1\)Hospital for Sick Children, Toronto, Canada, \(^2\)The Lunenfeld-Tanenbaum Research Institute, Mount Sinai Hospital, Toronto, Canada

P2.59 New generation antiplatelet therapies to prevent preeclampsia
Natalie Hannnan\(^1,2\), Tu’uhevaha Kaitu’u-Lino\(^1,2\), Sally Beard\(^1,2\), Natalie Binder\(^1,2\), Stephen Tong\(^1,2\), \(^1\)University of Melbourne, Department of Obstetrics and Gynaecology, Melbourne, Vic, Australia, \(^2\)Translational Obstetrics Group, Mercy Hospital for Women, Heidelberg, Vic, Australia

P2.60 A1M identified by machine learning guided search as a predictor of preeclampsia in a case-control study in the SCOPE cohort.
Åsa Näävä\(^1\), Lena Erlandsson\(^1\), Fredrik Nilsson\(^1\), Louise Kenny\(^2\), Stefan Hansson\(^1\), \(^1\)Institution of clinical sciences, Department of Obstetrics and Gynecology, Lund University, Lund, Sweden, \(^2\)Department of Obstetrics and Gynaecology, University College Cork, Cork, Ireland

P2.61 Decreased expression of indoleamine 2, 3-dioxygenase in villous stromal endothelial cells of placentas with preeclampsia and/or fetal growth restriction
Naoyuki Iwahashi\(^1\), Madoka Yamamoto\(^1\), Tamaki Yahata\(^1\), Mika Mizoguchi\(^1\), Sakiko Nanjo\(^1\), Aya Kobayashi\(^1\), Yuko Tanizaki\(^1\), Michihiisa Shiro\(^1\), Nami Ota\(^1\), Yasushi Mabuchi\(^1\), Shigetaka Yagi\(^1\), Sawako Minami\(^1\), Kazuhiko Ino\(^0\), \(^1\)Wakayama Medical University, Wakayama, Japan

P2.62 Mitochondrial biogenesis molecules AMPK, SIRT1 and PGC1α and their relationship to sFlt1 and sEng secretion in preeclampsia
Tu’uhevaha Kaitu’u-Lino\(^1\), Fiona Brownfoot\(^1\), Roxanne Hastie\(^1\), Natalie Hannnan\(^1\), Ping Cannon\(^1\), Minh Deo\(^1\), Stephen Tong\(^1\), \(^1\)Dept of Obstetrics and Gynaecology, Mercy Hospital for Women, University of Melbourne, Heidelberg, Melbourne, Australia

P2.63 Characterisation of extravillous trophoblast-derived exosomal protein content in response to hypoxia
Vyjayanthi Kinhal\(^1\), Katherin Scholz-Romer\(^1\), Omar Elfeky\(^1\), Gregory Rice\(^1,2\), Carlos Salomon\(^1,2\), \(^1\)Exosome 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, \(^2\)Department of Obstetrics and Gynecology, Ochsner Baptist Hospital, New Orleans, Louisiana, USA

P2.64 Microparticles cause preeclampsia and embryonic growth restriction by platelet-mediated inflammasome activation in the embryonic trophoblast
Shrey Kohli\(^1\), Moh’d Mohanad Al-Dabet\(^1\), Satish Ranjan\(^1\), Fabian Bock\(^1\), Khurrum Shahzad\(^1\), Berend Isermann\(^1\), \(^1\)Institute of Clinical Chemistry & Pathobiochemistry, Medical Faculty, Otto-von-Guericke University, Magdeburg, Germany, Germany

P2.65 Epigenetic Regulation of Placental Gene Expression in Transcriptional Subclasses of Preeclampsia
Katherine Leavey\(^1\), Samantha Wilson\(^2\), Shannon Bainbridge\(^3\), Wendy Robinson\(^2\), Brian Cox\(^1\), \(^1\)University of Toronto, Toronto, ON, Canada, \(^2\)University of British Columbia, Vancouver, BC, Canada, \(^3\)University of Ottawa, Ottawa, ON, Canada
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**P2.66** Activation of cholinergic anti-inflammatory pathway by nicotine ameliorates lipopolysaccharide-induced preeclampsia-like symptoms in pregnant rats
Yuanyuan Liu1,2, Jinying Yang1,2, Junjie Bao1,2, Xiaolan Li1,2, Aihua Ye1,2, Guozheng Zhang1,2, Huishu Liu1,2, 1Guangzhou Women and Children's Medical Center, Guangzhou, China, 2Guangzhou Medical University, Guangzhou, China

**P2.67** Decidual NK cells facilitate the interaction between trophoblastic and endothelial cells via producing VEGF-C and HGF
Li-Yang Ma1, Guanlin Li1, Yuchun Zhu2, Guangming Cao1, Mei-Rong Du1, Hao Wang1, Yanlei Liu1, Yanyan Yang1, Yu-xia Li1, Da-Jin Li1, Huixia Yang2, Yan-ling Wang1, 1Institute of Zoology, Chinese Academy of Sciences, Beijing, China, 2Peking University First Hospital, Beijing, China, 3Hospital and Institute of Obstetrics and Gynecology, Fudan University Shanghai Medical College, Shanghai, China

**P2.68** GLUT1-down-regulation leads to a "premature senescence" in preeclamptic syncytiotrophoblasts
Camilla Marini1,2,4, Benjamin P. Lüscher1,3, Daniel V. Surbek1,2, Marc U. Baumann1,2, 1Department of Obstetrics and Gynaecology, University Hospital of Bern, Bern, Switzerland, Switzerland, 2Department of Clinical Research, University of Bern, Bern, Switzerland, Switzerland, 3Institute of Biochemistry and Molecular Medicine, University of Bern, Bern, Switzerland, Switzerland, 4Graduate School for cellular and Biomedical Sciences (GCB), University of Bern, Bern, Switzerland, Switzerland

**P2.69** Low-dose aspirin improves TGFβ1-mediated trophoblast proliferation and migration in an in vitro model of pre-eclampsia.
Jessica E Davies1,2, Hannah EJ Yong1,2, Anthony J Borg2, Shaun P Brennecke1,2, Padma Murthi2,3, 1Department of Obstetrics and Gynaecology, The University of Melbourne, Melbourne, Victoria, Australia, 2Department of Maternal-Fetal Medicine, The Royal Women's Hospital, Melbourne, Victoria, Australia, 3Department of Medicine, Monash University, Clayton, Victoria, Australia

**P2.70** Histopathological changes in placenta of a preeclampsia-like L-name rat model treated with sildenafil-citrate
Wendy Phoswa1, 1University of Kwazulu Natal, Durban, South Africa

**P2.72** Characterization of a human trophoblast model to evaluate novel therapeutics for preeclampsia
Jiawu Zhao1, Rebecca McLeese1, Michelle Hookham2, Timothy Lyons1, Jeremy Yu1, 1Queen's University Belfast, Belfast, UK, 2Clinical Biochemistry Royal Victoria Hospital, Belfast, UK

**PRETERM LABOUR AND BIRTH**

**P2.73** When is a prostaglandin not a prostaglandin?
Hassendrini, N. Peiris1, Kanchan Vaswani1, Yong Qin Koh1, Leon Oh1, Sarah Reed1, Murray, D. Mitchell1, 1The University of Queensland, Centre for Clinical Research, Brisbane, Queensland, Australia
P2.74  Identification of exosomal miRNA biomarkers at early gestation (<18 weeks) in asymptomatic women at early gestation (<18 weeks) who subsequently develop spontaneous preterm birth.
Vyjayanthi Kinhal, Dominic Guanzon, Katherin Scholz-Romer, Sherri Longo, Stephen Fortunato, Ramkumar Menon, Gregory Rice, Carlos Salomon, Exosome Biology Laboratory, University of Queensland Centre for Clinical Diagnostics, Royal Brisbane and Women’s Hospital, The University of Queensland, Brisbane, QLD, Australia, Department of Obstetrics and Gynecology, Ochsner Baptist Hospital, New Orleans, Louisiana, USA, Division of Maternal-Fetal Medicine & Perinatal Research, Department of Obstetrics & Gynecology, The University of Texas Medical Branch at Galveston, Galveston, Texas, USA

P2.75  Histologic chorioamnionitis occurs equally often in women with preterm labour regardless of the fetal membrane status
Vedran Stefanovic, Leena Rahkonen, Minna Tikkanen, Anu Patari-Sampo, Jorma Paavonen, Tarja Myntti, Department of Obstetrics and Gynecology, Helsinki University Hospital, Helsinki, Finland, Department of Microbiology, Helsinki University Hospital, Helsinki, Finland

STEM CELLS

P2.76  Decellularized bovine cotyledon as biological scaffold for bioengineering
Rodrigo da Silva Nunes Barreto, Patricia Romagnolli, Paula Fratini, Maria Angelica Miglino, School of Veterinary Medicine and Animal Sciences, University of São Paulo, São Paulo, Brazil

P2.77  Understanding the behaviour of “stem like cells” derived from transformed first trimester trophoblast cell lines
Reham Balahmar, Vernon Justice Ebegbobi, Sankalita Ray, Shiva Sivasubramaniam, Nottingham Trent University, Nottingham Trent University, UK

P2.78  Survival Secrets: Identifying factors that aid the propagation of Hoechst-low side-population trophoblast
Teena Gamage, Larry Chamley, Joanna James, The University of Auckland, Auckland, New Zealand

P2.79  Human induced pluripotent stem cells (iPS cell) differentiate to trophoblast lineage in xeno free culture
Junya Kojima, Hidenori Akutsu, Hirotaka Nishi, Naoaki Kuji, Keiichi Isaka, Department of Obstetrics and Gynecology, Tokyo Medical University, Tokyo, Japan, National Center for Child Health and Development, Tokyo, Japan

P2.80  Role of decorin in trophoblast stem cell self-renewal and differentiation
Pinki Nandi, Peeyush Lala, Department of Anatomy and Cell Biology, University of Western Ontario, London, Ontario, Canada, Children’s Health Research Institute, University of Western Ontario, London, Ontario, Canada

P2.81  Characterizing the contribution of extracellular matrix (ECM) to mouse trophoblast stem and progenitor cell differentiation
Anirudha Harihara, Katarina Gustin, Kellie Breen, Bryony Natale, David Natale, University of California San Diego, La Jolla, CA, USA
POSTER SESSIONS

**P2.82** Characterization of the human blood-placental barrier choline transporter  
Masato Inazu\(^1\)\(^,\) Miki Yara\(^3\), Tsuyoshi Yamanaka\(^2\), \(^1\)Institute of Medical Science, Tokyo Medical University, Tokyo, Japan, \(^2\)Department of Molecular Preventive Medicine, Tokyo Medical University, Tokyo, Japan, \(^3\)Department of Anesthesiology, Tokyo Medical University, Tokyo, Japan

**P2.83** Placenta-derived multipotent cells shared the expression of several trophoblast-related genes  
Volodymyr Shablii\(^1\), Maria Kuchma\(^2,\)\(^3\), Hanna Svitina\(^1,\)\(^3\), Inessa Skrypkina\(^1,\)\(^2\), Pavlo Areshkov\(^1,\)\(^2\), Vitaliy Kyryk\(^4\), Yulia Shablii\(^1\), Pavlo Klymenko\(^4\), Lyubov Lukash\(^2\), Galyna Lobintseva\(^1\), \(^1\)Institute of Cell Therapy, Kyiv, Ukraine, \(^2\)Institute of Molecular Biology and Genetics of National Academy of Sciences of Ukraine, Kyiv, Ukraine, \(^3\)Educational and Scientific Centre “Institute of Biology”, Taras Shevchenko National University of Kyiv, Kyiv, Ukraine, \(^4\)State Institute of Genetics and Regenerative Medicine of Academy of Medicine of Ukraine, Kyiv, Ukraine

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**P2.84** Placental adaptive up-regulation of calcium transport in wild-type mice is not driven by parathyroid hormone-related protein (PTHrP)  
Christina Hayward\(^1\), Kirsty McIntyre\(^1\), Colin Sibley\(^1\), Susan Greenwood\(^1\), Mark Dilworth\(^1\), \(^1\)University of Manchester, Manchester, UK

**P2.85** Adaptive up-regulation of placental calcium transport in a mouse model of fetal growth restriction: what are the underpinning mechanisms?  
Christina Hayward\(^1\), Kirsty McIntyre\(^1\), Colin Sibley\(^1\), Susan Greenwood\(^1\), Mark Dilworth\(^1\), \(^1\)University of Manchester, Manchester, UK

**P2.86** The role of the L-type amino acid transporter 1 (LAT1) in placenta pathologies  
Xiao Huang\(^1,\)\(^2\), Jonas Zaugg\(^1,\)\(^2\), Meike Körner\(^3\), Matthias Rubin\(^1,\)\(^2\), Julien Graff\(^2,\)\(^4\), Marc Baumann\(^5\), Daniel Surbek\(^3\), Karl-Heinz Altmann\(^3,\)\(^4\), Christiane Albrecht\(^1,\)\(^2\), \(^1\)Institute of Biochemistry and Molecular Medicine, Faculty of Medicine, University of Bern, Bern, Switzerland, \(^2\)Swiss National Center of Competence in Research, NCCR TransCure, University of Bern, Bern, Switzerland, \(^3\)Pathologie Länggasse, Bern, Switzerland, \(^4\)Department of Chemistry and Applied Biosciences, Swiss Federal Institute of Technology in Zurich, Zurich, Switzerland, \(^5\)Department of Obstetrics and Gynecology, University Hospital, University of Bern, Bern, Switzerland

**P2.87** Expression and function of prostaglandin transporter in the murine placenta  
Mai Inagaki\(^1\), Tomohiro Nishimura\(^1\), Takeo Nakanishi\(^2\), Shin-ichi Akanuma\(^3\), Masanori Tachikawa\(^4\), Ikumi Tamai\(^5\), Ken-ichi Hosoya\(^6\), Emi Nakashima\(^1\), Masatoshi Tomi\(^1\), \(^1\)Faculty of Pharmacy, Keio University, Tokyo, Japan, \(^2\)Faculty of Pharmaceutical Sciences, Kanazawa University, Kanazawa, Japan, \(^3\)Graduate School of Medicine and Pharmaceutical Sciences, University of Toyama, Toyama, Japan, \(^4\)Graduate School of Pharmaceutical Sciences, Tohoku University, Sendai, Japan

**P2.88** Optimization of protocols for decellularization of the mouse placenta  
Patricia Romagnolli\(^1\), Rodrigo da Silva Nunes Barreto\(^1\), Paula Fratini\(^1\), Maria Angelica Miglino\(^0\), \(^1\)School of Veterinary Medicine and Animal Sciences, University of São Paulo, São Paulo, Brazil
P2.89  **Long-chain and very-long-chain fatty acid analogue uptake is greater into cytotrophoblast than into syncytiotrophoblast in vitro.**
Kent Thornburg¹, Amy Valent¹, Kevin Kolahi¹, ¹Oregon Health And Science University, Portland, OR, USA

P2.90  **Maternal choline supplementation during pregnancy improves placental vascularization and modulates placental nutrient supply in a sexually dimorphic manner**
Sze Ting (Cecilia) Kwan¹, Julia King¹, Jian Yan¹, Xinyin Jiang¹,³, Mark Roberson², Marie Caudill¹, ¹Division of Nutritional Sciences, Cornell University, Ithaca, NY, USA, ²Department of Biomedical Sciences, Cornell University, Ithaca, NY, USA, ³Department of Health and Nutrition Sciences, Brooklyn College, Brooklyn, NY, USA

P2.91  **Glucocorticoids modulate expression and function of the multidrug resistance transporters in the 1st trimester human placenta.**
Phetcharawan Lye¹, Lubna Nadeem¹, Jeremy Landry¹, William Gibb¹,⁴, Stephen Lye²,⁵, Stephen Matthews¹,², ¹Department of Physiology, University of Toronto, Toronto, Canada, ²Lunenfeld-Tanenbaum Research Institute, Toronto, Canada, ³Department of Obstetrics and Gynaecology, University of Ottawa, Ottawa, Canada, ⁴Department of Cellular and Molecular Medicine, University of Ottawa, Ottawa, Canada, ⁵Department of Obstetrics and Gynaecology, University of Toronto, Toronto, Canada

P2.92  **Placental transfer (clearance) of glutamine is dependent upon placental size in mice**
Kirsty McIntyre¹, Christina Hayward¹, Colin Sibley¹, Susan Greenwood¹, Mark Dilworth¹, ¹University of Manchester, Manchester, UK

P2.93  **Is the placenta the source of fetal glycine and glutamine? A human in vivo study**
Maia Blomhoff Holm¹, Ane Moe Holme¹, Hildegunn Horne¹, Tore Henriksen¹,², Trond Michelsen¹,², ¹Department of Obstetrics Rikshospitalet, Women’s Division, Oslo University Hospital, Oslo, Norway, ²University of Oslo, Oslo, Norway, ³Norwegian National Advisory Unit for Women’s Health, Oslo University Hospital, Oslo, Norway

P2.94  **On the oxygen transport of red blood cells in the feto-placental vasculature system of the mouse placenta**
Parisa Mirbod¹, Zhenxing Wu¹, ¹Clarkson University, Potsdam, NY, USA

P2.95  **Placental tryptophan metabolism and serotonin transport are disrupted in idiopathic human fetal growth restriction**
Padma Murthi¹,², Stacey Ellery²,³, Hayley Dickinson²,³, David Walker²,³, Euan Wallace²,³, Peter Ebeling¹, ¹Department of Medicine, Monash University, Clayton, Victoria, Australia, ²The Ritchie Centre, Hudson Institute of Medical Research, Clayton, Victoria, Australia, ³Department of Obstetrics and Gynaecology, Monash University, Clayton, Victoria, Australia

P2.96  **Pravastatin transport by human placental plasma membrane vesicles**
Tatiana Nanovskaya¹, Svetlana Patteryeva¹, Rabab Al Lahham¹, Mahmoud Ahmed¹, ¹University of Texas Medical Branch, Galveston, TX, USA
POSTER SESSIONS

P2.97  **Fatty acid transporter expression is decreased in placental macrovascular endothelial cells, not trophoblasts, in obese women**  
Xiaohua Yang1, Patricia Glazebrook1, Maricela Haghiac1, Judi Minium1, Sylvie Hauguel deMouzon1, Perrie O’Tierney-Ginn1, 1Case Western Reserve University, Cleveland, OH, USA

P2.98  **Transfer of ampicillin and cefotaxime through the placenta**  
Jana Pastuschek1, Andreas Butans2, Daniela Remane2, Frank T. Peters2, Udo R. Markert1, Ekkehard Schleußner1, Tanja Groten1, 1Placenta-Lab, Department of Obstetrics, University Hospital Jena, Jena, Germany, 2Institut of Legal Medicine, Department of Toxicology, University Hospital Jena, Jena, Germany

P2.99  **SNAT1 predominantly contributes to system A function in placental microvillous membranes**  
Yu Takahashi1, Tomohiro Nishimura1, Tetsuo Maruyama1, Emi Nakashima1, Masatoshi Tomi1, 1Faculty of Pharmacy, Keio University, Tokyo, Japan, 2School of Medicine, Keio University, Tokyo, Japan

P2.100  **Role of organic anion transporter 4 on the transport of olmesartan across the basal plasma membrane of human placental syncytiotrophoblast**  
Masatoshi Tomi1, Saki Noguchi1, Ayasa Fujibayashi1, Tetsuo Maruyama1, Emi Nakashima1, Tomohiro Nishimura1, 1Faculty of Pharmacy, Keio University, Tokyo, Japan

LATE BREAKING POSTERS

P2.101  **A light shines through the trees: fluid-filled bulb formation from placental villous explants in long-term culture**  
Yaqi Zhao1, Lance Davidson1, Simon Watkins1, Morgan Jessup1, Dale Lewis1, Zsolt Urban1, Jennifer Adibi1, 1University of Pittsburgh, Pittsburgh, PA, USA

P2.102  **Characteristic changes in decidual gene expression signature in spontaneous term parturition**  
Haidy El-Azzamy1, Andrea Balogh1,2, Roberto Romero1,2, Yi Xu1, Christopher LaJeunesse1, Olesya Plazyo1, Zhonghui Xu1, Theodore Price1, Zhong Dong1, Adi Tarca1,2, Zoltan Papp1, Sonia Hassan1,2, Tinnakorn Chaiworapongsa1,2, Chong Jai Kim1,2, Nardhy Gomez-Lopez1,2, Nandor Gabor Than1,2, 1Perinatology Research Branch, NICHD/NIH/DHHS, Detroit, MI, USA, 2Eotvos Lorand University, Budapest, Hungary, 3Wayne State University, Detroit, MI, USA, 4Maternity Clinic, Semmelweis University, Budapest, Hungary, 5University of Ulsan, Seoul, Republic of Korea, 6Research Centre for Natural Sciences, Hungarian Academy of Sciences, Budapest, Hungary

P2.103  **Genome-wide characterization of transposable elements activity during placenta development**  
Guillaume Cornelis1, Julie Baker1, 1Stanford University, Stanford, CA, USA

P2.104  **Multiplexed genotyping and allele-specific expression analysis verifies human placental imprinted gene expression is established early and is stable across gestation**  
Jill Reiter1, Xiaoling Xuei1, Hitesh Appaiah1, Howard Edenberg1, David Haas1, Men-Jean Lee1, 1Indiana University School of Medicine, Indianapolis, IN, USA, 2Beth Israel Medical Center, New York, NY, USA
P2.105 Transcriptomic profiling of invasive trophoblast cells within the hemochorial placentation site
Regan Scott1, Lisa Neums1, Masanaga Muto1, Damayanti Chakraborty1, Jeremy Chien1, Michael Soares1,
1University of Kansas Medical Center, Kansas City, Kansas, USA

P2.106 Effects of Chromium-VI toxicity on placental antioxidants and VEGF pathway.
Sakhila Banu1, Joe Arosh1, Robert Taylor1, Robert Burghardt1, 1Dept of Veterinary Integrative Biosciences,
Texas A&M University, College Station, USA

P2.107 Danger hallmarks of dysregulated complement cascade on placentas from severe early-onset preeclampsia
Manu Banadakoppa1, Kjersti Aagaard1, Chandra Yallampalli1, 1Baylor College of Medicine, Houston, Texas, USA

P2.108 Placental origin of fetal syndrome of endocannabinoid d(FSECD).
Natalia Schlabritz-Lutsevich1, Nadjezhda German1, Gary Ventolini1, Eneko Larumber1, Jacques
Samson1, 1TTUHSC, Odessa, TX, USA, 2TTUHSC, Amarillo, TX, USA, 3TTUHSC, Lubbock, TX, USA, 4UTHSC,
Memphis, TN, USA
Inverted Formin-2: a novel gene essential for female blood pressure regulation, placental development and fetal growth
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OBJECTIVES
Our new findings demonstrated the loss of inverted formin-2 (INF2), an important cytoskeletal modulator identified in renal and neuropathic disorders, increased gestation length in mice and altered pulsatility index and end diastolic velocity at E18.5. We hypothesize INF2 regulates placental vascular remodelling and its loss results in a preeclamptic phenotype. To determine this, we assessed placental vasculature, fetal growth, and maternal blood pressure (BP).

METHODS
Inf2⁻/⁻ and Inf2⁺/⁺ mice were time-mated. Maternal blood pressure was measured before pregnancy and at E18.5. After C-section, litter size, fetal, and placental weight were recorded. Following fixation, sectioning, and H&E, labyrinth depth and area were measured. Villus vasculature was identified using IHC for endomucin and vessels counted per high power field. In vitro, HTR-8/SVneo cells were transfected with siRNA against INF2 and Placenta growth factor (PlGF) and Human VEGF R1 measured by qPCR and ELISA respectively. Data were analysed by Student T-test and P< 0.05 was deemed significant.

RESULTS
Non-gravid Inf2⁻ females demonstrated increased blood pressure compared to Inf2⁺ females, (86.1±2.7 vs 73.5±3.5mmHg; p=0.03, n=3,3) and this difference was maintained during pregnancy (85.3±9.4 vs 75.9±7.5 mmHg, n=3). Inf2⁻ fetal weight was reduced (1.05±0.02 vs 1.14±0.03g; p=0.05, n>10) with no effect on placental weight. However, labyrinth depth was significantly smaller (672.8±14.7 vs 783.3±23.1μM; p<0.05, n=3,5) and area trended smaller (6735±806 vs 8549±253 mm², n=3,5). Blood vessel numbers in Inf2⁻ placentas were increased showing a trend towards significance (77.06±3.1 vs 64.0±6.0/hpf.; p=0.07 n=3,5). In vitro, loss of INF2 significantly increased both VEGFR1 secretion (254.2±3.1 vs 144.6±15.1pg/mL; p<0.05) and PlGF expression compared to control (2.02±0.2 vs 1.07±0.01; p<0.05).

CONCLUSIONS
In addition to causing renal and neuropathic diseases, INF2 is essential for appropriate placental development. Its loss impacts maternal BP and fetal growth, highlighting its potential as a novel target for therapeutic intervention.
Defects in fetoplacental vascularization and function in EGFL7 KO mice
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**BACKGROUND**
Survival and proper development of the mammalian embryo is dependent on a functional placenta. The placenta provides an interface for the maternal and fetal circulation, facilitating the exchange of nutrients, oxygen and metabolic waste, forming a protective barrier against the maternal immune system, and producing hormones. EGFL7, a secreted protein that binds to the extracellular matrix, is highly expressed in endothelial cells of the embryo, allantois, and placenta during development.

**OBJECTIVE**
To determine the function of Egfl7 during placental development in mice, and to examine EGFL7 a biomarker for preeclampsia in humans.

**RESULTS**
Egfl7 KO mice do not display any overt embryonic vascular phenotypes. However, we uncovered for the first time a crucial role for Egfl7 during development of the placental vasculature. Egfl7 KO concepti exhibit reduced placental weights and vascular patterning defects at E12.5, resulting in an irregular formed fetal vascular plexus with narrowed, poorly perfused fetal capillaries of the labyrinth and fetal growth restriction. These defects become visible as early as E8.5 when the allantois makes contact with and initiates branching morphogenesis of the chorionic plate. The observed phenotypes are accompanied by changes in the expression of genes that are involved in chorionic branching and in genes encoding ECM/EC adhesion proteins. Isolated endothelial cells from Egfl7 KO placentas show reduced cell migration, sprouting and cord formation.

Pregnant Egfl7 KO mice display preeclampsia-like symptoms, including elevated blood pressure in late gestation and increased urinary protein levels at mid-gestation. Our studies show dysregulation of EGFL7 in patients with preeclampsia. We will report on ongoing studies to determine the role of EGFL7 as a novel biomarker for preeclampsia in humans.
**Trabecular Transcriptomics: Comparative Analysis of Spider Monkey and Human Placental Transcriptomes**

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**INTRODUCTION**

The placenta of the New World monkey *Ateles fusciceps* (spider monkey) is of a trabecular hemochorial nature, and may represent a transition between the labyrinthine placenta seen in rodents and the villous placenta observed in humans. Understanding the molecular basis of placental function from an evolutionary perspective, particularly in nonhuman primates, may provide insight into the development of placental disorders leading to obstetrical syndromes in the human.

**OBJECTIVE**

To identify differences in placental gene expression between *A. fusciceps* and *H. sapiens*.

**METHODS**

RNA sequencing (RNA-seq) was performed using the Illumina Genome Analyzer to generate placental transcriptomic profiles for *A. fusciceps* (n=1) and *H. sapiens* (n=26). The transcriptome of *A. fusciceps* was aligned to the *Callithrix jacchus* (common marmoset) genome. 1:1 orthologs present between *A. fusciceps* and *H. sapiens* was determined using Ensembl v.80. Changes in placental gene expression between *A. fusciceps* and *H. sapiens* were ranked by *p*-values calculated from the Z-score and corrected for FDR using the Benjamini-Hochberg method. Pathway over-representation analysis was conducted on the top 10% of differentially expressed genes using the GATHER (Gene Annotation Tool to Help Explain Relationships) and InnateDB databases.

**RESULTS**

A total of 12,578 1:1 orthologous genes were expressed in *A. fusciceps*. Of these, 6,873 genes were differentially expressed compared to *H. sapiens*. The top 10% of differentially expressed genes in *A. fusciceps* were significantly enriched (adjusted-*p*<0.01) for genes involved in antigen processing and presentation (GO:0030333). Placental expression of 7 genes including orthologs for human leukocyte antigens HLA-A, -DMA, -DMB, -DOB, and -DRA were increased in *A. fusciceps* compared to *H. sapiens*.

**CONCLUSION**

In human pregnancy, HLA genes play a key role in modulating immune tolerance to the allogenic fetus. Studying nonhuman primate placentas may provide insight into the mechanisms of success or failure in establishing immune tolerance during human pregnancy.
Maternal Insulin and Placental Growth in the Common Marmoset Monkey
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INTRODUCTION
The insulin pathways regulate placental and fetal growth over gestation. The common marmoset is a nonhuman primate with growing significance in studies of the primate placenta and fetal programming. Yet the relationships among maternal insulin across gestation and placental growth are largely unknown.

OBJECTIVES
To preliminarily describe maternal insulin profiles across gestation and examine the relationships among maternal insulin, maternal weight and placental weight.

METHODS
Serum insulin and maternal weight were measured at day 60, 90 and 120 of a 143-day gestation (N=18). Placental weights were collected at birth. Relationships were assessed using Spearman correlations.

PRELIMINARY RESULTS
Maternal insulin levels increased across gestation and were positively associated with maternal weight at each time point. There was a strong positive association between maternal insulin at day 60 and placental weight (R=0.8061, p=0.0049), however there was no significant association between maternal insulin at day 90 or 120 with placental weight.

CONCLUSIONS
Much like humans, early pregnancy is a time of rapid placental growth in marmosets with 50% of placental weight achieved by day 70-80 of gestation. The strong association between maternal insulin at day 60 and placental weight suggests that insulin-dependent processes of placental growth are strongly influenced by maternal insulin in early gestation. The lack of association between maternal insulin at days 90 and 120 and placental weight suggests that insulin-dependent placental processes are less influenced by maternal insulin later in pregnancy. In the human placenta, location and expression of insulin receptors in the placenta shifts from the maternal facing surface in early pregnancy to the fetal facing surface in later pregnancy and thus control of insulin-dependent placental processes shifts from maternal to fetal control. Similar alterations in insulin receptors may occur in the marmoset and suggests that maternal insulin is most influential on placental processes in early pregnancy.
Sex differences in susceptibility to activity based anorexia are mediated by placental miR-340

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OBJECTIVES

Anorexia nervosa (AN) is a devastating eating disorder characterized by self-starvation that mainly affects women. Its etiology is unknown, which handicaps successful treatment options leading to a only 50-60% chance of full recovery. Here, we focused on gestation as a vulnerable window in which environmental factors can influence the predisposition to AN.

METHODS

We used placenta-specific lentiviral transgenes and embryo transfer, combined with in vitro studies, to demonstrate the key role placental miR-340 plays in the mechanism involved in early life programming of activity based anorexia (ABA).

RESULTS

We identified placental microRNA-340 (miR-340) as a sexually dimorphic regulator involved in prenatal programming of a predisposition to activity-based anorexia (ABA) in adolescent mice.

CONCLUSIONS

Placental miR-340 modifies the uterine environment through its targets, affecting hypothalamic programming in the fetus and causing an abnormal response to food restriction/high activity levels during adolescence.
Definitive non Classical Human Leukocyte Antigen expression in human placentation: HLA-F, E G and C in EVT invasion.

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INTRODUCTION

Human pregnancy is one of the most interesting examples of immune tolerance seen in mammalian biology. The placenta is the single organ simultaneously expressing all non-classical MHC class I antigens HLA-E, F, and G, and C. Still, there is conflicting evidence mainly regarding HLA-F expression by trophoblast. Recently, following our finding of a unique interaction between HLA-F and β2m-associated HLA-C, we have proposed a novel model for a broader interaction between HLA-I open conformers and KIR receptors, for which HLA-F may serve as the prototype. Another complex, HLA-E/G interacts strongly with inhibitory NK receptors. Thus the goal of this work is to define placental HLA-F, G, E and C expression through gestation and to elucidate their contribution to the immune inhibitory response in early placentation.

MATERIAL AND METHODS

Immunohistochemistry, q-PCR, and western blot were used from 5-40wks and from placenta in labour. Placental EVT explants and Swan-71 cells were used to assess HLA-F and HLA-C.

RESULTS

The 3D11 antibody consistently recognized cytoplasmic staining for HLA-F in EVT across pregnancy. Abcam and 4A11 antibodies showed that HLA-F and HLA-C were strongly expressed on the extracellular membranes of EVT (weeks 5-12). Both HLA-F and HLA-C were also strongly expressed by the syncytiotrophoblast in early pregnancy and decreased to term. HLA-E was only expressed in very early EVT before the 7th week of gestation, while HLA-G was expressed throughout pregnancy. Western blots demonstrated increasing levels of a 40kDa form of HLA-F with gestation while a 100kDa dimer of HLA-F was highest in the 1st trimester. HLA-C was highly expressed in villous mesenchyme of placenta in labor.

CONCLUSIONS

1. HLA-F and HLA-C are highly expressed on the cell surface of 1st trimester EVT; the existence of a high weight dimer of HLA-F during this time suggests that a HLA-F/C complex may play an important role in regulating EVT uNK interactions during uterovascular transformation. 2. High levels of HLA-E and HLAG in very early placenta suggest that this complex is essential in the inhibitory NK response during implantation 3. The significant high levels of HLA-C in labor may indicate a role in parturition.
**Uterine NK cell cells (CD56hiCD16-) are outnumbered by CD56loCD16+ NK cells at the uteroplacental interface**

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**OBJECTIVES**

Placental pregnancy is an immunological conundrum: The baby is genetically 50% self (mother) and 50% non-self (father), but not rejected the same way any allogeneic solid organ would be. The main interaction site between mother and baby is the uteroplacental interface (UPI), where the decidua (maternal structure) develops between the uterus and the placenta (fetal structure). Utilizing a novel technique that takes advantage of the standard way surgeons clean the uterus after cesarean section, we isolated lymphocytes specific to the UPI and characterized the natural killer (NK) cells in that population.

**METHODS**

Peripheral blood and lymphocytes from the UPI of 3rd trimester healthy pregnant women were purified and immunostained for analysis by flow cytometry. The major end-point was characterization of different NK subsets and surface molecules.

**RESULTS**

We found increased proportions of CD56hi CD16- NK cells, uterine NK cells, at the UPI compared to the PB of the same woman (CD56hi CD16- PB: mean 1.86 Std Dev 1.2; UPI: mean 19.84 std dev 9.94). Importantly, the uterine NK cells represented a mean of 19.84% of the total lymphocyte population (std dev 9.94) while a mean of 50.53% of the lymphocyte population at the UPI were CD56loCD16+(std dev 10.03). Further, uterine NK cells but not PB NK cells exclusively display an activated (CD69+) phenotype and a significantly higher amount of the apoptosis-inducing molecule Fas (mean PB 21.74 +/- 4.5; mean UPI 43.61 +/- 2.02 p=0.0007).

**CONCLUSIONS**

The immune system at the UPI fulfills a dramatically different role than the peripheral immune system, and the cellular phenotypes in the UPI vs. PB reflect the role of these cells for maintaining tolerance. Increased understanding of the NK cell function at the UPI is essential for filling in our gaps in knowledge about maternal-fetal tolerance, and will likely inform transplant immunology.
A tight infection bottleneck undermined by inadequate early immune responses define the dynamics of decidual listeriosis

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OBJECTIVE

Placental infection with Listeria monocytogenes (LM) is associated with high morbidity/mortality. Although it is known that LM spreads from maternal blood to the decidua prior to the placenta, and that adaptive immunity is compromised within the murine decidua, the pathogenesis of decidual listeriosis remains poorly understood. We assessed the dynamics of LM infection in human explants and the pregnant mouse uterus, with a focus on pathways affecting infectivity independent of adaptive immunity.

METHODS

Human decidua and endometrium explants were infected ex vivo with LM, and pregnant mouse models were used to study the immune response in vivo. Microscopy and flow cytometry were used to examine the localization of bacteria and immune cells.

RESULTS

Human explants showed low initial colonization by LM. Significant bacterial expansion was observed in decidual, but not endometrial explants. Human decidual infection was accompanied by a lack of macrophage aggregation near foci of bacteria. In vivo experiments in the mouse showed that the decidua is a tissue bottleneck, with LM foci predominately near the decidual/myometrial border and a lack of NK cell aggregation near these foci. The infected decidua also showed a decrease in cells important for early defense against LM at other tissue sites, including Ly6Chi monocytes and macrophages, whereas the infected myometrium accumulated Ly6Chi monocytes. Consistent with this observation, the protein levels of inflammatory chemokines, including Monocyte Chemoattractant Protein-1 (MCP-1), were significantly lower in the decidua than the myometrium, despite a higher bacterial burden in the decidua.

CONCLUSION

LM infection of the decidua shows a two-phased response, with restricted access followed by rapid growth and impaired early immunity. In contrast, the myometrial response is more similar to that of other tissues, showing that the innate immune defects are microanatomically restricted to the decidua. Perhaps restriction of initial infection evolved to protect the decidua without compromising fetoplacental homeostasis. Future identification of the host factor responsible for the decidual bottleneck requires consideration beyond traditional innate/adaptive immunity, and exploration of factors such as physical barriers, antimicrobial peptides, or niche availability.
A Pathogenic Role for High Risk Human Papillomavirus in Villitis of Unknown Etiology

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OBJECTIVES

Villitis of unknown etiology (VUE) is attributed to an alloimmune response ¹ ². We recently described the histopathological and obstetric consequences of human papillomavirus infection of the placenta ³, in which a subset of HPV cases demonstrated VUE-like features. The objective of this study is to investigate for the presence of HPV in 103 clinically diagnosed VUE cases.

METHODS

Two VUE cohorts were retrospectively retrieved from the Otago Placenta Study, Dunedin, New Zealand (n=39) and archives of Tokyo Metropolitan Tama Medical Centre, Japan (n=64), which conformed to the histopathological features of VUE. VUE was defined as a patchy mononuclear infiltrate of the villi associated with fibrinoid necrosis in the absence of an identifiable infectious agent, following the examination of at least four histological sections. Ethical approval was obtained from both centres and parents provided signed informed consent. Personalized fetal growth weights according to the gestation network were available for the Otago cohort (http://www.perinatal.org.uk). Estimates of deviation from the mean were calculated according to the gestational age-specific birth physique standard value directed by the Japan Pediatric Society. None of the women had been vaccinated against HPV.

A positive reaction for HPV was determined by HPV L1 immunohistochemistry which detects high-risk types 16, 18, 31, 33, 51, 52, 56 and 58, and low-risk types 6, 11, and 42 (Dako, Glostrup, Denmark and Abcam, Cambridge, UK). In situ hybridization to HPV DNA using the GenPoint HPV DNA Probe cocktail, which identifies high risk HPV types 16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59, and 68, was also performed. Eighty five cases negative for HPV L1 IHC and in-situ hybridization to HPV DNA by these techniques were examined for a histopathological VUE lesion. Chi-square tests were used to compare differences between groups for categorical measures and 1-way ANOVA for continuous measures. Statistical significance is indicated by a two-tailed P-value.
RESULTS

Maternal age was higher for the cases from Japan. Otago cases had higher maternal BMI (both p<0.01). Both cohorts delivered prematurely (35.8-36.6 weeks gestation) compared to the negative HPV cases (both p<0.0001). The non-pre-eclamptic and non-diabetic associated VUE cases had lower mean personalized growth centile in the Otago cohort, compared to the Otago pre-eclampsia associated VUE cases, probably influenced by the smoking rate of 50% in the former. In the Japan cohort, non-pre-eclamptic and non-diabetic associated VUE fetal growth was comparable to the pre-eclampsia associated VUE group, wherein smoking rates were similar. Pre-eclampsia rates were similar in the VUE cohorts at 28.2% and 31.2%. A positive reaction for HPV L1 immunohistochemistry was found in the syncytiotrophoblast and villous stromal cells in the areas of inflammation in 102/103 cases. All of the cases in both the Otago and Japan cohorts (110/110) tested positive for one or more of the high-risk HPV types by in situ hybridization. The single case that was negative for HPV L1 IHC was positive for HPV DNA in situ hybridization.

CONCLUSION

This study provides evidence that high risk type HPV may have a pathogenetic role in VUE. HPV vaccination is a viable preventive measure that may improve fetal growth restriction and prematurity rates.

REFERENCES


**INTRODUCTION**

Tissue metabolic profiles depend on metabolic rate, tissue composition and physiological status. The specific metabolic fingerprint could be used as a unique diagnostic tool. RS is the methodology, which allows an investigation of tissue physiology at the cellular and tissue levels, using photon scattering and has been gaining attention recently as an analytical tool in cancer and reproductive research. Taking in consideration the urgent need to develop non-invasive methods of diagnostic of placental disturbances, the aim of this study was to analyze fetal RS in in vitro model of maternoplacental hypoxia in the ex vivo human dual placental perfusion model.

**MATERIAL AND METHOD**

Fetal venous perfusate was analyzed from a modified perfusion technique to achieve a mean soluble oxygen tension within the intervillous space (IVS) of 5-7% for normoxia (n=5) and <3% for hypoxia (n=6) as described in *Lab Invest. 2014 Aug;94(8):873-80*. In this published work the results showed a significant increase under hypoxia in the levels of different cytokines and markers of oxidative stress, including IL-6, IL-8, TNF-α, IFN-γ, ET-1, malondialdehyde and 8-iso-prostaglandin F2α in maternal venous samples and ET-1 in fetal samples at 360 min. A hand-held Raman instrument (Mira M-1, Metrohm, CA, USA) was used to analyze perfusates within borosilicate glass vials, inserted into vial holder for measurements at ambient temperature. Data was collected using Mira Cal software (Metrohm, CA, USA).

**RESULTS**

We discovered two patterns of placental hypoxia fetal fingerprints: time independent (pattern A, Fig.1, yellow boxes) and time-dependent (Pattern B, Fig.1, red boxes). Patterns in the wave length 24390 nm, 19230 nm, 16666 nm, 11900 nm, 8700 nm, 7400 nm and 6050 nm showed the differences in the form and patterns in the wave length 18200 nm, 11400 nm, 10800 nm, 9090 nm and 6150 nm showed differences in the amplitude.

**DISCUSSION AND CONCLUSION**

The two RS fingerprints patterns represent unique preliminary data, utilizing a potentially new obstetric technology, which could help diagnose the duration of placental hypoxia, ultimately providing novel targets for treatment and prognosis of placental related disorders.
Human trophoblast survival at low oxygen during the first trimester requires MMP2 mediated shedding of HBEGF

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OBJECTIVE

Human trophoblast cells must survive in a low (~2%) O₂ environment during the first ten weeks of pregnancy. In vitro studies have shown that exposure of human trophoblast cells to 2% O₂ dramatically increases heparin-binding EGF-like growth factor (HBEGF) within 4 hours. At low O₂, inhibition of HBEGF downstream signaling prevents its upregulation and increases apoptosis. HBEGF signaling is initiated by metalloproteinase-mediated shedding of its transmembrane HBEGF. To understand how HBEGF shedding is induced at 2% O₂, we hypothesized that a matrix metalloproteinase (MMP) is responsible for HBEGF cleavage.

METHODS

Human first trimester placental villous explants and HTR-8/SVneo trophoblast cells were cultured at 2%, 8% or 20% O₂. MMP antibody arrays were used to screen trophoblast cell extracts. MMP2, HBEGF, HIF1A and HIF2A/EPAS1 were quantified by ELISA. Apoptosis was quantified with a Roche TUNEL kit. RNA was extracted from trophoblast cells for next-generation sequencing (NGS), using a Nugen kit and the Illumina HiSeq-2500 sequencer.

RESULTS

The MMP array showed only MMP2 increasing at 2% O₂, and was confirmed by ELISA in both cell lines and villous explants. HIF1A and HIF2A accumulated at 2% O₂. HBEGF failed to increase when transcription was inhibited by a-amanitin at 2% O₂; however, recombinant MMP2 restored HBEGF upregulation. Of nine HIF-target transcripts identified by NGS from trophoblast cells cultured at 2% O₂ for 0-4 hours, only HSPA6 (HSP70B’) remained elevated after 1 hour. The HSP70 chaperone inhibitor VER155008 blocked upregulation of both MMP2 and HBEGF at 2% O₂, and increased apoptosis. However, both HBEGF upregulation and apoptosis were rescued by exogenous MMP2.

CONCLUSION

We propose that MMP2-mediated shedding of HBEGF, initiated by HSP70, contributes to trophoblast survival in the low O₂ environment encountered during the first trimester, and is essential for successful pregnancy outcomes.
Periconceptional alcohol exposure in the rat reduces maternal blood space volume at mid-gestation, which may lead to perturbed fetal growth.

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OBJECTIVES
We have shown previously that maternal periconceptional alcohol (PC-EtOH) exposure causes fetal growth restriction and sex-specific changes to placental morphology in late gestation. It is currently unknown whether perturbation to placental development mediates this fetal growth restriction.

METHODS
Sprague Dawley dams were administered 12.5% v/v EtOH or a control diet from 4 days prior (E-4) to 4 days after conception (E4) in a liquid diet. On E5 dams were returned to standard chow until sacrifice. An E15 cohort (N=9/treatment) was assessed for fetal and placental weights and dimensions at post-mortem. Stereological analysis quantified volume of the whole placenta, junctional zone and labyrinth compartments, fetal (FBS) and maternal blood spaces (MBS). In situ hybridisation for Mest marked fetal endothelial cells to demarcate the FBS. Fetal sex was also determined.

RESULTS
PC-EtOH exposure resulted in no change to fetal or placental weights, placental length or width, but did reduce placental depth (PTrt<0.01). This was likely attributed to a reduction in labyrinthine wet weight (PTrt=0.05). Stereological analysis showed PC-EtOH did not affect whole placental, labyrinth or junctional zone volumes. But placentas from females had smaller volumes in all compartments when compared to males (PSex<0.05). PC-EtOH caused marked reductions to the MBS of both male and female exposed placentas (PTrt=0.05), without altering the FBS. The FBS was also reduced in placentas of females when compared with males (PSex<0.01).

CONCLUSION
This study has shown that early PC-EtOH exposure during the periconception period can alter placental formation and the development of the maternal blood spaces of the labyrinthine zone. This may lead to alterations in branching of the labyrinth vasculature, blood flow, and nutrient exchange required to sustain fetal growth. This further implicates the placenta as a critical mediator of fetal growth, development and the programming of adult disease.
Transcriptomic analysis of trophoblast cells obtained non-invasively in the first trimester of ongoing pregnancies to investigate severe uteroplacental insufficiency

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OBJECTIVE

The survival and differentiation of extravillous trophoblast (EVT) cells at the beginning of pregnancy is crucial, and deficiencies are linked to uteroplacental insufficiency, which when most severe produces an early pregnancy loss (EPL), and when milder can cause fetal growth restriction or preeclampsia. It is anticipated that EVT gene expression in the first trimester is altered prior to the onset of overt disease in the third trimester. Placental cells obtained from ongoing pregnancies by a novel method were interrogated by next-generation sequencing (NGS) to provide an unbiased approach to determine the effect of EPL on the transcriptome.

METHODS

Trophoblast cells were obtained non-invasively from ongoing pregnancies with either a normal term delivery or an EPL by our published protocol for Trophoblast Retrieval and Isolation from the Cervix (TRIC). RNA was extracted from isolated cells for NGS, using an Epicentre kit and the Illumina HiSeq-2500 sequencer. Differentially expressed genes (DEGs) were validated by qPCR and analyzed using GO pathway and BioPython library programs.

RESULTS

NGS identified ~400 DEGs, comparing fetal and maternal cells from the same cervical specimens. However, no significant DEGs were noted between replicate RNA aliquots, or among fetal cells from nine control patients. Bioinformatics demonstrated that 21 DEGs (10 validated) were characteristic of EVT cells. Comparison of the fetal transcriptomes of the control pregnancies to those from two with EPL revealed over 300 DEGs (13 validated).

CONCLUSION

These finding suggests that high quality RNA can be isolated from cells obtained by TRIC, and used to reliably interrogate the transcriptome of cells that appear to have an EVT phenotype. TRIC and NGS could provide for the first time molecular profiles that reveal early pathophysiological events postulated to occur in EVT cells at the inception of pregnancies that develop obstetric disorders resulting from placental insufficiency.
SATB1 promotion of trophoblast stem cell renewal through regulation of threonine dehydrogenase

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Trophoblast stem (TS) cell renewal and differentiation are essential processes in placentation. We have identified a chromatin organizer/transcription factor called special AT-rich binding protein 1 (SATB1) as a key regulator in the maintenance of TS cell renewal. The mechanism of SATB1 action in TS cells is unknown. In this study, we have investigated SATB1 downstream targets. RNA-seq analysis was performed in Rcho-1 TS cells expressing control or Satb1 shRNAs in order to identify candidate SATB1 targets. Disruption of SATB1 expression had a profound effect on Rcho-1 TS cell gene expression, resulting in the downregulation of 339 transcripts (>1.5 fold) and upregulation of 618 transcripts (>1.5 fold). Among the differentially-regulated transcripts, were downregulated transcripts known to affect the TS cell stem state (Eomes, Cdx2, Id1, Id2, Klf5, Elf3) and upregulated transcripts associated with trophoblast differentiation (Prl3d1, Prl3b1, Tpbpa). Components of WNT/BMP signalling pathways (Wnt3a, Bambi, Bmp4) were downregulated and Cyp11a1 (encoding the rate-limiting enzyme in steroidogenesis) was upregulated in response to SATB1 knockdown.

L-threonine 3-dehydrogenase (Tdh) expression was exquisitely responsive to SATB1 dysregulation. TDH catalyses the conversion of threonine into glycine and acetyl-CoA and has been reported to regulate pluripotency in embryonic stem cells. This prompted an evaluation of TDH in TS cells. Tdh expression was high in the stem state and decreased as trophoblast cells differentiated. Treatment of Rcho-1 TS cells with a TDH inhibitor (Qc1, Millipore) interfered with cell proliferation in a dose-dependent manner. TDH inhibitor treatment also attenuated stem state-associated transcripts and elevated differentiation-association transcripts. The role of TDH on ex vivo blastocyst outgrowth was investigated. TDH inhibitor treatment decreased TS cell colony size and blastocyst outgrowth. Our findings suggest that the actions of SATB1 on TS cell maintenance may be mediated, at least in part, through the regulation of TDH. (Supported by NIH grants: HD020676 and HD079363)
A light shines through the trees: fluid-filled bulb formation from placental villous explants in long-term culture

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OBJECTIVES

Human placental explants, which contain trophoblasts and other cell types, can be a useful tool to study placental function in 3-dimensions (3D). Previous studies have used short-term (< 24h) culture of the whole explant, or long-term culture of outgrowth extravillous trophoblast (EVT) in a monolayer. The objective of the current study is to develop a long-term culture system that retains or mimics the cellular architecture of the placental villi.

METHODS

First trimester normal human placentas (5-13 weeks gestation) were obtained from elective termination of pregnancies. The placental villi were carefully dissected and cultured in Matrigel droplets in DME/H21 medium (supplemented with 10% FBS and antibiotic/antimycotic), at 37 °C under hypoxia conditions (5% CO2 / 5% O2). The morphology and viability of the villous explant and their cell outgrowth were monitored at regular intervals. Endpoints related to size, morphology, cell viability and protein localization were collected using phase contrast and fluorescence confocal microscopy.

RESULTS

We observed outgrowth cells from the villous cell column tips, which then migrated into the Matrigel and self-organized to form a bulb-like 3D structure. This “explant bulb” remained viable in culture for at least 30 days. It consisted of outer layer cells with flat nuclei, inner cells and a fluid-filled cavity. Whole mount immunostaining for Cytokeratin 7 revealed the presence of trophoblast, and other cell types (Cytokeratin 7 negative). Immunostaining for cell-specific markers (trophoblast progenitor cell, fibroblast and endothelial cell) and extracellular matrix molecules are being conducted for in-depth characterization.

CONCLUSION

The placental explant bulb is a promising 3D tissue culture system to study first trimester placenta functions including de novo villous formation and molecule secretion.
Characteristic changes in decidual gene expression signature in spontaneous term parturition

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OBJECTIVES

The decidua has been implicated in the “terminal pathway” of term parturition. However, decidual transcriptomic changes leading to terminal pathway activation have not been systematically explored. We aimed to compare the decidual expression of developmental signaling and inflammation-related genes before and after spontaneous term labor to reveal their involvement in this process.

METHODS

Chorioamniotic membranes were obtained from normal pregnant women who delivered at term with spontaneous labor (TIL, n=14) or without labor (TNL, n=15). Decidual cells were isolated from snap-frozen chorioamniotic membranes with laser microdissection. The expression of 46 genes involved in decidual development, sex steroid and prostaglandin signaling, and pro- and anti-inflammatory pathways was analyzed using qRT-PCR. Membrane sections were immunostained and semi-quantified for five proteins, and immunoassays for three chemokines were performed on maternal plasma samples.

RESULTS

1) Genes with highest decidual expression included IGFBP1, LGALS1 and PAEP; 2) The expression of ESR1, HOXA11, IL1B, IL8, PGRMC2, and PTGES was higher in TIL than in TNL cases; 3) The expression of CCL2, CCL5, LGALS1, LGALS3 and PAEP was lower in TIL than in TNL cases; 4) Immunostaining confirmed qRT-PCR data for IL-8, CCL2, galectin-1, galectin-3, and PAEP; and 5) No correlations between decidual gene expression and maternal plasma protein concentrations of CCL2, CCL5 and IL-8 were found.

CONCLUSION

The increased decidual expression of signaling factors is preceded by the increased expression of chemokines that may stimulate the early recruitment of monocytes. With the initiation of parturition, the decidual expression of anti-inflammatory mediators decreases while pro-inflammatory mediators and steroid receptors increases. This shift in expression affects downstream signaling pathways that may lead to membrane weakening and myometrial contractions. Our results strengthen findings on the decidua being the earliest among gestational tissues primed during parturition and on the concept of the “decidual clock” regulating the timing of birth.
Genome-wide characterization of transposable elements activity during placenta development

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OBJECTIVES

Transposable elements (TEs) have evolved as key components of the development and evolution of placental mammals. Co-evolution of TEs and their mammalian hosts’ genomes resulted in the cooption of specific TEs both as protein coding genes and as regulatory elements essential for placentation. However, the activity of recent TE families that are still competent for transposition is likely to be detrimental, with new insertions disrupting cellular genes. Consequently, active transposition of TEs during placenta development might contribute to the development of placental diseases. In somatic tissues, transposons are mostly silenced by DNA and histone methylation. The hypomethylated status of the placenta compared to somatic tissues is thought to create a permissive environment favoring transposon reactivation. However, the extent of TE activity at a genome-wide level in placental tissues is not known.

METHODS

In order to characterize the level of TE activity in placental tissues, we performed RNA-sequencing of mouse placenta at three developmental stages: post-implantation, mid-gestation and late-gestation.

RESULTS

Most transposons show no or very limited expression during gestation, suggesting placenta specific mechanisms restricting their activity during gestation. However, one TE family, the IAP family, shows high and sustained level of expression during placenta development. IAPs are expressed in trophoblast stem cells (TSCs) in vitro, at levels similar to those of in vivo placental tissues, but IAP transposition is restricted in TSCs, again suggesting mechanisms regulating the post-transcriptional activity of these highly mutagenic elements.

CONCLUSION

In conclusion, although the placenta is hypomethylated, transposon activity is restricted in placental tissues either at the transcriptional or post-transcriptional level. However, the placenta allows the expression of specific transposons, including the IAP family. Future work will focus on the regulation as well as the functional characterization of these mobile elements inducing rapid evolution of the mammalian placenta and potentially favoring significant pathologies associated with this organ.
Multiplexed Genotyping and Allele-Specific Expression Analysis Verifies Human Placental Imprinted Gene Expression Is Established Early and Is Stable Across Gestation

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OBJECTIVES
Genomic imprinting is an epigenetic mechanism of gene regulation that results in parent-of-origin-specific monoallelic expression. First trimester human placentas have been reported to show biallelic expression of some imprinted genes that are monoallelic at term delivery, suggesting that silencing of the imprinted allele might be developmentally regulated across gestation. Our objective was to assess the extent that biallelic expression of imprinted genes occurred in first trimester and term human placentas.

METHODS
We developed a multiplexed assay to both genotype and quantify allele-specific expression (ASE) to verify the imprinted status of known and putative imprinted genes. First trimester (N=22) and term placentas (N=56) were obtained from uncomplicated pregnancies. DNA and RNA were isolated from the same homogenates and isolations were repeated from two sampling sites. Placenta ASE and placenta and maternal genotypes were determined for 23 SNPs representing 19 genes in 10-plexes using the Sequenom MassArray system.

RESULTS
We verified that 11 imprinted genes showed monoallelic expression (DLK1, H19, IGF2, KCNQ1OT1, MEG3, MEST, PEG3, PEG10, PHLDA2, PLAGL1, SNRPN), 3 genes showed skewed ASE in a parent-of-origin-specific manner (KCNQ1, PHACTR2, SLC22A18), and 5 genes showed biallelic expression (CD44, EPS15, SLC22A3, STX11, TP73) in both first trimester and term placentas. Genotyping 20 SNPs in each sample revealed that maternal contamination was more common in first trimester (8/20) compared to term placentas (3/56). Possible loss of imprinting (LOI) was observed for H19 in four first trimester and one term placenta, but without reciprocal LOI for IGF2. SNRPN LOI was also observed in two first trimester and one term placenta. Nevertheless, average LOI allele ratios (84:16) indicated that the imprinted allele remained largely repressed.

CONCLUSION
Our results indicate that LOI occurs infrequently in human placenta and that imprinted gene expression is established early and remains stable through gestation. Maternal cell contamination of placental samples was readily detected and could be mistaken for LOI.
Transcriptomic profiling of invasive trophoblast cells within the hemochorial placentation site

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The hemochorial placenta is composed of specialized trophoblast cell types that interact with uterine spiral arteries. These trophoblast cells are endowed with invasive properties allowing them to exit the developing placenta and direct changes in the uterine vasculature that promote the effective delivery of nutrients to the placenta. Furthermore, this migratory trophoblast cell population is undermined in diseases of pregnancy such as preeclampsia, intrauterine growth restriction, and early pregnancy loss. However, their location within the uterine wall precludes their routine analysis in human placentation sites. The rat exhibits deep intrauterine trophoblast invasion and represents an effective animal model for investigating the biology of trophoblast invasion and uterine spiral artery remodelling. The purpose of this investigation was to profile the transcriptome of invasive trophoblast cells in the rat. Transgenic male rats constitutively expressing enhanced green fluorescent protein (GFP) were mated with wild type female rats. On gestation day 18.5, placenta sites were dissected into metrial gland (site of intrauterine trophoblast invasion) and junctional zone (site of invasive trophoblast progenitors and other differentiated trophoblast lineages) compartments. GFP-positive tissues were enzymatically dissociated and GFP-positive trophoblast cells collected by flow cytometry and cell sorting. RNA was isolated from the GFP-positive cells, libraries generated, and sequenced to generate 100 bp paired-end reads. Bioinformatic and pathway analyses were performed. Transcript signatures consistent with cellular movement, inflammatory response, and cellular development were identified. Transcript expression profiles were validated by qRT-PCR on independent metrial gland and junctional zone compartment samples. Striking differences in metrial gland versus junctional zone transcript profiles for specific cell signalling pathways (Igf, Notch, Wnt, Hgf, Prl family, chemokine) and transcription factors (Maf, Irf5, Zeb2) were observed. The dataset provides a platform to uncover candidate regulators for in vivo investigation of pathways controlling the invasive trophoblast lineage. (Supported by NIH grants: HD020676 and HD079363)
Effects of Chromium-VI toxicity on placental antioxidants and VEGF pathway.

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Environmental contamination with hexavalent chromium (CrVI) is a growing problem both in the U.S and developing countries. CrVI is a heavy-metal endocrine disruptor. Women working in Cr industries exhibit an increased incidence of premature abortion and infertility accompanied by increased oxidative stress. The placentas of all species contain a population of trophoblast cells, which possess a barrier function. These cells regulate molecular and cellular transit between maternal and fetal compartments. Increased uterine vascular resistance and reduced uterine blood flow can be used as predictors of high risk pregnancies and are associated with fetal growth retardation. The rates of placental blood flow, in turn, are dependent on placental vascularization, and placental angiogenesis is therefore critical for the successful development of viable, healthy offspring. The current study was designed to understand the mechanism of CrVI toxicity on angiogenesis mediated through the VEGF pathway. Pregnant mothers were treated with or without CrVI (50 ppm K₂Cr₂O₇) through drinking water from gestational day (GD) 9.5 – 14.5, and placentas were analyzed on GD 18.5. Results indicated that CrVI decreased GPx1, catalase, Prdx3 and Txn2 in metrial gland (MG), labyrinth zone (LZ), and junctional zone (JZ). Whereas CrVI increased SOD2 in MG and decreased SOD2 in LZ and JZ. CrVI also spatio-temporally modulated VEGFR1, VEGFR2 and VEGFR3, as well as VEGFR-signaling pathway machinery in MG, LZ and JZ compartments. Thus our data suggest that CrVI disrupts placental VEGF-signaling and antioxidant system in a spatio-temporal manner. This work was supported by National Institute of Environmental Health Sciences (NIEHS) grant ES025234-01A1 (S.K.B.)
Danger hallmarks of dysregulated complement cascade on placentas from severe early-onset preeclampsia

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Preeclampsia (PE) is considered to be of maternal and placental origins. Placental PE typically has an early-onset (before 34 weeks), whereas maternal PE develops at or after 34 weeks. Early-onset PE is associated with placental dysfunction, and adverse maternal and neonatal outcomes. Complement (C) cascade activation and antiangiogenic milieu have been implicated in pathogenesis of PE. We hypothesized that C is dysregulated on placenta in severe early-onset preeclampsia (SPE) due to decreased cell surface C inhibitory proteins (CIPs) leading to the deposition of higher amounts of C products and production of elevated antiangiogenic molecules in placental trophoblast cells. We measured C activation products C3b, C4b, terminal membrane attack complex (MAC) and CIPs (CD55, CD46 and CD59) by Western blot, and sFlt1 mRNA by qPCR from 10 SPE and 9 matched unaffected placentas obtained from Perinatal data and specimen repository (PERIBANK) of Baylor College of Medicine. We also assessed if C activation will lead to higher amounts of C product deposition and elevated sFlt1 mRNA levels in HTR-8/SVneo cells in which endogenous CD46 was knocked out. Significantly \((p=0.02)\) higher amounts of C3b and MAC, moderate decreases in CD55 \((p=0.053)\) and CD59 levels \((p=0.078)\) and higher sflt1 mRNA levels \((p=0.03)\) were observed in SPE compared to unaffected placentas. Upon C activation, higher amounts of C3b and MAC were deposited and sFlt1 mRNA levels were also increased significantly \((p=0.006)\) in CD46 knockout HTR cells compared to CD46 intact HTR cells. Thus C is dysregulated with increased sFlt1 mRNA levels in SPE placentas, partially due to decreased expression of CIPs. Moreover, knock out of CD46 in trophoblast cells can lead to over amplification of activated C with increases in sflt1 mRNA levels. These results suggested that deficiency of CIPs on trophoblast cells can cause hyper-activation of C and produce antiangiogenic milieu.
**LATE BREAKING ABSTRACTS**

**P2.108**

**Placental origin of fetal syndrome of endocannabinoid deficiency (FSECD).**

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**INTRODUCTION**

The epidemic of obesity, with more than 64% of women being overweight or obese, has been associated with conditions in later life such as mental disorders, diabetes, asthma, and Irritable Bowel Syndrome. Interestingly, these diseases were classified a decade ago as Clinical Syndrome of Endocannabinoid Deficiency (CECD), which was first described by Russo in 2004. We hypothesize that the deteriorating effect of maternal obesity on offspring health is explained by the mechanism of Fetal Syndrome of Endocannabinoid Deficiency (FSECD).

**MATERIAL AND METHODS**

Patient’s population was IRB-approved protocol (UTHSC). All data were summarized as mean ± SEM, categorized by MO (obese (N=4) vs. non-obese (N=5)) Kruskal-Wallis ranks tests were used to assess the differences between groups. Cohen's d was used as a standardized estimate of effect size. Significance level was set at 0.05.

**RESULTS**

Maternal and fetal 2-AG concentrations in women carrying male fetuses were decreased. Although no statistically significant differences were shown in ECS using nonparametric tests, large effect sizes were calculated for maternal 2-AG (d = 0.83), maternal AEA (d = -1.50), and fetal 2-AG (d = 1.16). These differences were even larger for maternal/fetal 2-AG ratio (d = 3.36). However, small effect size (d = -0.15) was found for fetal AEA. Based on maternal 2-AG effect size, assuming a significance level of 0.05 and power 0.80, the samples from 48 individuals (24 obese and 24 non-obese) would be required to detect differences between the two groups within one sex using Student’s t-test.

**CONCLUSION**

The decrease in 2-AG is in agreement with our previous observations of decreased placental 2-AG concentrations in the baboon model of obesity and decreased ECS receptors expression in maternal obesity. Fetal Syndrome of Endocannabinoid Deficiency (FSECD) might be considered as a clinical diagnosis in maternal obesity.
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