Obstetrics & Gynaecology
UNIVERSITY OF TORONTO

27th ANNUAL RESEARCH DAY & Henderson Lecture

FRIDAY, MAY 7, 2010

8:00 a.m. to 6:30 p.m.
Northrop Frye Hall, Ground Floor
Victoria University, 73 Queen's Park Crescent East
University of Toronto M5S 1K7

Lecturer: Jane Norman MD

Professor of Maternal and Fetal Health,
University of Edinburgh, UK
Co-Director, Edinburgh Tommy's Centre for Maternal and Fetal Health Research

Topic: Being Born Too Soon – Do Obstetricians Have Anything To Offer?

Abstract deadline: Friday, March 5, 2010

http://www.obgyn.utoronto.ca/Research/ResearchDay.htm

For additional information or assistance, please contact Helen Robson at helen.robson@utoronto.ca

PROGRAMME-AT-A-GLANCE
(A.M.)
Department of Obstetrics & Gynaecology
27th Annual Research Day, Friday, May 7, 2010
Northrop Frye Hall, Victoria University, University of Toronto,
73 Queen's Park Crescent

NF=Northrop Frye Hall **Burwash Hall is located at the north end of the Victoria Quad**

7:30 a.m. on	**Poster Set-up for Poster Session I** [NF, ground floor, Rms. 004, 006, 007 & 008]
8:00 a.m.	**Registration & Continental Breakfast** [NF, ground floor lobby]
8:25 – 8:30 a.m.	**Welcome: Dr. Alan Bocking, Chair** [NF, ground floor Lecture Hall, Rm. 003]
8:30 – 9:45 a.m.	**Oral Session I (O1-O5)** [NF, ground floor Lecture Hall, Rm. 003]
9:45 – 10:05 a.m.	**Coffee Break & Poster Session I Walkabout** [NF, ground floor lobby; Rms. 004, 006, 007 & 008]
10:05 – 11:05 a.m.	**Poster Session I Tour** [NF, Rms. 004, 006, 007 & 008] **Groups A-F** **Poster Takedown for a.m. session**
Until 2:50 p.m.	**Poster Set-up for p.m. session** [NF, Rms. 004, 006, 007 & 008]
11:10 – 12:10	**Oral Session II (O6-O9)** [NF, ground floor Lecture Hall, Rm. 003]
12:15 – 1:15 p.m.	**Lunch** [Dining Room, Burwash Hall]

DRAFT PROGRAMME-AT-A-GLANCE
(P.M.)

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1:20 – 2:50 p.m.	**Oral Session III (O10-O15)** [NF, ground floor Lecture Hall, Rm. 003]
2:50 **–** 3:10 p.m.	**Coffee Break & Poster Session II Walkabout** [NF, ground floor lobby, Rms. 004, 006, 007 & 008]
3:10 – 4:10 p.m.	**Poster Session II** [NF, Rms. 004, 006, 007 & 008] **Groups G-L**
	Poster Takedown
4:15 – 5:15 p.m.	**Henderson Lecture** [NF, ground floor Lecture Hall, Rm. 003] **Jane Norman MD** Professor of Maternal and Fetal Health, University of Edinburgh, UK Co-Director, Edinburgh Tommy's Centre for Maternal and Fetal Health Research **Topic: "Being born too soon – do obstetricians have anything to offer?"**
5:15 – 5:20 p.m.	**Closing Remarks: Dr. Alan Bocking** [NF, ground floor Lecture Hall, Rm. 003]
5:25 – 6:30 p.m.	**Wine & Cheese Reception** and **JW Knox Ritchie Research Awards & Papsin Award Presentations** [Dining Room, Burwash Hall]

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UNIVERSITY OF TORONTO

27th Annual

RESEARCH DAY

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University of Toronto

HENDERSON LECTURE: 4:15 p.m. – 5:15 p.m.
"Being born too soon – do obstetricians have anything to offer?"
Dr. Jane Norman
Professor of Maternal and Fetal Health, University of Edinburgh, UK
Co-Director, Edinburgh Tommy's Centre for Maternal and Fetal Health Research

WINE & CHEESE RECEPTION: 5:25 – 6:30 P.M.

Obstetrics & Gynaecology
UNIVERSITY OF TORONTO

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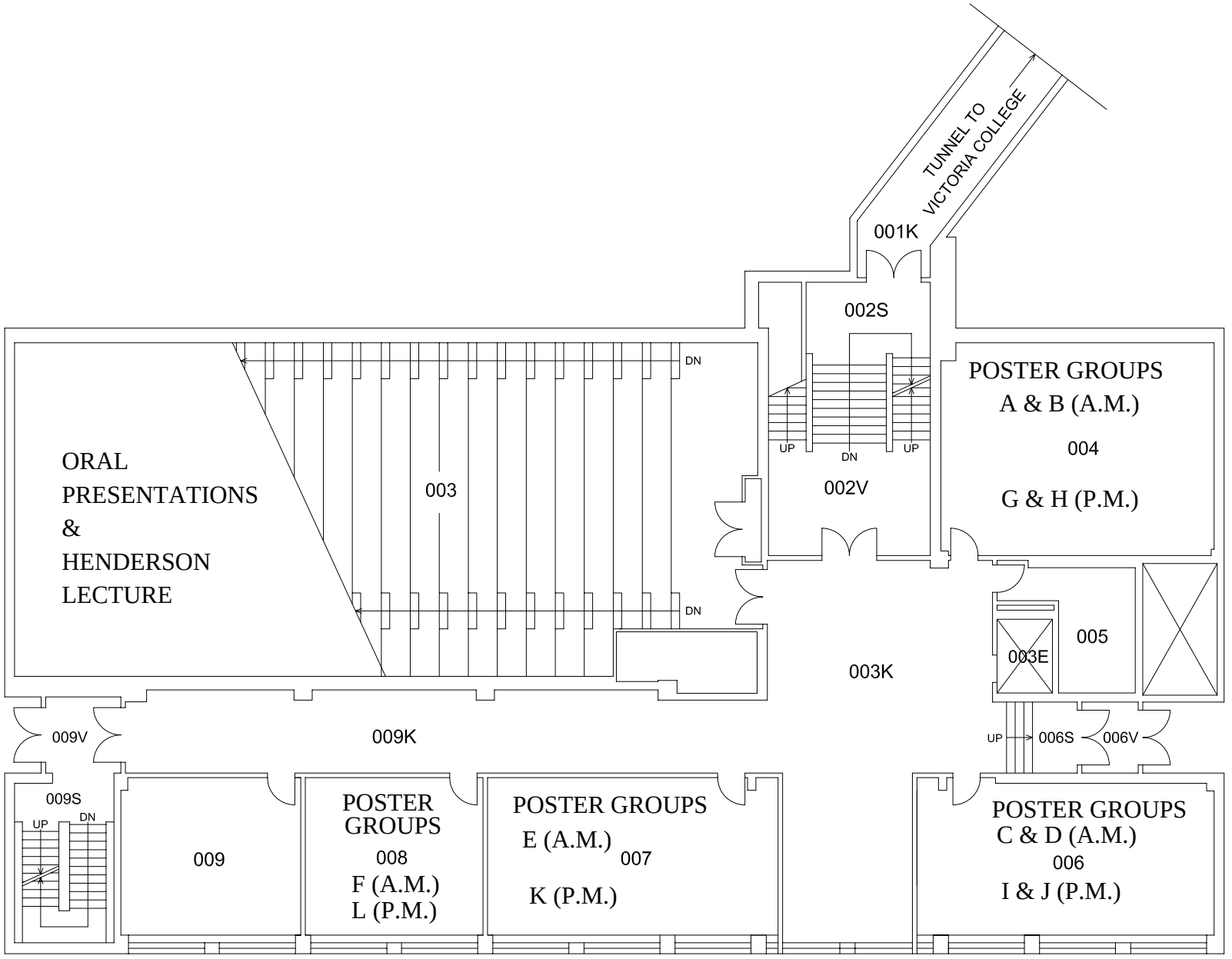
27th Annual Research Day.

Obstetrics & Gynaecology
UNIVERSITY OF TORONTO

27th ANNUAL RESEARCH DAY
Friday May 7, 2010

TABLE OF CONTENTS



University of Toronto

Campus & Facilities Planning

Northrop Frye Hall

Bldg #515

Ground Floor

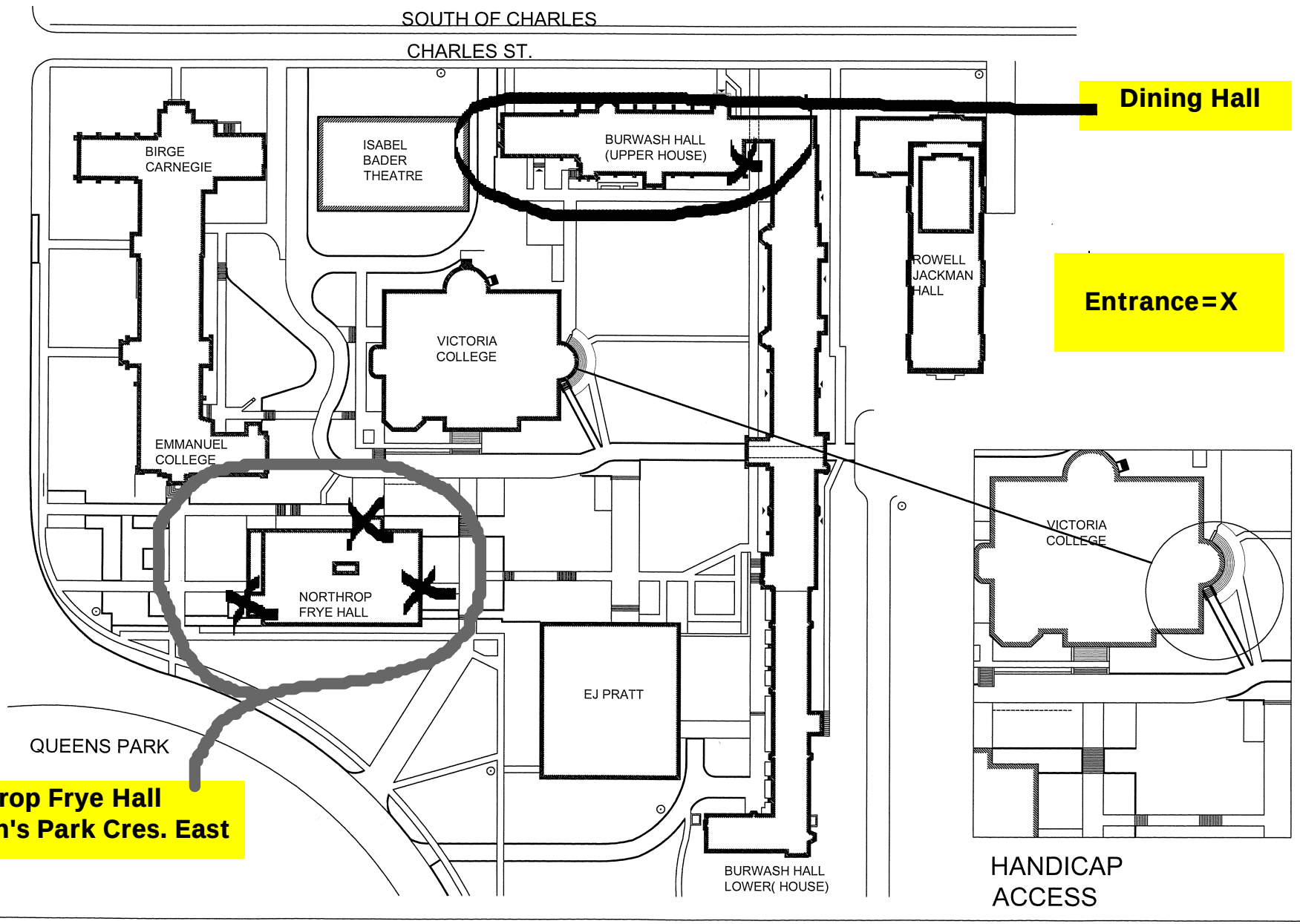
06/10

Revisions

DWG. #

2 of 5

VICTORIA UNIVERSITY



Dining Hall

Entrance = X

Northrop Frye Hall
73 Queen's Park Cres. East

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Groups G-L
Group: Chairs/Judges; Judge
G: Drs. Isabella Caniggia & Clifford Librach; Drs. Ellen Greenblatt & Robert Casper
H: Drs. Andrea Jurisicova & Kellie Murphy; Dr. Richard Pittini
I: Drs. Theodore J Brown & Allan Covens; Dr. Jason Dodge
J: Drs. John Kingdom & Rose Kung; Dr. Kimberly Liu
K: Drs. Wendy Whittle & Mark Yudin; Dr. Michael Shier
L: Drs. Adrian Brown & Prati Sharma; Dr. Cynthia Maxwell

Poster Takedown

4:15 – 5:15 p.m. **Henderson Lecture** [NF, ground floor Lecture Hall, Rm. 003]
Dr. Jane Norman
Professor of Maternal and Fetal Health,
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PROGRAMME

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8:30 – 9:45 a.m. **ORAL SESSION I**

Oral Session I (O1-O5) (5 presentations @15 minutes: 10 minute presentation + 5 minutes for questions) [NF, ground floor Lecture Hall, Rm. 003]
Chair/Judge: Dr. Theodore J Brown
Judges: Drs. Isabella Caniggia & Rose Kung

8:30-8:45 **O1 EFFECT OF REDUCED PLACENTAL EXPRESSION OF GLIAL CELL MISSING-1 ON PREGNANCY OUTCOME.**
Shannon A Bainbridge[PD](1), Kathie J Whiteley (1), Dawei Qu (1), John CP Kingdom (1,2) and S Lee Adamson (1,2,3).
(1) Samuel Lunenfeld Research Institute, Mount Sinai Hospital, (2) Department of Obstetrics & Gynaecology, Mount Sinai Hospital, (2) Department of Physiology, University of Toronto.

8:45-9:00 **O2 CORTICOSTEROIDS INCREASE P-GLYCOPROTEIN FUNCTION IN PRIMARY GUINEA PIG BRAIN ENDOTHELIAL CELLS**
Majid Iqbal[G](1),William Gibb(4),Stephen G Matthews(1,2,3 (1) Departments of (1)Physiology, (2)Obstetrics and Gynaecology and (3)Medicine, Faculty of Medicine, University of Toronto, Medical, Ontario, Canada. Department of Obstetrics and

9:00-9:15 **O3 THE "DUC" TRIAL: A PILOT RANDOMIZED CONTROLLED TRIAL OF IMMEDIATE VS. DELAYED CORD CLAMPING IN PRETERM INFANTS BORN BETWEEN 24 AND 32 WEEKS' GESTATION**

Kelly S Chu [R](1), Kellie E Murphy(1,3), Wendy L Whittle(1,3), Rory Windrim(1,3), Prakesh Shah(2,4)
University of Toronto, Department of Obstetrics/Gynaecology (1), University of Toronto, Department of Paediatrics (2), Department of Maternal-Fetal Medicine, Mount Sinai Hospital (3), Department of Paediatrics, Mount Sinai Hospital (4).

9:15-9:30 **O4 EXPRESSION OF ENZYMES REGULATING EXTRACELLULAR MATRIX BIOGENESIS IN VAGINAL TISSUE OF WOMEN WITH AND WITHOUT PELVIC ORGAN PROLAPSE**

Maria Bortolini [F](1,4), Oksana Shynlova (1), Nadiya Oleksiv(1), Harold Drutz, (2,4), Stephen Lye (1,2,3), May Alarab (1,4)
(1)Samuel Lunenfeld Research Institute, Mount Sinai Hospital, (2)Ob/Gyn;(3)Physiology and (4)Urogynaecology, Mount Sinai Hospital, University of Toronto.

9:30-9:45 **O5 DAB2 ENHANCES GLUCOCORTICOID RECEPTOR-MEDIATED ANTI-INFLAMMATORY SIGNALLING IN ES2 OVARIAN CANCER CELLS**

Alicia Tone [PD] (1-3), Carl Virtanen (5), Patricia Shaw (2-4) and Theodore J Brown (1,3).
(1)Samuel Lunenfeld Research Institute, Mount Sinai Hospital, Departments of (2)Laboratory Medicine & Pathobiology, and (3)Obstetrics and Gynecology, University of Toronto, and (4)Department of Pathology, and the (5)Microarray Centre, University Health Network.

9:45 – 11:05 a.m. **POSTER SESSION I**

9:45 – 10:05 a.m. **Coffee Break & Poster Session I Walkabout** [NF, ground floor lobby; Rms. 004, 006, 007 & 008]

10:05 – 11:05 a.m. **Poster Session I Tour** (3-5 minute presentation + 5 minutes for questions) [NF, Rms. 004, 006, 007 & 008]
Groups A-F

GROUP A:

Chairs/Judges: Drs. Stephane Laframboise & Stephen Lye
Judge: Dr. Sarah Ferguson

P-A1 INVESTIGATING BIOMARKERS FOR THE EARLY DETECTION OF OVARIAN CANCER
Joshua Durbin [G](1,4), Guo Xiong Xu (1), Despina Voulgaraki (1), Thomas Kislinger (4), Theodore Brown (2,3), Michelle Letarte (1,3,4)(1)Molecular Structure and Function, Hospital for Sick Children, Toronto, ON, Canada, (2)Department of Physiology, University of Toronto, Toronto, ON, Canada, (3)Department of Obstetrics and Gynecology, University of Toronto, Toronto, ON, Canada, (4)Department of Medical Biophysics, University of Toronto, Toronto, ON, Canada

P-A2 THE OVARIAN MICROENVIRONMENT OVERCOMES THE ANTI-TUMOUR EFFECT OF SPARC ON OVARIAN CANCER PROGRESSION
James Greenaway[PD](1), Anne Koehler(2), Chris McCulloch(2), Jim Petrik(3), Maurice Ringuette(4) and Theodore Brown(1),
(1)Department of Obstetrics & Gynecology, University of Toronto and Samuel Lunenfeld Research Institute, Toronto, (2)Matrix Dynamics, Faculty of Dentistry, University of Toronto, (3)Dept of Biomedical Sciences, University of Guelph, (4)Dept of Cell and Systems Biology, University of Toronto

P-A3 FEMALE ONCOLOGY PATIENTS AND FERTILITY PRESERVATION: ONE FERTILITY CENTRE'S EXPERIENCE
Kaajal Abrol[R](1), Madeline Tonelli(2), Samantha Yee(2), Catherine Dwyer(2), Kimberly Liu(1,2).
(1)Department of Obstetrics and Gynecology, University of Toronto.
(2)Reproductive Biology Unit, Mount Sinai Hospital, Toronto.

P-A4 RISK OF MALIGNANCY INDEX: HOW FEASIBLE IS IT TO CALCULATE THE SCORE?
Hannah Chiu [M] (1), Jason Dodge (2)
(1) Faculty of Medicine, University of Toronto (2) Department of Gynecologic-oncology, Princess Margaret Hospital, University Health Network

P-A5 PREVALENCE OF BRCA1 AND BRCA2 GERM LINE MUTATIONS AMONG WOMEN WITH CARCINOMA OF THE FALLOPIAN TUBE

Danielle Vicus [F](1,3), Amy Finch(1), Ilana Cass(2), Barry Rosen(3), Joan Murphy(3), Isabel Fan(4), Robert Royer(1), John McLaughlin(4,5), Beth Karlan(2), Steven A. Narod(1)
(1)Women's College Research Institute, (2)Cedars Sinai Medical Centre, Los Angeles, California, USA (3)Department of Gynecology Oncology, University Health Network, University of Toronto(4)Samuel Lunenfeld Research Institute, (5)Cancer Care Ontario, Toronto.

P-A6 VEPH1 IS A NOVEL REGULATOR OF TGF-ß SIGNALING IN OVARIAN CANCER CELLS.

Premalatha Shathasivam [G](1,2,3**)**, A. Kollara(1,3), J. Wrana(1,4), and T. J. Brown(1,2,3).(1) Samuel Lunenfeld Research Institute, Mount Sinai Hospital; Departments of (2)Physiology, (3)Obstetrics and Gynecology, and (4)Medical Genetics and Microbiology, University of Toronto.

GROUP B:

Chairs/Judges: Drs. Rory Windrim & Stephen Matthews
Judge: Dr. Evelyn Lambe

P-B1 GLYCOGEN SYNTHESIS EVALUATION IN LIVERS OF C57BL/6J FETAL MALE MICE SUBJECTED TO ANTENATAL DIETARY RESTRICTION

Lauren A Chun[G](1,2), Brian Knight (1,2) Stephen Lye(1,2,3).
(1)Depts. of Physiology and Ob/Gyn, University of Toronto, Toronto, Canada; (2)Samuel Lunenfeld Research Institute, Mount Sinai Hospital, Toronto, (3)Maternal-Fetal Medicine Division, Department of Obstetrics & Gynaecology, Mount Sinai Hospital.

P-B2 PLACENTAL LOCATION AND NEWBORN WEIGHT

Karthika Devarajan [R] (1), Sari Kives (1,2), Joel G Ray (1,2,3)
St. Michael's Hospital, Toronto (1); Obstetrics and Gynecology, Faculty of Medicine, University of Toronto (2); Departments of Medicine, Health Policy Management and Evaluation and the Division of Endocrinology and Metabolism, St. Michael's Hospital, Toronto (3).

P-B3 PERINATAL OUTCOMES IN A PRENATALLY DIAGNOSED GASTROSCHISIS COMPLICATED WITH GASTRIC DILATATION

Malikah Alfaraj(F)(1), Greg Ryan(1), Gareth Seaward(1), Jacob Langer(2), John Kingdom (1)
(1)Maternal-Fetal Medicine Division, Departments of Obstetrics & Gynecology, Mount Sinai Hospital,(2) Pediatric **Surgery, Hospital for Sick Children, University of Toronto.**

P-B4 COMBINATIONS OF FIRST AND SECOND TRIMESTER MATERNAL SERUM BIOCHEMICAL MARKERS AND PREDICTION OF ADVERSE PREGNANCY OUTCOMES: SYSTEMATIC REVIEW AND META-ANALYSIS

Dini Hui [F] (1), Prakeshkumar Shah (1), Kellie Murphy (1), Elizabeth Uleryk (2), John Kingdom (1), Nan Okun (1)
(1) Maternal Fetal Medicine Division, Department of Obstetrics and Gynaecology, University of Toronto (2) Hospital Library & Archives, Hospital for Sick Children, University of Toronto

P-B5 CONGENITAL MEGALOURETHRA: PRENATAL DIAGNOSIS AND POSTNATAL/ AUTOPSY FINDINGS. REPORT ON 9 CASES

Hagai Amsalem[F](1), Brendan Fitzgerald(2), Sarah Keating(2), Greg Ryan(1), Joao L. Pippi Salle(3), Howard Berger(4), Horacio Aiello(5), Otano Lucas(5), Francois Bernier(6), David Chitayat (7) (1)Department of Obstetrics and Gynaecology, Division of Maternal-Fetal Medicine, Mount Sinai Hospital, University of Toronto, (2) Department of Pathology and Laboratory Medicine, Mount Sinai Hospital, University of Toronto, (3)Department of Pediatric Urology, The Hospital for Sick Children, University of Toronto, (4)Department of Obstetrics and Gynaecology, St Michael's Hospital, University of Toronto, (5)Unidad de Medicina Fetal Servicio de Obstetricia, Hospital Italiano de Buenos Aires, (6) Department of Medical Genetics, University of Calgary, Calgary, Alberta, (7) The Prenatal Diagnosis and Medical Genetics Program, Mount Sinai Hospital, University of Toronto.

P-B6 THE ROLE OF TRANVAGINAL EARLY ANATOMY ULTRASOUND IN THE MANAGEMENT OF FETUSES WITH LARGE NUCHAL TRANSLUCENCY

Erika Frasca [M] (1,2), A Toi (3), D Chitayat (4), D Farine (5), K Chong (4), K Fong (3), O Nevo (1). (1) Maternal-Fetal Medicine, Department of Obstetrics and Gynaecology, Sunnybrook Health Sciences Centre, (2) Faculty of Medicine, University of Toronto, (3) Department of Medical Imaging, Mount Sinai Hospital, (4) Prenatal Diagnosis and Medical Genetics Program, Mount Sinai Hospital, (5) Maternal-Fetal Medicine, Department of Obstetrics and Gynaecology, Mount Sinai Hospital, University of Toronto.

GROUP C:

Chairs/Judges: Drs. S Lee Adamson & John Kingdom
Judge: Dr. Rachel Kupets

P-C1 GENERATION OF SOLUBLE FORMS OF ENDOGLIN FOR STRUCTURAL STUDIES

Allison Gregory [G] (1,2,3), Guoxiong Xu (1,2,3), Luca Jovine (4), Michelle Letarte (1,2,3).
(1) Molecular Structure and Function Program, Hospital for Sick Children, Toronto, (2) Immunology Department, University of Toronto, (3) Heart and Stroke Richard Lewar Center of Excellence, (4) Center for Structural Biochemistry, Karolinska Institutet, Sweden.

P-C2 ANGIOGENIC RESPONSE OF THE HUMAN PLACENTA TO HEPARIN: IMPLICATIONS FOR THE PREVENTION OF PRE-ECLAMPSIA?

Mara Sobel [R] (1), Sascha Drewlo (2), John Kingdom (1,2)
(1) Department of Obstetrics & Gynaecology and (2) Samuel Lunenfeld Research Institute, Mount Sinai Hospital, University of Toronto

P-C3 ROLE OF FACTOR INHIBITING HIF-1 (FIH-1) IN THE SELECTIVE REGULATION OF HIF-1 TARGET GENES IN PREECLAMPSIA

Antonella Racano[G](1,2), Tullia Todros (4), Isabella Caniggia (1,2,3).(1) Samuel Lunenfeld Research Institute, Mount Sinai Hospital; Depts. of (2) Physiology & (3) Obstetrics and Gynaecology, University of Toronto; (4) Dept. of Obstetrics and Gynaecology, University of Turin, Turin, Italy.

P-C4 HEPARIN ELEVATES SFLT-1 SECRETION AND IMPAIRS VILLOUS TROPHOBLAST PHYSIOLOGY IN A DOSE-DEPENDENT MANNER: IMPLICATIONS FOR THE PREVENTION OF SEVERE PREECLAMPSIA?

Sascha Drewlo [PD](1), Khrystyna Levytska(1), Dora Baczyk(1), Mara Sobel (2), John Kingdom (1,2).
(1)Samuel Lunenfeld Research Institute, Mount Sinai Hospital, University of Toronto, (2)Obstetrics & Gynaecology, Mount Sinai Hospital, University of Toronto

P-C5 A STORY OF LIFE AND DEATH: THE DUAL ROLE OF MTD/BOK IN TROPHOBLAST CELL FATE

Jocelyn Ray[G](1), Julia Garcia(1), Yuan Wu(1), Tullia Todros(2) Andrea Jurisicova(1), Isabella Caniggia(1).
(1) Departments of Obstetrics/Gynaecology & Physiology, SLRI, Mount Sinai Hospital, University of Toronto, (2) Dept. of Obstetrics and Gynecology, University of Turin, Turin, Italy.

GROUP D:

Chairs/Judges: Drs. Robert Casper & Navid Esfandiari
Judge: Dr. Fay Weisberg

P-D1 FLUORESCENT IN SITU HYBRIDIZATION (FISH) IS AN EFFECTIVE TOOL TO DETECT UNSUSPECTED TRISOMIES IN HYDROPIC GESTATIONS

Raheela Siddiqui [R](1), Kathy Chun (2), Zeina Ghorab (1), Nadia Ismiil (1), Mahmoud Khalifa (1), Sharon Nofech-Mozes (1), Raymond Osborne (3), Reda Saad (1), Christopher Sherman (1), Valérie Dubé (1).
(1) Department of Pathology, Sunnybrook HSC, (2) Genetics Program, North York General Hospital, (3) Division of Gynecologic Oncology, Sunnybrook Health Sciences Centre, University of Toronto

P-D2 UTILIZATION OF FLUORESCENCE IN SITU HYBRIDIZATION (FISH) METHODOLOGY FOR EVALUATION OF NUCLEAR ORGANIZATION IN HUMAN SPERMATOZOA

Sergey I Moskovtsev[O](1, 2), Naazish Alladin(1), Shlomit Kenigsberg(1), J. Brendan M. Mullen(2), Clifford L Librach(1, 3)
(1)CReATe Fertility Center, (2)Department of Pathology and Laboratory Medicine, Mount Sinai Hospital, (3)Division of Reproductive Endocrinology and Infertility, Department of Obstetrics and Gynaecology, Sunnybrook Health Sciences Centre.

P-D3 MODULATION OF NUCLEAR FACTOR KAPPA β SIGNALING BY NALP5 (Work in Progress)

Taline Naranian [G](1), Tong ZhiBin(2), Lawrence Nelson(2) and Dr. Andrea Jurisicova(1,3).
(1) Department of Physiology, University of Toronto, (2) Developmental Endocrinology Branch, National Institute of Child Health and Human Development, (3) Department of Obstetrics & Gynaecology, Mount Sinai Hospital

P-D4 CHARACTERIZATION OF THE OOCYTE PHENOTYPE CAUSED BY MCL-1 DEFICIENCY

Shakib Omari[G](1), Andrea Jurisicova(1,2)**.**
(1) Department of Physiology, University of Toronto, (2) Department of Obstetrics and Gynaecology, Samuel Lunenfeld Research Institute, Mount Sinai Hospital

P-D5 TAP73 KNOCKOUT MICE AS A MODEL OF REPRODUCTIVE AGING

Tetyana Yavorska [G](1,2), Tak W Mak (3), Andrea Jurisicova (1,2,4).
(1)Department of Physiology, University of Toronto, (2) Samuel Lunenfeld Research Institute, Mount Sinai Hospital, (3) The Campbell Family Institute, Ontario Cancer Institute, University Health Network, (4) Division of Reproductive Endocrinology and Infertility, Department of Obstetrics & Gynecology, University of Toronto.

GROUP E:

Chairs/Judges: Drs. Ori Nevo & Theodore J Brown
Judge: Dr. Andrea Lausman

P-E1 IN VITRO DERIVATION OF FUNCTIONAL INSULIN-PRODUCING CELLS FROM HUMAN FIRST TRIMESTER UMBILICAL CORD STEM CELLS
Rong Xiao[PD], Shang-mian Yie, Junhai Zhao, Wei Gong, Clifford L. Librach
Department of Obstetrics and Gynaecology, Sunnybrook Health Sciences Centre and Women's College Hospital, University of Toronto.

P-E2 ENHANCED FUNCTION OF PREFRONTAL SEROTONIN 5-$HT_{2A/C}$ RECEPTORS IN A RAT MODEL OF PSYCHIATRIC VULNERABILITY
Nathalie M Goodfellow[G](1), Madhurima Benekareddy(2), Farhan Mohammed(2), Vidita A. Vaidya(2), Evelyn K Lambe (1,3)
(1)Department of Physiology, University of Toronto, (2) Department of Biological Sciences, Tata Institute of Fundamental Research, Mumbai, India, (3)Department of Obstetrics and Gynaecology, University of Toronto.

P-E3 THE EFFECT OF CHRONIC MATERNAL ADVERSITY (CMA) ON ACTIVITY AND ATTENTION IN JUVENILE GUINEA PIG OFFSPRING
Jeff Emack[G](1) and Stephen Matthews(1,2,3)
Departments of (1)Physiology, (2)Obstetrics and Gynaecology, and (3)Medicine, Faculty of Medicine, University of Toronto.

P-E4 ALTERED mGLUR1/5 SIGNALING IN MICE LACKING THE GENERAL TRANSCRIPTION FACTOR GTF2IRD1
Eliane Proulx [G](1), Edwin J Young (2), Lucy R Osborne (2), Evelyn K Lambe(1,3); (1)Department of Physiology, (2) Department of Molecular Medical Genetics, (3) Department of Obstetrics &Gynaecology.

P-E5 TRANSGENERATIONAL EFFECTS OF SYNTHETIC GLUCOCORTICOIDS ON THE EXPRESSION OF KEY REGULATORY GENES IN THE HIPPOCAMPUS
Vasilis Moisiadis[O](1), A Kostaki(1), SG Matthews(1, 2, 3).
Departments of (1)Physiology, (2)Obstetrics and Gynaecology and (3)Medicine, Faculty of Medicine, University of Toronto.

P-E6 ACUTE AROMATASE INHIBITION DECREASES BREAST PARENCHYMAL ENHANCEMENT OBSERVED ON MAGNETIC RESONANCE IMAGING IN POSTMENOPAUSAL WOMEN
Noha Mousa [F] (1), R Eiada (2), P Crystal (2), D Nayot (1), Robert Casper (1)
(1) Department of Obstetrics and Gynaecology, Mount Sinai Hospital, University of Toronto, (2) Department of Radiology, Mount Sinai Hospital, University of Toronto.

GROUP F:

Chairs/Judges: Dr. Howard Berger & Harold Drutz
Judge: Dr. Elliott Lyons

P-F1 COST COMPARISON OF THE LAPAROSCOPIC BURCH COLPOSUSPENSION, LAPAROSCOPIC TWO-TEAM SLING AND THE TRANS-OBTURATOR TAPE PROCEDURE FOR THE TREATMENT OF STRESS URINARY INCONTINENCE

Stacey L Grossman [F], Violaine Marcoux, Rose Kung, Patricia E Lee
Division of Urogynaecology, Department of Obstetrics and Gynaecology, Sunnybrook Health Sciences Centre

P-F2 USE OF THE INTRAUTERINE SYSTEM FOR MENSTRUAL SUPPRESSION IN THE DEVELOPMENTALLY DELAYED ADOLESCENT

Erin Barlow [F], Lisa Allen, Nicolette Caccia, Joley Johnstone, Sari Kives, Rachel Spitzer, Melanie Ornstein
The Hospital for Sick Children, Toronto, Ontario

P-F3 A HIERARCHICAL ANALYSIS OF TRIAL OF LABOUR IN ONTARIO: DO WOMEN, DOCTORS, OR HOSPITALS CHOOSE?

Michelle Wise [G](1), Kellie Murphy(2), Mary Hannah(3), Geoff Anderson(1)
(1) Department of Health Policy, Management and Evaluation, (2) Department of Obstetrics and Gynaecology, Mount Sinai Hospital, (3) Department of Obstetrics and Gynaecology, Faculty of Medicine, University of Toronto

P-F4 CHARACTERISTICS OF WOMEN REQUIRING HOSPITAL ADMISSION FOR TREATMENT OF PELVIC INFLAMMATORY DISEASE (PID) AT AN INNER CITY HOSPITAL

Fatuma A Estanbul [R], Mark H Yudin
University of Toronto, St. Michael's Hospital, Department of Obstetrics and Gynaecology

P-F5 CONSISTENCY OF ANTIBIOTIC REGIMENS FOR SUSPECTED CHORIOAMNIONITIS IN A DOWNTOWN TORONTO TEACHING HOSPITAL

Alice Pham[R](1), Kelly Chu(1), Mark Yudin (2)
(1) Department of Obstetrics & Gynaecology, University of Toronto, (2) Department of Obstetrics and Gynaecology, St. Michael's Hospital, University of Toronto.

****POSTER TAKEDOWN IMMEDIATELY AFTER POSTER SESSION****

ORAL SESSION II

11:10 – 12:10 **Oral Session II** (4 presentations @15 minutes: 10 minute presentation + 5 minutes for questions) **(O6-O9)** [NF, ground floor Lecture Hall, Rm. 003]

Chair: Dr. May Alarab
Judges: Drs. S Lee Adamson & Sony Sierra

11:10-11:25 **O6 VALIDATION OF THE MODIFIED SEXUAL ADJUSTMENT AND BODY IMAGE SCALE IN WOMEN WITH A DIAGNOSIS OF GYNAECOLOGIC CANCER (SABIS-G)**

Marie Wegener[M](1)**,** Sara Urowitz (2), Catherine Classen(3), David Wiljer(4), Christine Massey(5), Sarah E. Ferguson(1).
(1)Department of Gynaecologic Oncology, Princess Margaret Hospital, (2)Department of Psychiatry, Princess Margaret Hospital, (3)Women's College Research Institute, Women's College Hospital, (4)Department of Radiation Oncology, Princess Margaret Hospital, (5)Department of Biostatistics, Princess Margaret Hospital.

11:25-11:40 **O7 MCL-1/MTD RHEOSTAT DETERMINES AUTOPHAGY IN PLACENTAL DEVELOPMENT AND DISEASE**

Manpreet Kalkat[G](2,3), Julia Garcia(3), Tulia Todros(4) and Isabella Caniggia(1,2,3)
(1)Department of Obstetrics and Gynaecology, (2)Department of Physiology, University of Toronto, (3)Samuel Lunenfeld Research Institute, Mount Sinai Hospital, (4)Department of Obstetrics/Gynaecology, University of Turin, Italy.

11:40-11:55 O8 DREAM-MEDIATED REGULATION OF GCM1 IN THE HUMAN PLACENTAL TROPHOBLAST

Dora Baczyk [O](1), Khrystyna Levytska (1), Sascha Drewlo (1), Steve Lye (1) and John Kingdom (1,2).
(1) Research Centre of Women's and Infants' Health at the Samuel Lunenfeld Research Institute of Mount Sinai Hospital, University of Toronto, (2) Maternal-Fetal Medicine Division, Department of Obstetrics & Gynaecology, Mount Sinai Hospital

11:55-12:10 **O9 CHARACTERISTICS AND MANAGEMENT OF ADNEXAL MASSES IN A CANADIAN PAEDIATRIC AND ADOLESCENT POPULATION**

Yolanda Kirkham [R](1), Judith A Lacy (2), Sari Kives (3), Lisa Allen (3)
(1) Department of Obstetrics & Gynaecology, University of Toronto, (2) Overlake Hospital Medical Center, Bellevue, Washington, USA, (3) Section of Paediatric Gynecology, Hospital for Sick Children, University of Toronto

12:15 – 1:15 p.m. **LUNCH** [Dining Room, Burwash Hall]

****Poster Takedown for a.m. session and Set-up for p.m. session****
Poster Set-up for p.m. session [NF, ground floor, Rms. 004, 006, 007 & 008]

ORAL SESSION III

1:20 – 2:50 p.m. **Oral Session III (O10-O15)** (6 presentations @15 minutes – 10 minute presentation + 5 minutes for questions)

Chair: Dr. Andrea Jurisicova
Judges: Drs. P Gareth Seaward & Marcus Bernardini

1:20-1:35 **O10 LOW MALIGNANT POTENTIAL TUMOUR REPRODUCTIVE RISK FACTOR ANALYSIS FROM THE FOTS: A MATCHED CASE-CONTROL ANALYSIS**
Jacob McGee[F](1), Ping Sun (2), Isabel Fan(3), Joel Moody(3), John McLaughlin (3), Steven Narod (2)
1)Princess Margaret Hospital, 2)Women's College Research Institute, 3)Samuel Lunenfeld Research Institute

1:35-1:50 **O11 OBSTETRIC MANAGEMENT OF WOMEN WITH HEART DISEASE**
Julie Robertson [F] (1), Candice Silversides (2), May Ling Mah (2),Mathew Sermer (1)
(1)Division of Maternal Fetal Medicine, Mount Sinai Hospital, University of Toronto, (2) Department of Cardiology, Toronto General Hospital, University of Toronto

1:50-2:05 **O12 SIGNIFICANCE OF ABNORMAL SONOGRAPHIC FINDINGS IN POSTMENOPAUSAL WOMEN WITH AND WITHOUT BLEEDING**
Rebecca Menzies [M](1), Sarah Wallace (1), Marguerite Ennis (2), Alison Bennett (3), Michelle Jacobson (4), Gina Yip (3), Wendy Wolfman (1)

2:05-2:20 **O13 ROBOTICALLY-ASSISTED LAPAROSCOPIC MYOMECTOMY: A CANADIAN EXPERIENCE**
Fady W Mansour [F], Guylaine Lefebvre, and Sari Kives
Department of Gynecology, St-Michael's Hospital, University of Toronto

2:20-2:35 **O14 PERIPHERAL LEUKOCYTES AS NOVEL TARGETS FOR THE PREVENTION OF PRETERM LABOUR**
Tamara Nedd-Roderique[G](1,2), Oksana Shynlova(1), Anna Dorogin(1) and Stephen Lye(1,2,3) (1) Samuel Lunenfeld Research Institute, Mount Sinai Hospital; (2) Physiology, University of Toronto and (3) Obstetrics & Gynaecology, University of Toronto.

2:35-2:50 **O15 PARACRINE ACTIONS OF IMMUNE AND TROPHOBLAST CELLS IN CYTOKINE PRODUCTION WITH LPS AND PROBIOTIC LACTOBACILLI IN HUMAN PLACENTA**
Maryam Yeganegi [PD](1,3), Chiashan G Leung(1), Andrew Martins(2), Sung Kim(2), Gregor Reid(2), John RG Challis(1) and Alan D Bocking(1,3).
(1)Department of Physiology & Obstetrics & Gynaecology, University of Toronto, (2)Department of Microbiology & Immunology, University of Western Ontario, (3)Samuel Lunenfeld Research Institute, Mount Sinai Hospital.

2:50– 4:10 p.m. **POSTER SESSION II**

2:50 – 3:10 p.m. **Coffee Break & Poster Session II Walkabout** [NF ground floor lobby, Rms. 004, 006, 007 & 008]

3:10 – 4:10 p.m. **Poster Session II Tour** (3-5 minute presentation + 5 minutes for questions) [NF Rms. 004, 006, 007 & 008]
Groups G-L

GROUP G:

Chairs/Judges: Drs. Isabella Caniggia & Clifford Librach
Judges: Drs. Ellen Greenblatt & Robert Casper

P-G1 THE ROLE OF ANDROGEN SUPPLEMENTATION IN OVULATION INDUCTION IN OLDER WOMEN
Sonia Blanco Mejia [G](1)(2), EA Claessens (1)(3), Manuela Maroleanu (1), Edward A Ryan (1)(3).
(1) Toronto West Fertility Center, Etobicoke, Ontario, (2)Department of Nutritional Sciences, St. Michael's Hospital, University of Toronto, (3)Department of Obstetrics & Gynaecology, University of Toronto.

P-G2 EFFECT OF VERY HIGH SERUM ESTRADIOL LEVELS ON IMPLANTATION IN HIGH-RESPONDERS UNDERGOING IN VITRO FERTILIZATION (IVF)
Farheen Mussani[M](1), Pratibhasri A Vardhana(2), Hanna Balakiier(3), Clifford Librach(4)
(1) University of Toronto, (2),(3),(4)Create Fertility Centre, Division of Reproductive Endocrinology and Infertility, Department of Obstetrics and Gynaecology, Women's College and Sunnybrook Health Sciences Centre.

P-G3 FUNCTIONAL ANALYSIS OF NALP5 AND ITS POSSIBLE INTERPLAY WITH SPINDLE ASSEMBLY CHECKPOINT PROTEINS DURING OOGENESIS

Russanthy Velummailum [G] (1, 2). Zhi-BinTong (3), Lawrence Nelson (3), Andrea Jurisicova (1, 2, 4) (1) Department of Physiology, University of Toronto, (2) Samuel Lunenfeld Research Institute, Mount Sinai Hospital, (3) Developmental Endocrinology Branch, National Institutes of Health, Bethesda, MD (4) Department of Obstetrics & Gynaecology, University of Toronto.

P-G4 EXPRESSION OF SURVIVIN IN HUMAN OOCYTES AND EMBRYOS

Shirin Zaver [G], Rong Xiao, Junhai Zhao, Rodica Mandel, Shang-main Yie, Hanna Balakier, Clifford Librach
CReATe Fertility Centre, Department of Obstetrics and Gynaecology, Sunnybrook Health Sciences Centre and Women's College Hospital, University of Toronto

P-G5 NON-INVASIVE GENOMIC ANALYSIS OF HUMAN ENDOMETRIAL RECEPTIVITY

Crystal Chan[G, R](1,2), Carl Virtanen(3), Neil Winegarden(3), Terence Colgan(4), Theodore Brown(1,2), Ellen Greenblatt(1).
(1)Division of Reproductive Endocrinology and Infertility, Department of Obstetrics & Gynaecology, Mount Sinai Hospital, (2)Samuel Lunenfeld Research Institute, Mount Sinai Hospital, (3)Microarray Centre, University Health Network, (4)Department of Laboratory Medicine and Pathobiology, Mount Sinai Hospital.

P-G6 DIFFERENTIAL EXPRESSION OF MAESTRO (MRO) GENE SPLICE VARIANTS IN CULTURED GRANULOSA AND CUMULUS CELLS IN RESPONSE TO HORMONAL STIMULATION

Shlomit Kenigsberg [O](1), Rana El-rass(1) Chantal Lackan(1), Naazish Alladin(1), Sergey I. Moskovtsev(1), and Clifford L Librach(1, 2).
(1)CReATe Fertility Center, Toronto, Ontario, (2)Department of Obstetrics and Gynaecology, Sunnybrook Health Sciences Centre.

GROUP H:

Chairs/Judges: Drs. Andrea Jurisicova & Kellie Murphy
Judge: Dr. Richard Pittini

P-H1 THE EFFECTS OF SELECTIVE SEROTONIN REUPTAKE INHIBITORS (SSRIs) ON PLACENTAL P-GLYCOPROTEIN

Manzerul Bhuiyan[G](1), Sophie Petropoulos(1), William Gibb(4, 5), Stephen Matthews(1, 2, 3).
(1)Departments of Physiology, (2)Obstetrics & Gynaecology, and (3)Medicine, University of Toronto, (4)Departments of Obstetrics and Gynaecology, and (5) Cellular and Molecular Medicine, University of Ottawa.

P-H2 DIFFERENTIAL PLACENTAL PATTERNING BY FETAL SEX IN HIGH-RISK PREGNANCIES WITH PLACENTAL DYSFUNCTION

Melissa Walker [O](1), Sarah Keating (2), Rory Windrim (1) & John Kingdom (1)
(1) Placenta Clinic, Maternal-Fetal Medicine Division, Department of Obstetrics & Gynaecology, Mount Sinai Hospital, (2) Department of Pathology and Laboratory Medicine, Mount Sinai Hospital, University of Toronto.

P-H3 ANTENATAL DEXAMETHASONE TREATMENT DOWN-REGULATES SYSTEM A TRANSPORT AT TERM IN THE MURINE PLACENTA.

Melanie Audette MC [G] (1), JRG Challis (1-4), SG Matthews (1-3)
Departments of (1)Physiology, (2)Obstetrics & Gynaecology and (3)Medicine, University of Toronto, (4)Michael Smith Foundation for Health Research, Vancouver, BC.

P-H4 DECIDUAL LEUKOCYTE POPULATIONS EXHIBIT DRAMATIC CHANGES ACROSS GESTATION IN HEALTHY HUMAN PREGNANCY.

Aleah Hazan[G](1,2), Caroline Dunk(2), Rebecca L Jones(3), Wendy Whittle(4) and Stephen J Lye(1,2,4). (1)Department of Physiology, University of Toronto, (2)Samuel Lunenfeld Research Institute, Mount Sinai Hospital, (3)Maternal and Fetal Health, St. Mary's Hospital, Manchester, UK, (4)Department of Obstetrics & Gynaecology, Mount Sinai Hospital.

P-H5 EXPANSION OF THE PLACENTAL VASCULATURE IN LATE GESTATION IS STRAIN DEPENDENT IN MICE

Monique Y Rennie [G](1,2), Anum Rahman (1), Kathie J Whiteley (3), S Lee Adamson (3,4), John G Sled (1,2) (1) Mouse Imaging Centre, Hospital for Sick Children, (2) Department of Medical Biophysics, (3) SLRI, Mount Sinai Hospital, (4) Departments of Obstetrics & Gynaecology and Physiology, University of Toronto

P-H6 THE ROLE OF VHL IN REGULATING THE EXPRESSION OF CCND1 IN PHYSIOLOGICAL AND PATHOLOGICAL PLACENTAL CONDITIONS

Livia Deda [G] (1,2,3), Jocelyn Ray (1,2,3), Tullia Todros (4) and Isabella Caniggia (1,2,3)
(1) Mount Sinai Hospital, Samuel Lunenfeld Research Institute, Departments of (2) Physiology and (3) Obstetrics and Gynaecology, Faculty of Medicine, University of Toronto, (4) Department of Obstetrics and Gynecology, University of Turin, Turin, Italy.

GROUP I:

Chairs/Judges: Drs. Theodore J Brown & Allan Covens
Judge: Dr. Jason Dodge

P-I1 THE IMPACT OF ETHNICITY ON AWARENESS, KNOWLEDGE, AND ATTITUDES OF THE HPV VACCINE IN ADULT WOMEN

Sharon Sadry [M](1), Leanne De Souza (2), Mark Yudin, (2). (1) Department of Undergraduate Medicine, Faculty of Medicine, University of Toronto (2) Department of Obstetrics, Gynaecology, & Reproductive Infectious Diseases, St. Michael's Hospital, University of Toronto.

P-I2 MOVING BEYOND PRIMARY SCREENING FOR CERVICAL CANCER IN LOW-RESOURCE SETTINGS

Naana Afua Jumah [R](1), Barry Rosen(2)
(1) Department of Obstetrics and Gynaecology, University of Toronto, (2) Gynaecologic Oncology Division, Princess Margaret Hospital.

P-I3 INTERNATIONAL COOPERATION FOR CANCER CARE: PROTOCOL DEVELOPMENT FOR OVARIAN CANCER TREATMENT IN KENYA

Luc van Lonkhuijzen [F,G](1), Lynn Sterling(2), Job Nyangena (3), Elkanah Orango (3), Barry Rosen (1)
(1) Division of Gynaecologic Oncology, University of Toronto(2) University of Toronto Medical School, (3) Moi Teaching and Referral Hospital, Eldoret, Kenya

P-I4 WITHDRAWN

P-I5 TRAJECTORY OF CARE OF WOMEN REFERRED TO A TERTIARY HEALTH CARE CENTRE WITH ATYPICAL SQUAMOUS CELLS OF UNDETERMINED SIGNIFICANCE (ASCUS) OR LOW-GRADE SQUAMOUS INTRAEPITHELIAL LESIONS (LSIL) ON CERVICAL CYTOLOGY: A CROSS-SECTIONAL ANALYSIS

Andrea N Simpson [M](1), L Le(2), K Joan Murphy(3)
(1) Faculty of Medicine, University of Toronto (2) Biostatistics, Princess Margaret Hospital (3) Department of Gynaecologic Oncology, Princess Margaret Hospital, University of Toronto.

GROUP J:

Chairs/Judges: Drs. John Kingdom & Rose Kung
Judge: Dr. Kimberly Liu

P-J1 SPATIAL AND TEMPORAL EXPRESSION OF ACID CERAMIDASE IN PLACENTAL DEVELOPMENT AND IN PREECLAMPSIA
Reshef Tal [PD], Isabella Caniggia.
Samuel Lunenfeld Research Institute, Department of Obstetrics & Gynaecology, Mount Sinai Hospital, University of Toronto.

P-J2 DUAL SPECIFICITY PHOSPHATASE 9 (DUSP9): A CANDIDATE GENE TO EXPLAIN THE MALE BIAS IN SEVERE PLACENTAL INSUFFICIENCY SYNDROMES
Marie J Czikk [R](1), Sascha Drewlo(2), Dora Baczyk(2), Thomas Kislinger(3,4), S Lee Adamson(1,2), John CP Kingdom(1,2). (1)Obstetrics and Gynaecology, University of Toronto; (2)Samuel Lunenfeld Research Institute, Mount Sinai Hospital; (3)Ontario Cancer Institute, University Health Network; (4)Medical Biophysics, University of Toronto.

P-J3 THE ROLE OF PAR6 IN REGULATING TROPHOBLAST CELL POLARITY
Tharini Sivasubramaniyam [G], Isabella Caniggia.
Departments of Obstetrics & Gynaecology and Physiology, University of Toronto; SLRI, Mount Sinai Hospital.

P-J4 EXPRESSION OF VEGF-A IN THE PLACENTA, MATERNAL ORGANS, AND CIRCULATION DURING PREGNANCY IN MICE
Abhijeet Minhas [G], Shannon Bainbridge, Dawei Qu, Hoon-ki Sung, Andras Nagy, and S Lee Adamson. Samuel Lunenfeld Research Institute, Mount Sinai Hospital; Department of Obstetrics & Gynaecology, Mount Sinai Hospital and Department of Physiology, University of Toronto.

P-J5 HER-1 SIGNALING AND EXTRAVILLOUS TROPHOBLAST MIGRATION
Caroline E Dunk[O](1), JKWright(1,2), H Amsalem(1), C Maxwell(3), S Keating(4) & SJ Lye(1,2,3). (1)Women's and Infant's Health Research Centre, Samuel Lunenfeld Research Institute, Mount Sinai Hospital, (2)Depts of Physiology, (3)Obstetrics and Gynaecology and (4)Pathology, University of Toronto.

GROUP K:

Chairs/Judges: Drs. Wendy Whittle & Mark Yudin
Judge: Dr. Michael Shier

P-K1 HUMAN AMNION TIGHT JUNCTIONS AND INTRAUTERINE INFECTION AND INFLAMMATION

Rebecca Koscik [G](1,2), Wei Li (2), Andrew Martins (3), Sun O Kim (3), Gregor Reid (3), John RG Challis (1), Alan D Bocking (1,2).
(1) Departments of Physiology and Obstetrics and Gynaecology, University of Toronto
(2) Samuel Lunenfeld Research Institute, Mount Sinai Hospital, (3) Department of Microbiology and Immunology, Siebens-Drake Research Center, and the Canadian Research and Development Center for Probiotics, University of Western Ontario

P-K2 PREVALENCE AND CHARACTERISTICS OF GROUP B STREPTOCOCCUS POSITIVE PREGNANT WOMEN IN AN INNER CITY TERTIARY CARE CENTRE

Eliane Shore [R](1), Mark Yudin(2).
(1) Department of Obstetrics & Gynaecology, University of Toronto, (2) Department of Obstetrics, Gynaecology, & Reproductive Infectious Diseases, St. Michael's Hospital, University of Toronto.

P-K3 H1N1 IN PREGNANCY: A TERTIARY CARE CENTRE EXPERIENCE

Ann Malinowski [F](1), Julie Robertson(1), Cynthia Maxwell(1), Allison McGeer(2), Matthew Sermer(1), Dan Farine(1).
(1)Maternal-Fetal Medicine Division, Department of Obstetrics & Gynaecology, Mount Sinai Hospital, (2)Department of Microbiology, Mount Sinai Hospital

P-K4 A NOVEL ROLE OF NUCB2 IN REGULATING THE INITIATION OF LABOUR

Yunqing Li [G](1,2), Oksana Shynlova(1), Xuesen Dong(4) and Stephen J Lye(1,2,3).
(1)Samuel Lunenfeld Research Institute, Mount Sinai Hospital, (2)Department of Physiology, University of Toronto, (3)Department of Obstetrics and Gynaecology, University of Toronto, (4)Prostate Center, Vancouver General Hospital.

P-K5 IMMUNOPHENOTYPING OF PERIPHERAL BLOOD LEUKOCYTES IN PREGNANT AND TERM LABOURING WOMEN

Sally Sabra [G](1,4), Oksana Shynlova (1), Stephen Lye (1,2,3),
(1)Samuel Lunenfeld Research Institute, Mount Sinai Hospital, Department of (2) Ob/Gyn; (3) Physiology and (4) IMS, University of Toronto.

GROUP L:

Chairs/Judges: Drs. Adrian Brown & Prati Sharma
Judge: Dr. Cynthia Maxwell

P-L1 PPROM in Twins: From A to B, In Progress
Susan Pakenham [R](1), Sarah Scattolon(2), Jon Barrett(3) and Ori Nevo(3)**.**
(1)Department of Obstetrics & Gynaecology, University of Toronto (2)Faculty of Undergraduate Medicine, University of Toronto (3)Division of Maternal-Fetal Medicine, Department of Obstetrics & Gynaecology, Sunnybrook Health Sciences Centre.

P-L2 SURVEY OF MENSTRUAL CYCLES: THE PREVALENCE OF MENORRHAGIA AND ITS IMPACT IN THE WORKFORCE
Marilyn Sutandar [R](1), Wusun Paek(1)
(1)Gynaecology Division, Department of Obstetrics & Gynaecology, Mount Sinai Hospital, University of Toronto

P-L3 HOW DOES THE UNIVERSITY OF TORONTO HYSTERECTOMY TRAINING COMPARE TO THE REST OF CANADA?
Jamie Kroft [R], Joel RK Moody, Patricia Lee
Department of Obstetrics and Gynaecology, Sunnybrook Health Sciences Centre, University of Toronto

P-L4 DO PODCASTS MEASURE UP? MEDICAL STUDENTS' PERCEPTIONS OF THE UTILITY OF PODCASTS IN MEDICAL EDUCATION (WORK IN PROGRESS)
Lyana Sisca [R](1), Lynne Zolis(1), Filomena Meffe (2), Michele Farrugia (2)
(1)University of Toronto, (2)Department of Obstetrics and Gynaecology, University of Toronto.

P-L5 THE EFFECT OF REIKI ON PAIN IN WOMEN AFTER ELECTIVE CAESAREAN SECTION – A DOUBLE-BLINDED RANDOMIZED CONTROLLED TRIAL
Sondra vanderVaart [G](1,2), Howard Berger(3), Carolyn Tam(2,4), Y Ingrid Goh(1,2), Violette MGJ Gijsen(2), Saskia N de Wildt(5), Anna Taddio(2), Gideon Koren(1,2,6)
(1)Department of Pharmaceutical Sciences, University of Toronto, (2)Division of Clinical Pharmacology and Toxicology, The Hospital for Sick Children, (3) Department of Obstetrics and Gynaecology, St. Michael's Hospital, (4)Department of Pharmacology and Toxicology, Faculty of Medicine, University of Toronto, (5)Department of Pediatric Surgery, Erasmus MC Sophia Children's Hospital, Rotterdam, The Netherlands, (6)Ivey Chair in Molecular Toxicology, Department of Medicine, University of Western Ontario

4:10 -5:30 p.m. ****Poster Takedown for p.m. session ****

4:15 – 5:15 p.m. **Henderson Lecture** [NF, ground floor Lecture Hall, Rm. 003]
(See biography page 26)

Dr. Jane Norman
Professor of Maternal and Fetal Health
University of Edinburgh, UK
Co-Director, Edinburgh Tommy's Centre for Maternal and Fetal Health Research
Topic: "Being born too soon – do obstetricians have anything to offer?"

5:15 – 5:20 p.m. **Closing Remarks: Dr. Alan Bocking** [NF, ground floor Lecture Hall, Rm. 003]

5:25 – 6:30 p.m. **Wine & Cheese Reception and JW Knox Ritchie Research Awards & Papsin Award Presentations** [Dining Room, Burwash Hall]

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THE HENDERSON LECTURE

The D. Nelson Henderson Lectureship in Obstetrics and Gynaecology was established in 1965, through the generosity of the Henderson family, in honour of Dr. Donald Nelson Henderson, a highly respected clinician-scientist and eminent member of the Department of Obstetrics and Gynaecology at the Toronto General Hospital.

Dr. Jane Norman

We are most pleased to have Dr. Jane Norman present the Henderson Lecture on Research Day, speaking on the topic, "Being Born Too Soon – Do Obstetricians Have Anything to Offer?" Dr. Norman is a graduate of the University of Edinburgh and has held positions at that institution and at the University of Glasgow. She is currently the Director of the Edinburgh Tommy's Centre for Maternal and Fetal Health, and the Research Director for the Jennifer Brown Research Laboratory. Her current research focuses on obesity in pregnancy and preterm birth and she is Chief Investigator of the clinical trial "OPPTIMUM" – a UK multicentre study (over 30 centres), funded by the Medical Research Council (£2.7 million), which will determine if progesterone prevents preterm labour and improves neonatal outcome in high risk singleton pregnancy. She has played both national and international roles in health care and was awarded the 2009 Society for Gynecologic Investigation (SGI) President's Achievement Award, as her "record in scientific investigation is outstanding and assures a continued productive career in research".

Previous Henderson lecturers and topics:

2009 **Dr. David L Keefe,** University of South Florida, Tampa, Florida, USA
Burning the candle at both ends – A telomere theory of reproductive aging

2008 **Dr. Andrew Berchuck**, Duke University Medical Center, Durham, North Carolina, USA
Individualized ovarian cancer treatment and prevention in the genomic era

2007 **Dr. David Phillips,** University of Southampton, UK
Small babies, stress and the metabolic syndrome

2006 **Dr. Robert L Reid,** Queen's University, Kingston, Ontario.
Bringing scientific discovery into the public domain: Rigour and Responsibility

2005 **Dr. Chris Redman,** University of Oxford, UK
A new view of pre-eclampsia

2004 **Dr. JB Trimbos,** Leiden University, The Netherlands
Nerve sparing in radical surgery: Technique and proof of principle

2002 **Dr. David A Grimes,** Family Health International, North Carolina, USA
Potholes on the road to evidence-based practice

2001 **Dr. DT Baird,** University of Edinburgh, UK
Hormonal control of folliculo-genesis: The key to successful reproduction

2000 **Dr. Les Myatt,** University of Cincinnati, USA
Prediction of preeclampsia – Is it possible?

AWARDS

The Papsin Award

The Dr. Frederick R. Papsin Postgraduate Award was inaugurated in 2003 in memory of Dr. Frederick R Papsin, Chief of the Department of Obstetrics and Gynaecology at Mount Sinai Hospital from 1971 to 1988. The award is presented to a postgraduate resident in the final year of training, and is based on teaching ability, mentorship activities and leadership, as chosen by the winner's peers. There have been four recipients, Dr. Andrea Lausman (2005), Dr. Kerry Myckan (2006), Dr. Matthew Morton (2007), Dr. Shereen Chirayilkalam (2008), and Dr. Lynne Zolis (2009).

JW Knox Ritchie Research Awards

Dr. JW Knox Ritchie

The JW Knox Ritchie Research Awards were endowed by a grateful. medical staff at the Department of Obstetrics and Gynaecology, Mount Sinai Hospital and the University of Toronto on the occasion of Dr. Ritchie's retirement from the position of Chief for Mount Sinai and Chair for the University of Toronto Departments of Obstetrics and Gynaecology in 2003.

The JW Knox Ritchie Research Awards are awarded for best abstract/presentation by trainee category (Graduate Student, Resident, Clinical Fellow, Post-Doctoral Fellow, Medical Student).

Previous recipients of the JW Knox Ritchie Research Awards:

2009
Post-Doctoral Fellow: **Sascha Drewlo** (Supervisor: J. Kingdom)
Clinical Fellow: **Clarissa Bambao** (Supervisor: M. Shier)
Resident: **Kelly Chu** (Supervisor: K. Murphy)
Graduate Student: **Shadab Rahman** (Supervisor: R. Casper)
Medical Student: **Erika Frasca** (Supervisor: O. Nevo)

2008
Post-Doctoral Fellow: **Christine Wong** (Supervisor: Robert Casper)
Clinical Fellow: **Marcus Bernardini** (Supervisor: Allan Covens)
Resident: **Taymaa May** (Supervisor: Theodore J Brown)
Graduate Student: **Maryam Yeganegi** (Supervisor: Alan Bocking)
Medical Student: **Sue Jin Kim** (Supervisor: Wendy Whittle)

2007
Post-Doctoral Fellow: **Sascha Drewlo** (Supervisor: J. Kingdom)
Clinical Fellow: **Kimberly Liu** (Supervisor: E. Greenblatt)
Resident: **Taymaa May** (Supervisor: T. Brown)
Graduate Student: **Ingrid Lai** (Supervisor: A. Jurisicova)
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ORAL ABSTRACTS

ABSTRACT #O1

EFFECT OF REDUCED PLACENTAL EXPRESSION OF GLIAL CELL MISSING-1 ON PREGNANCY OUTCOME.

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Preeclampsia (PE) is a disorder of placental origin that affects ~5% of pregnant women. Clinical signs include high blood pressure, proteinuria, and elevated sFLT-1 in placentas and maternal plasma. Placental syncytiotrophoblast is dysfunctional in PE but whether this plays a role in causing PE is unknown. Differentiation into syncytiotrophoblast requires the transcription factor, *Gcm1*. In PE, placental *Gcm1* expression is reduced, and the dysfunctional syncytial layer releases excess sFLT1 into the maternal bloodstream where it is believed to circulate to cause maternal signs of PE. *Gcm1* knockdown in human placental explants also causes syncytial dysfunction and elevates sFLT1 release. We therefore hypothesize that impaired syncytial differentiation caused by hypomorphic *Gcm1* expression is an important underlying cause of PE.

Objective: The aims of the current study were to: A) examine placental villous morphogenesis, angiogenic gene expression, and feto-placental vascularisation and hemodynamic function in heterozygous *Gcm1* knockout mice ($Gcm1^{+/-}$); B) determine whether hypomorphic placental *Gcm1* expression results in clinical signs of PE.

Methods: We crossed heterozygous *Gcm1* males with wild type females to obtain pregnancies with ~50% heterozygous and ~50% wildtype fetuses. At gestational days E13.5 and E17.5 we quantified 1) placental gene expression of *Vegf* and *sFlt1* by qRT-PCR; 2) placental morphology by histology; 3) feto-placental vascularity by vascular corrosion casts and micro-CT imaging; and 4) evaluated maternal hypertension and proteinuria.

Results: The $Gcm1^{+/-}$ labyrinth demonstrated significant retention of proliferating cells thus possibly recapitulating dysregulated syncytiotrophoblast differentiation seen in *Gcm1* silenced human explants. Although placental and fetal weights were unaltered, we observed a hypervascular placental phenotype. This phenotype was quite intriguing in light of paradoxical findings of decreased *Vegf* and increased *sFlt1* expression within the labyrinth of $Gcm1^{+/-}$ placentas. Maternal arterial blood pressure by tail cuff plesthmography was mildly elevated however proteinuria was not observed.

Conclusions: This study provides *in vivo* evidence that *Gcm1* expression affects placental expression of *Vegf* and *sFlt1* and feto-placental vascularisation. The downstream effects of this may result in mild elevations in maternal blood pressure. Whether a more severe maternal phenotype would be apparent without the potential protective effects of wild type litter mate placentas is currently under investigation.

Funded by: Molly Towell Foundation, MSH/UHN Dept of Ob/Gyn, and CIHR

ABSTRACT #O2

CORTICOSTEROIDS INCREASE P-GLYCOPROTEIN FUNCTION IN PRIMARY GUINEA PIG BRAIN ENDOTHELIAL CELLS

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Objective: Multidrug resistance phosphoglycoprotein (P-gp) is a membrane transporter that actively removes a wide range of substrates from cells. Placental P-gp decreases with advancing gestation, and the resultant increase in maternal-fetal transfer leaves the fetus vulnerable to teratogenic factors. However, we have evidence that there is a dramatic up-regulation of P-gp in brain endothelial cells – cells which constitute the primary *bbb*. This leads to decreased penetration of P-gp substrates into the fetal brain with advancing gestation. Therefore, P-gp in the fetal *bbb* compensates for the decreased global protection previously provided by the placenta. Virtually nothing is known about the regulation of P-gp, and the *Mdr1* genes which code for P-gp, in the developing *bbb*. The purpose of these experiments is to establish a novel brain endothelial cell model, and determine the roles corticosteroids play in P-gp regulation.

Methods: Primary brain endothelial cell cultures were established from 2-week old male guinea pigs. Endothelial nature was confirmed by the presence of Von Willebrand factor. Confluent cultures were treated with varying doses (10^{-8} – 10^{-5} M) of Dexamethasone (DEX), Cortisol (CORT), and Aldosterone (ALDO) for 2, 8 or 24 hours. Cells were then incubated for 1 hour with 1 μM Calcein-AM (P-gp substrate) immediately following treatment and accumulation of fluorescent Calcein was measured (Em/Ex = 485/530 nm) to assess changes in P-gp function.

Results: DEX treatment resulted in a significant dose- and time-dependant increase ($p<0.05$) in P-gp activity resulting in increased exclusion of P-gp substrate. There was no significant effect of treatment at 2 hours but significant effects at 8 and 24 hours. CORT doses of 10^{-6} M and higher resulted in a similar increase ($p<0.05$) in P-gp function, but these effects occurred even earlier – at 2 hours. CORT-induced increases in P-gp function were greatest at 8 hours. ALDO exhibited much slower effects on P-gp function;. doses greater then 5×10^{-7} M caused a significant increase ($p<0.05$) in P-gp function, but only after 24 hour of treatment.

Conclusions: Glucocorticoids (DEX and CORT) have strong up-regulatory effects on P-gp function in brain endothelial cells. The late gestational surge in fetal plasma glucocorticoids (GC) parallels the increase in *bbb* P-gp expression, and may therefore contribute to *bbb* P-gp upregulation. Furthermore, 10% of pregnant women are at risk of preterm delivery, and receive high doses of synthetic GCs (such as DEX) to mature the fetal lungs. Nothing is known as to how this common treatment affects P-gp at the fetal *bbb*. *Mdr1* genes are heavily influenced by epigenetic processes and GC-induced epigenetic processes may play a role in *Mdr1* up-regulation. On-going experiments in fetal brain endothelial cell cultures (of varying gestational age), help to determine the mechanisms behind the late-gestational increase in P-gp expression.

Funded by: Canadian Institutes for Health Research

ABSTRACT #O3

THE "DUC" TRIAL: A PILOT RANDOMIZED CONTROLLED TRIAL OF IMMEDIATE VS. DELAYED CORD CLAMPING IN PRETERM INFANTS BORN BETWEEN 24 AND 32 WEEKS GESTATION

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Introduction: The current practice in Canada is to clamp the umbilical cord immediately at time of birth in preterm infants to allow for immediate neonatal resuscitation. There is some evidence that a short delay in umbilical cord clamping can improve neonatal outcomes.

Objective: To determine the feasibility of delayed cord clamping in preterm infants in the author's institution. Furthermore, this study would examine our rates of intraventricular hemorrhage (IVH), sepsis, anemia, and hyperbilirubinemia in these groups in anticipation of pursuing a large randomized controlled trial.

Methods: This trial was conducted over an 18 month period. Women with preterm birth (PTB) anticipated between 24-32 weeks gestation were approached for the study. Women were randomly assigned to the immediate cord clamping (ICC) or delayed cord clamping (DCC) group when delivery was anticipated. The cord was cut at 0-15s for ICC and 30-45s for DCC.

Results: The recruitment rate was 33%. The compliance rate was 92% with 3 protocol violations. The average time of cord clamping in the ICC (n=19) and DCC (n=19) groups were 5.4s and 39.7s, respectively ($p<0.05$). The incidence of IVH and sepsis were the same in both groups (15% and 10%, respectively). Thirty-five percent in ICC and 21% in DCC required blood transfusion. The incidence of hyperbilirubinemia requiring phototherapy was 66.7% and 74% in the ICC and DCC groups, respectively. No infant in either group required exchange transfusion. There was 1 neonatal death in the ICC group.

Conclusion: A short delay in umbilical cord clamp (30-45s) is feasible and safe in preterm infants (24-32 weeks) in our institution.

ABSTRACT #O4

EXPRESSION OF ENZYMES REGULATING EXTRACELLULAR MATRIX BIOGENESIS IN VAGINAL TISSUE OF WOMEN WITH AND WITHOUT PELVIC ORGAN PROLAPSE

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Hypothesis and Objectives: We have reported earlier that the expression of some of the Lysyl Oxidase (LOX) family enzymes was significantly reduced in premenopausal women with pelvic organ prolapse (POP). LOX proteins regulate the biogenesis of extracellular matrix (ECM) proteins by cross-linking collagen and elastin polymers, whereas Procollagen C Proteinase (PCP) participates in the maturation of collagen molecules by cleaving the procollagen precursor. Ovarian hormones can influence the LOX and PCP proteins expression in the pelvic floor tissues of young women. This study examines in vaginal tissues the expression of (1) LOX family enzymes in postmenopausal women and (2) PCP in pre and postmenopausal patients with severe POP and healthy women. To account for the age-related changes (3) we compared the LOX family and PCP gene expression between pre and postmenopausal controls.

Methods: Patients with severe POP (≥grade 3) and control patients (no POP) undergoing total hysterectomy for benign conditions were recruited. Hospital Ethics Board Approval was obtained. During the surgical procedure, 0.5cm^2 of full thickness vaginal tissue was obtained from the surgical cuff. Total RNA was extracted using TRIZOL. Real time PCR was performed to quantify mRNA levels of LOX, LOXL1-4 and PCP. Total protein was extracted with RIPA and analyzed by Western blot to quantify protein levels of LOXL1-2 and PCP. Mann-Whitney test ($p<0.05$) was used for statistical analysis.

Results: 39 pre-menopausal (26 patients and 13 controls), and 18 post-menopausal women (13 patients and 5 controls) were enrolled. (1) LOX and LOXL2 ($p<0.05$) genes were down-regulated in postmenopausal POP patients compared to controls, whereas LOXL1 ($p<0.05$) and LOXL3 were increased. In contrast to the gene expression, LOXL2 protein expression was significantly higher in postmenopausal patients compared to controls ($p<0.05$). (2) PCP gene expression was significantly down-regulated in pre and postmenopausal POP patients compared to controls ($p<0.001$). (3) The postmenopausal control group showed lower gene expression of the majority of ECM-modifying enzymes such as PCP, LOX, LOXL1 and LOXL4. However, LOXL2 ($p<0.05$) and LOXL3 gene expression was higher in vaginal tissue after menopause.

Conclusions: Patients with severe POP showed altered expression of genes and proteins regulating collagen and elastin biogenesis, which may result in defective assembly of pelvic tissues and development of POP. Age-related hormonal changes influence the expression of genes related to the ECM biogenesis in vaginal tissue, which may explain the physiopathology of the genitourinary problems affecting women after the menopause.

Funded by: Dean's Fund New Staff Grant Competition Fall 2008 – U of T

ABSTRACT #O5

DAB2 ENHANCES GLUCOCORTICOID RECEPTOR-MEDIATED ANTI-INFLAMMATORY SIGNALLING IN ES2 OVARIAN CANCER CELLS

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Objective: We previously obtained and compared gene expression profiles from laser-capture microdissected non-malignant fallopian tube epithelium from *BRCA1/2*-mutation carriers (FTEb) and control patients, and adnexal high-grade serous carcinoma (SerCa) to identify alterations predisposing to malignant transformation. Notably, FTEb samples obtained during the luteal phase showed global gene expression profiles closely resembling SerCa specimens, and exhibited decreased expression of the adaptor molecule disabled homolog 2 (DAB2) (Clinical Cancer Research, 2008). Further data analysis indicated elevated expression of pro-inflammatory and tumour-promoting nuclear factor-κB (NFκB)-dependent target genes consistent with diminished glucocorticoid receptor (GR) signalling in DAB2-deficient FTEb luteal samples. The objective of this study was to determine whether DAB2 is involved in anti-inflammatory signalling by GR.

Methods: To determine the effect of DAB2 on GR transactivation activity, ES2 ovarian cancer cells were co-transfected with DAB2 cDNA and a GRE-responsive luciferase reporter gene. Cells were treated with 10nM dexamethasone (dex) or vehicle and harvested 24h later for luciferase activity determination. To determine the impact of DAB2 on GR transrepression of NFκB, cells were co-transfected with DAB2-specific siRNA and an NFκB-responsive luciferase reporter, treated with 10ng/mL of tumour necrosis factor (TNF)-α and/or 100nM dex, and harvested 8h later for luciferase activity determination. To determine if DAB2 alters mRNA expression of NFκB target genes, cells were transfected with DAB2 siRNA, treated with dex and/or TNFα and harvested for total RNA extraction and RT-qPCR. Co-immunoprecipitation studies were performed to determine if DAB2 interacts with GR or RelA.

Results: DAB2 overexpression enhanced dex-induced GR transactivation activity relative to empty vector. Conversely, DAB2-specific siRNA increased TNFα-induced NFκB activity relative to non-targeting siRNA, suggesting that DAB2 may also enhance GR-mediated transrepression of NFκB. Consistently, DAB2 siRNA combined with TNFα treatment greatly increased mRNA expression of the NFκB-dependent target gene superoxide dismutase 2 (SOD2). Endogenous DAB2 protein was found to interact with both GR and NFκB, suggesting that DAB2 may impact GR-mediated suppression of NFκB signalling in part via protein-protein interactions.

Conclusions: Altogether, these data support a novel role for DAB2 in activation of GR-mediated transactivation and suppression of NFκB signalling. We therefore postulate that decreased DAB2 expression and GR signalling observed in select luteal phase FTE may result in increased pro-inflammatory signalling thereby promoting serous carcinogenesis.

Funded by: This work was supported by the TOCRN with funds donated by the Toronto Fashion Show. AT was a recipient of an Ontario Women's Health Council/CIHR Institute of Gender and Health Doctoral Research Award.

ABSTRACT #O6

VALIDATION OF THE MODIFIED SEXUAL ADJUSTMENT AND BODY IMAGE SCALE IN WOMEN WITH A DIAGNOSIS OF GYNAECOLOGIC CANCER (SABIS-G)

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Objective: There is evidence that treatment of gynecologic cancer negatively effects sexuality however there is no measure validated in this population to identify these women. Our research objective is to validate the Sexual Adjustment and Body Image Scale-Gynecologic Cancer (SABIS-G) a newly developed measure to assess disturbances in sexuality and body image after gynecologic cancer.

Methods: Women with a history of gynecologic cancer without evidence of disease completed self-report measures on sexuality and psychosocial functioning including the SABIS-G. The SABIS-G, a 9-item measure, was modified from a previously validated measure assessing changes in sexuality and body image in women with breast cancer. Maximum likelihood factor analysis with Promax rotation was performed. Factors would be retained if their eigenvalues were greater than the average eigenvalue. Spearman correlation coefficients between SABIS-G and other instruments were calculated as measures of validity.

Results: 232 patients (pts) were approached to participate, 166 (72%) pts consented to the study and 132 (80%) pts completed the SABIS-G. The median age was 53 (range 26-79) and the primary site of disease was: 53 (40%) endometrium, 45 (34%) cervix and 29 (22%) ovary. Two variables were dropped based of high correlations with other variables (> 0.7). The median SABIS-G score on the final 7-item measure was 40.6 (range 0-75) with lower score indicating greater disturbance. Further analysis was performed on the final 7 variables.
A two factor solution was favored and suggested two underlying constructs: disturbance in body image and sexual adjustment. Both subscales demonstrated good internal consistency (Cronbach's α 0.76 and 0.87). Good concurrent validity was demonstrated between SABIS-G and the revised Female Sexual Distress Scale (-0.72). Good convergent validity was demonstrated with the depression and anxiety subscales of the Hospital Depression and Anxiety Scale (-0.52 and -0.44). Good discriminant validity was demonstrated with the theoretically unrelated diet and religious expression questions from the Illness Intrusiveness Ratings Scale, (-0.25 and -0.18). The estimated intra-class correlation coefficient was 0.92, indicating high test-retest reliability.

Conclusions: This analysis of the SABIS-G reports a two factor structure with excellent psychometric properties. These results need to be confirmed in an independent cohort of women with gynecologic cancer.

Funded by: Princess Margaret Hospital

ABSTRACT #O7

MCL-1/MTD RHEOSTAT DETERMINES AUTOPHAGY IN PLACENTAL DEVELOPMENT AND DISEASE

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Objective: Macroautophagy is a catabolic cellular pathway for the degradation of damaged cytoplasmic constituents and has been proposed as an important mediator of cell fate in response to a variety of stimuli including stress. Excessive autophagy has been implicated in cell death and conversely, as a cytoprotective mechanism. Preeclampsia (PE) and Intra-Uterine Growth Restriction (IUGR) are placental pathologies characterized by oxidative stress and increased trophoblast cell death. Biochemical analysis has implicated an essential autophagy inducer, Beclin, to interact with several Bcl-2 family proteins. We have previously reported that the balance between pro-survival Myeloid Cell Leukemia Factor-1 (Mcl-1) and its pro-apoptotic binding partner Matador (Mtd) guides trophoblast cell fate and changes in this rheostat characterize preeclamptic placentae. As Mcl-1 and Mtd are Bcl-2 family members, herein we sought to examine Mcl/Mtd regulation on autophagy in normal and pathological placentation.

Methods: Placentae from early gestation (4-15 weeks; n=36), severe preeclampsia, IUGR (PE n=16; IUGR n=8) preterm (n=8) and term controls (n=9) were used. Immunoblotting was performed to determine the expression of Mcl-1, Beclin and the autophagosome marker LC3-II. Mcl-1 was overexpressed in choriocarcinoma JEG3 cells and a human embryonic kidney (HEK) cell line stably expressing GFP-hMtdL was generated. siRNA technology was used to silence Mcl-1 expression. JEG3 cells were treated with the nitric oxide donor sodium nitroprusside (SNP; 2.5 mM). Lysotracker red staining was performed to image lysosomal activity. Immunofluorescence staining was used to establish the subcellular localization of GFP-hMtdL upon doxycycline treatment to induce MtdL expression.

Results: Increased LC3-II expression was found at 6-8 weeks of gestation, correlating to increased MtdL expression. Exposure of JEG3 cells to SNP resulted in both increased expression of LC3-II and lysosomal activity and by reduced Mcl-1 expression. Overexpression of Mcl-1 decreased LC3-II expression; conversely Mcl-1 silencing resulted in increased lysosomal activity and LC3-II levels. Notably, GFP-hMtdL induction increased LC3-II expression and lysosomal activity. Increased expression of LC3-II was observed in PE placentae while, in contrast, LC3-II levels were decreased in IUGR.

Discussion: Our data demonstrate that Mcl-1 and Mtd directly exert opposing effects on autophagic induction, thereby maintaining trophoblast cell homeostasis. Increased autophagy in PE, but not in IUGR, may reflect changes in the Mcl-1/Mtd system and thus contribute to the pathogenesis of this disease.

Funded by: CIHR, OWH/IGH and Genesis Research Foundation.

ABSTRACT #O8

DREAM-MEDIATED REGULATION OF GCM1 IN THE HUMAN PLACENTAL TROPHOBLAST

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Objective: The trophoblast specific transcription factor GCM1 regulates the balance of proliferation and differentiation in both villous and extravillous human trophoblast. Reduced GCM1 expression is a key feature of severe pre-eclampsia (sPE). Our i*n silico* analysis of GCM1 gene identified a novel calcium-dependent transcriptional repressor – DREAM as a regulatory candidate for GCM1. Thus our objective was to determine the role of DREAM inGCM1 expression and villous trophoblast turnover in human placenta.
Methods: Loss of function and gain off function studies were performed in human choriocarcinoma cell line, BeWo, and floating placental villous explant model. Calcium-dependency was interrogated in both models by contrasting the effects of ionomycin and nimodipine. A direct interaction between DREAM and GCM1 promoter was explored using EMSA and ChIP assays. Furthermore, DREAM expression levels in pre-eclampsia and age-matched controls were determined using qRT-PCR.
Results: DREAM is a calcium dependent negative regulator of GCM1. siRNA-mediated DREAM silencing in both BeWo and floating villous explants significantly upregulated GCM1 causing reduced cytotrophoblast proliferation but increased level of syncytiotrophoblast apoptosis. Calcium dependency was demonstrated in both BeWo cells and floating villous explant models. Elevation in intracellular calcium levels results in DREAM's conformational change, dissociation from the promoter region resulting in de-repression of its target. A direct interaction of DREAM with DRE sites found with in a 43 bp region of the 5' GCM1 promoter was demonstrated using EMSA and ChIP assay. As assessed by qRT-PCR, DREAM mRNA levels are 4-fold upregulated in pre-eclampsia vs age-matched controls.
Conclusion: DREAM exerts a physiologic negative upstream regulatory function on GCM1 expression in human placenta via direct binding to its promoter. DREAM may participate in calcium-dependent trophoblast turnover and be relevant to sPE pathogenesis.
Funding: CIHR to JK.

ABSTRACT #O9

CHARACTERISTICS AND MANAGEMENT OF ADNEXAL MASSES IN A CANADIAN PAEDIATRIC AND ADOLESCENT POPULATION

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Objective: To determine if presentation, imaging, tumour markers differed between adnexal masses managed expectantly versus surgically in pediatric/adolescent gynecology.

Study Methods: Retrospective review of patients with adnexal masses between 2003 and 2006 at Toronto's Hospital for Sick Children. T-tests, Chi-Square, and Pearson correlation tests were used.

Results: 114 patients were identified. Fifty-nine percent had surgery (laparotomy 41.8%, laparoscopy 58.2%) while 41% were managed conservatively. Mean age was 12.7 years (range 7 days to 18 years) with no difference between management groups ($p = 0.59$). The most common symptom was abdominal pain (72.8%). Increased abdominal girth was the only presenting complaint that differed, found only in the surgical group ($p<0.001$).

Size was the only feature on imaging that differed between groups (11.1cm surgical vs. 5.3cm observed, $p<0.001$). CT scans were ordered in 35 patients; 94.3% of whom had surgery ($p<0.001$). Tumour markers were drawn in 41.2% of patients, more often in surgical patients ($p<0.001$). Twenty-seven percent were abnormal, all in the surgical group. Twelve percent of the surgeries were for malignancies. Mean diameter of malignancies was larger than benign masses (16.1cm vs 10.5cm, $p<0.05$). Mean time to resolution of simple or complex masses in the expectant group was 89.6 days ($p=0.41$). There is a linear relationship between mass size and time to resolution ($p<0.05$).

Conclusions: Presentation with increased abdominal girth, larger masses, and abnormal tumour markers were associated with surgical intervention. More investigations were performed in surgically managed patients. Forty-one percent of masses consulted to PAG specialists resolved with expectant management.

ABSTRACT #O10

LOW MALIGNANT POTENTIAL TUMOUR REPRODUCTIVE RISK FACTOR ANALYSIS FROM THE FOTS: A MATCHED CASE-CONTROL ANALYSIS

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Objectives: We undertook a matched case-control study designed to identify risk factors associated with low malignant potential (LMP) ovarian tumours.

Methods: The Ontario Cancer Registry was used to identify all cases of LMP/borderline ovarian neoplasms diagnosed within the Canadian province of Ontario between January 1, 1995 and December 31, 1997, with diagnosis confirmed through pathology review. Of 302 patients, 229 agreed to participate and completed risk factor questionnaires. Controls were obtained from a database comprised of BRCA 1 and 2 mutation negative patients, living within Canada, originally referred for BRCA testing because of a high risk family history for breast and/or ovarian cancer. We matched on age, ethnicity, and date of control interview (later than date of surgery of matched cases). Conditional logistic regression analysis was performed estimating associated OR for LMP tumours for reproductive risk factors – parity, OCP use, history of tubal ligation, smoking status, history of infertility, and clomid use. Univariate and multivariate analysis was performed.

Results: Questionnaires were completed by 229 women with a history of LMP ovarian tumour, with 200 pairs matched. All factors significant on multivariable analysis were significant on univariate analysis. Parity was associated with a decreased risk of LMP tumour, with an OR per birth of 0.78 (0.63-0.94) p=0.008. OCP usage as a categorical variable was not statistically significant OR=0.74 (0.34-1.61) p=0.45, but when trended per year of use, it was found to be protective OR=0.92 (0.86-0.99), p=0.02. Clomid use was associated with an increased risk of LMP tumour, OR=5.07 (1.10-23.3) p=0.04.

Conclusions: The risk of LMP tumours is increased in women who use clomid as an ovulation induction agent for treatment of infertility.

ABSTRACT #O11

OBSTETRIC MANAGEMENT OF WOMEN WITH HEART DISEASE

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Objectives: The hemodynamic changes that occur in pregnancy put women with heart disease at risk of complications. Current guidelines for obstetric management of women with heart disease recommend early epidural, a shortened second stage of labour, and assistance at vaginal delivery with avoidance of Valsalva; invasive monitoring, prolonged postpartum stay, and elective cesarean section are sometimes recommended for high-risk individuals. These recommendations have not been scientifically validated. Our aim was to demonstrate that obstetric management of women with cardiac disease is safe, does not result in high rates of peripartum cardiac events, and results in rates of adverse obstetric and neonatal outcomes comparable to those in women without cardiac disease.

Methods: 563 singleton pregnancies not ending in miscarriage and followed at Mount Sinai Hospital between January 2000 and April 2009 were identified. A control group of 1132 women without heart disease and with singleton pregnancies receiving care at Mount Sinai Hospital during the same time period was randomly selected. Data recorded on all patients included age, gravidity, parity, presence of comorbid conditions, whether labour was spontaneous or induced, mode of delivery, type of anesthesia, maternal monitoring during labour and delivery, duration of the second stage, and gestational age at delivery. Peripartum outcomes were divided into cardiac, obstetric, and neonatal outcomes and were classified according to previously proposed definitions described by Siu and colleagues.

Results: Peripartum cardiac adverse events occurred in 1.9% of women with heart disease. More women with heart disease underwent induction of labour compared with controls (50.5% vs 28.0%, $p < 0.0001$). The rate of cesarean section was not significantly different (32.3% in women with heart disease vs 31.4% in controls, $p = 0.717$). Spontaneous vaginal delivery was significantly less frequent in those with heart disease (38.2% vs 58.0%, $p < 0.0001$) ; assisted vaginal delivery was more frequent, with forceps-assisted delivery performed in 12.0% of women with heart disease vs 2.4% without ($p < 0.0001$) and vacuum-assisted delivery in 17.2% of women with vs 8.2% without heart disease ($P<0.0001$). Lacerations were not increased (32.2% no laceration in women with heart disease vs 38.2% in women without heart disease, Chi Square $p=0.5857$). The second stage of labour was prolonged by 38 minutes in women with heart disease who underwent assisted vaginal delivery when compared to women without heart disease with normal vaginal delivery ($p < 0.0001$). The rate of postpartum hemorrhage was slightly higher in women with heart disease but was within normal limits. There were no differences between the groups in gestational age at delivery, rates of small-for-gestational age babies, rates of admission to NICU, stillbirth, or fetal or neonatal death. The rate of recurrence of congenital cardiac disease among babies was 7.8%.
Conclusions: This study provides evidence that current guidelines for obstetric management of patients with cardiac disease are safe. When these management strategies are implemented, rates of cesarean section, maternal obstetric complications, and fetal and neonatal adverse events are comparable between patients with cardiac disease and controls, and cardiac adverse events are rare.

ABSTRACT #O12

SIGNIFICANCE OF ABNORMAL SONOGRAPHIC FINDINGS IN POSTMENOPAUSAL WOMEN WITH AND WITHOUT BLEEDING

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Objective: To determine the incidence of endometrial cancer in hysteroscopy patients who were bleeding or asymptomatic with sonographic evidence of endometrial thickening. To compare pathologic outcomes in bleeding postmenopausal patients with asymptomatic non-bleeding postmenopausal patients who underwent operative hysteroscopy.

Method: A retrospective chart review was performed comparing 294 postmenopausal women with abnormal uterine bleeding and 142 postmenopausal women without abnormal uterine bleeding who underwent hysteroscopy between 2004 - 2009 at Mount Sinai Hospital, Toronto. Primary endpoints were evidence of cancer and surgical complications of hysteroscopy**.** The 11mm cut-off developed by Smith-Bindman for asymptomatic women was applied to determine whether this endometrial thickness threshold would differentiate the asymptomatic group by risk for endometrial cancer.

Results: BMI was significantly higher in bleeding women. Polyps were significantly more common in asymptomatic women. Fourteen endometrial cancers and 10 cases of endometrial hyperplasia were found in symptomatic patients. Only two cases of cancer with average thickness 17.5mm (1.4%) and one case of hyperplasia (0.71%) was found in the asymptomatic group. Characteristics of non-bleeding patients with endometrial thickness >11mm resembled those of bleeding patients >4mm. Logistic regression models showed risk of a bleeding patient to develop endometrial cancer at 4mm thickness was the same risk as a non-bleeding patient at 15mm. There were 19 complications in the asymptomatic group (13.8%) and 25 complications in the symptomatic group (8.6%).

Conclusion*:* In asymptomatic postmenopausal patients, operative hysteroscopy for TVUS findings of thickened endometrium or endometrial polyps did not diagnose endometrial hyperplasia or cancer. Asymptomatic postmenopausal women have a low risk of significant endometrial pathology. Cancer was approximately four times more prevalent in bleeding compared to non-bleeding women. Asymptomatic women with endometrial thickness greater than 11mm have similar risk for cancer as bleeding women with thickness greater than 4mm.Thus, asymptomatic women with endometrial thickness greater than 11mm should undergo further investigation.

ABSTRACT #O13

ROBOTICALLY-ASSISTED LAPAROSCOPIC MYOMECTOMY: A CANADIAN EXPERIENCE

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Objectives: Evaluate the first series of robotically-assisted laparoscopic myomectomy performed in Canada. Compare the findings to myomectomy performed with laparotomy.

Methods: A retrospective chart review of all robotic myomectomies performed at our tertiary centre was conducted and compared to open myomectomies performed by the same surgeon prior to August 2008. Data was collected with *Microsoft Excel*™ and analyzed using unpaired *t*-test.

Results: Fourteen of 16 cases booked for possible robotic myomectomy were completed successfully from August 2008 to January 2010: one patient was reconsidered for laparotomy prior to robot setup and one case was converted to laparotomy due to intra-operative suspicion of malignancy. One to six fibroids were resected with their size ranging from 6 to 16 centimeters; the total weight ranging from 273 to 870 grams. When compared to open myomectomy, the robotic approach was associated with reduced blood loss (hemoglobin decrease of 26.6 vs. 37.7 g/l, $p = 0.036$), shorter hospitalization (1.3 vs. 2.5 days, $p < 0.01$), and longer operating times (238 vs. 93 minutes, $p < 0.01$). However, there was a trend towards decreased operative times with numbers of robotic cases performed (from 400 to 135 minutes). Three patients required transfusion with open myomectomy, none in the robotic group. There were no major complications in either group.

Conclusions: When compared to laparotomy, robotically-assisted myomectomy is associated with decreased blood loss and shorter hospital stay. Accumulated evidence on risks and benefits will hopefully contribute to enhancing access of this technology for Canadian women and their surgeons.

ABSTRACT #O14

PERIPHERAL LEUKOCYTES AS NOVEL TARGETS FOR THE PREVENTION OF PRETERM LABOUR

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Objective: Previous studies in rat have shown increased expression of uterine cytokines prior to the onset of term labor in association with myometrial/decidual infiltration of peripheral leukocytes (PLs). We hypothesize that PLs are recruited to uterine tissues by locally produced cytokines where they contribute to the initiation of term and preterm labour (PTL). In this study we characterize leukocyte infiltration in the mouse uterus, (1) throughout normal gestation, during term labour and post-partum (PP), as well as, (2) lipopolysaccharide (LPS)-induced and (3) during RU486-induced PTL. We also investigate the mechanism by which PLs are recruited to uterine tissues during labour by analyzing the cytokine profile.
Methods: (1) Non-pregnant (NP) and pregnant CD-1 mice were euthanized on gestational day (d) 15, 17, 18.5, 20 (during labour) and 1d PP. On d15 of gestation pregnant CD-1 mice (n=14) (2) were injected with RU486 (0.15mg sc) or (3) underwent a laparotomy in the lower abdomen. LPS (250μg or 125μg) or sterile saline (sham, n=4) was infused into the uterus between the two gestational sacs most proximal to the cervix. Animals were euthanized during PTL or 24 hours post injection/surgery. One whole uterine horn (myometrium and decidua) was used for immunohistochemical analysis. Immunolocalization of macrophages and neutrophils was defined using newCast stereology software with systematic randomized sampling of 1-2% of the total myometrial area. The second uterine horn was used for protein analysis of cytokine profile in the myometrium. The decidua basalis and parietalis were carefully removed from the myometrial tissue by cutting and mechanical scraping. Protein was extracted from the myometrium using RIPA lysis buffer and analyzed by Bio-Plex Pro Mouse cytokine 23-plex assay (BioRad).
Results: LPS and RU486 injected mice delivered within 24 hours post surgery/injection. Specific antibodies were used to compare the level of macrophage (F4/80) and neutrophil (7/4) infiltration into the myometrium. Preliminary quantitative stereologic analysis suggests that there is a significant increase in both leukocyte sub-groups (macrophage and neutrophil) numbers during term labour. However mainly neutrophils, infiltrate the myometrium during LPS-induced PTL. These changes in leukocyte number were associated with a significant increase in multiple cytokines during normal term labour and LPS-induced PTL: (1) Protein levels of IL-9, IL-12(p40), KC and MCP-1 were increased in labouring myometrial samples compared to NP ($p<0.05$); (2) Protein levels of IL-1β, IL-6, IL-17, G-CSF, KC, MIP-1α, RANTES, IL-12(p40), and MIP-1β were increased and IL-13 was decreased in LPS-induced labouring myometrial samples compared to sham ($p<0.05$). These cytokines are known to mediate both monocyte and neutrophil recruitment. In contrast to the infection-induced PTL, non-infection induced PTL using RU486 was not accompanied by an increase in neutrophil or macrophage infiltration into the myometrium or a change in cytokine expression compared to vehicle controls.
Conclusions: Our results have identified neutrophils and macrophages as two major leukocyte sub-groups that may play a role in the activation of the myometrium leading to delivery. Targeting these cells by blocking the cytokine recruitment might represent a novel approach for the delay or blockage of PTL. **Funded by:** March of Dimes 21-FY10-204

ABSTRACT #O15

PARACRINE ACTIONS OF IMMUNE AND TROPHOBLAST CELLS IN CYTOKINE PRODUCTION WITH LPS AND PROBIOTIC LACTOBACILLI IN HUMAN PLACENTA

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Objective: We have previously shown that *Lactobacillus rhamnosus* GR-1 supernatant up-regulates anti-inflammatory and down-regulates pro-inflammatory cytokine outputs in LPS-treated human primary placenta cultures. However, the cell types responsible for regulating cytokine output are not known. We hypothesize that both immune and placental trophoblast cells could contribute to the modulation of cytokine expression.

Methods: Placental trophoblast cells were isolated from term elective Caesarean section placentae using a percoll gradient and separated from CD45+ cells using magnetic purification, confirmed with immunocytochemistry. Both CD45+ and trophoblast cell types were grown individually and combined at a density of 10^6 cells/well. After 72h incubation period, cells were serum starved for 12h and then divided to four groups: 1) No treatment, 2) Treatment with LPS alone (100 ng/ml) after a further 12h, 3) Treatment with the supernatant from lactobacilli cultures alone (1:20 dilution) for 12h. 4) Pretreatment with lactobacilli supernatant for 12h and subsequent treatment with 100 ng/ml of LPS. Culture media was collected 8h after LPS addition. TNF-α, IL-10 and G-CSF concentrations were measured by ELISA.

Results: LPS increased TNF-α output in mixed, pure trophoblast and CD45+ culture sets by 25.0-, 9.6- and 10.5-fold respectively (N=7). GR-1 supernatant completely abolished this increase in all the cell preparations. The TNF-α output detected for the mixed cultures is a combination of those in pure trophoblast and CD45+ cells. Unlike pure trophoblast and CD45+ cell preparations, in the mixed culture set, LPS, *L. rhamnosus* GR-1 supernatant and the combination of both treatments increased IL-10 (9.7-, 5.6- and 7.1- fold respectively; N=7) and G-CSF (7.3-, 20.9- and 27.4- fold respectively; N=7).

Conclusion: The observed effect of LPS and *L. rhamnosus* GR-1 is an additive effect requiring both pure trophoblast as well as immune cells. Therefore, in an *in vivo* setting, both cell types are necessary components of the maternal-fetal interface for the initiation of an inflammatory cascade and modulation of cytokine output in response to bacterial endotoxins or probiotics.

Funded by: CIHR (#MOP-82799) and the Margaret J. Santalo Fellowship, University of Toronto.

POSTER ABSTRACTS

ABSTRACT #P-A1

INVESTIGATING BIOMARKERS FOR THE EARLY DETECTION OF OVARIAN CANCER

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Objective: To identify proteins differentially expressed between *BRCA1* mutation carriers (OSEb1) and control ovarian surface epithelium (OSE) samples that may serve as biomarkers for the early detection of ovarian cancer (OC).

Methods: To identify proteins differentially expressed between *BRCA1* mutation carriers and control samples, we conducted Multidimensional Protein Identification Technology (MudPIT) on immortalized OSE cell lines from normal individuals and those harboring a *BRCA1* mutation. We compared data to urine and plasma proteome databases to narrow our search. The ideal biomarker for early disease detection would be a secreted protein present at elevated levels in the urine/plasma of OC patients. We validated any proteomics hits by Western Blot analysis on immortalized cultures of OSEb1 and OSE to validate antibody efficacy, followed by Western Blot analysis on protein lysates derived from cultures of primary patient samples (3 OSEb1, 6 OSE).

Results: Comparison of the protein expression profiles revealed 122 proteins up-regulated in OSEb1 samples. Of the 122 up-regulated proteins, 46 were observed in human plasma and urine proteome datasets. Antibodies were available for 27 of these proteins, and eight were followed up by Western Blot analyses. Of those eight selected for follow up, the most promising candidates were Annexin A1, Peroxiredoxin 2 (PRDX2), STAT1, and Annexin A3 (ANXA3). There were no differences in Annexin A1 levels, while ANXA3 was reduced by 2.5 fold in OSEb1 samples. STAT1 was increased (1.5 fold) in OSEb1 samples. PRDX2 dimer was increased in OSEb1, while the monomer was decreased; total levels were slightly increased.

Conclusions: Of the validated bio-marker candidates identified in the initial proteomics screen, Annexin A3, STAT1 and PRDX2 demonstrated significant differences in protein expression in primary ovarian surface epithelium cell lysates from both control and *BRCA1* mutation carrier ovarian tissues. Validation of any of these potential biomarker candidates will have be done using a tissue micro-array that contains samples from OC patients, OSEb1 and OSE. This will assess their staining pattern, subcellular localization and expression level to determine if any of these antigens can eventually serve as early markers for the detection of high risk serous ovarian cancer, with high sensitivity and specificity. Any potential secreted protein that shows differential expression in the tissue micro-arrays, should then be tested in patient plasma and/or urine by ELISA. Functional studies should also be conducted on the effects of over-expression or knockdown of ANXA3, PRDX2, and STAT1 in ovarian cancer cells.

Funded by: Ovarian Cancer Canada

ABSTRACT #P-A2

THE OVARIAN MICROENVIRONMENT OVERCOMES THE ANTI-TUMOUR EFFECT OF SPARC ON OVARIAN CANCER PROGRESSION

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Objective: Several recent studies indicate that SPARC, a multifunctional matricellular glycoprotein, inhibits progression of ovarian cancer. Decreased survival has been reported in SPARC null ($SPARC^{-/-}$) mice injected intraperitoneally (IP) with ID8 murine ovarian cancer cells as compared to wild-type (WT) controls. Women diagnosed with high-grade serous cancer, the most prevalent and lethal form of ovarian cancer, typically present with a prominent ovarian mass that is not reproduced with an IP injection approach. We have previously shown that injection of ID8 cells into the ovarian bursa of mice recapitulates high-grade serous cancer, characterized by the presence of an ovarian tumour and numerous peritoneal metastases. Furthermore, interaction of the ID8 cells with the ovarian microenvironment imparts a more aggressive phenotype. In the present study, we compared IP and intrabursal (IB) injection of ID8 cells into $SPARC^{-/-}$ and WT mice to determine if absence of host SPARC alters progression in the IB model.

Methods: IB and IP injections of $1.0X10^6$ ID8 cells into $SPARC^{-/-}$ and WT age-matched controls were conducted under the guidelines of the Canadian Council for Animal Care (n=10/group). Mice were assessed until they became moribund due to ascites formation. Fibrillar collagen content was determined by trichrome and picosirius red staining of formalin fixed paraffin embedded sections.

Results: $SPARC^{-/-}$ mice injected IP had significantly ($p<0.05$) reduced survival compared to WT controls, whereas no differences in survival were observed in IB-injected mice. Despite a more rapid disease progression in $SPARC^{-/-}$ IP-injected mice, these mice had fewer abdominal lesions as compared to IP-injected WT mice. In contrast, no difference in abdominal lesions was observed in IB-injected animals. Since SPARC affects collagen remodelling and inhibits adipocyte differentiation, fibrillar collagen and adipocyte content were examined in ovarian tumours from IB-injected mice. Fibrillar collagen content in tumours from IB-injected mice showed no differences due to genotype, whereas preliminary assessment indicated higher adipocyte content in tumours from $SPARC^{-/-}$ mice.

Conclusions: These findings indicate that anti-tumour effects of stromal-derived SPARC are overcome by factors present in the ovarian/oviductal microenvironment.

ABSTRACT #P-A3

FEMALE ONCOLOGY PATIENTS AND FERTILITY PRESERVATION: ONE FERTILITY CENTRE'S EXPERIENCE

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Objective: As the incidence of cancer continues to rise in women of reproductive age, future fertility is becoming a growing concern. Many patients are being referred to fertility clinics to explore their options for fertility preservation before or during their cancer treatment. The objective of this study was to describe characteristics of the patient population, assess aspects of the quality of care received, and evaluate treatment decisions and outcomes.

Methods: Female oncology patients, who were seen in consultation at a hospital-based fertility clinic between July 2005 and July 2008 to discuss fertility preservation, were invited to participate in the study by consenting to a chart review. Data for the chart review was collected directly from patients' medical charts. This included patient demographics such as age, relationship status, and reproductive history (e.g. gravidity, parity); efficiency of the referral process (e.g. referral date, appointment date, cancer treatment date); the referral source; oncology details including type of cancer and type of treatment planned or received; patient's decision regarding fertility preservation at time of consultation; patient's final decision regarding fertility preservation; and treatment outcomes.

Results: Of 103 patients who were referred to the fertility clinic during this time period for fertility preservation, 68 patients could be contacted. Fifty patients consented to the chart review. The mean age (range) at time of consultation was 33 (22 to 42). Thirty-eight patients (76%) were in a relationship (married or with a partner), and 33 (66%) were nulliparous. The mean time period (range) from date of referral to date of consultation was 1.69 (0.14 to 13.86) weeks. The most common types of cancers among these patients were breast (28/50, 56%), ovary (7/50, 14%), and hematologic (7/50, 14%). The largest number of referrals (22/50, 44%) came from medical oncologists. After consultation, the majority of patients were undecided (35/50, 70%) on which treatment option, if any, to pursue. Ultimately, fourteen patients (28%) proceeded with cryopreservation through IVF, one patient (2%) underwent a fresh IVF cycle, and two patients (4%) chose ovarian suppression with OCP. To date, only two patients have been seen in follow-up for use of frozen embryos, neither of whom conceived.

Conclusions: The results of this study indicate that female oncology patients referred for fertility preservation consultation were seen in a timely fashion. Many patients left their initial consultation undecided as to whether to pursue any of the treatment options discussed. In the end, almost one-third of all the patients seen chose to undergo fertility preservation therapy. Future follow-up of this population is needed to assess the outcomes of cryopreservation for fertility preservation.

ABSTRACT #P-A4

RISK OF MALIGNANCY INDEX: HOW FEASIBLE IS IT TO CALCULATE THE SCORE?

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Objective: To evaluate the feasibility of Risk of Malignancy Index triage at our centre using CA-125 and ultrasound (US) reporting, as measured by the proportion of patients for whom an RMI score could be determined.

Methods: **A**ll patients referred to a single gynecologic oncologist for an adnexal mass seen by a between 2005 – 2009 were reviewed. Patients excluded from the study were those with known ovarian cancer, prophylactic surgery candidates, or those without an US evaluation. Patient charts were reviewed to confirm menopausal status, CA-125, and US results. For each US report, morphology features (multilocularity, bilateraliyy, solid components, ascites, metastases) were classified as "reported/present," "reported/absent," or "not reported". US reports were considered complete if they commented on all 5 features. We compared the proportion of complete US reports between external and internal examinations (Fisher's exact test). We determined the proportion of patients for whom an RMI score was calculable. This study received institutional ethics approval.

Results: 376 patients were eligible. CA-125 was determined in 84%. US reports were complete in only 7.7% of patients (external-5.9% vs. interal-10.4%,p=0.08), although 41% more US reports contained enough information to be considered suspicious by the RMI (external-44.8% vs. internal-34.9%, p=0.04). The RMI score could be calculated in 37.8% of patients.

Conclusions: The high rate of incomplete US reporting and non-routine measurement of CA-125 renders RMI-based triage for surgery inconsistently feasible. Further studies should explore the potential benefit of synoptic reporting of US and CA-125 results.

ABSTRACT #P-A5

PREVALENCE OF BRCA1 AND BRCA2 GERM LINE MUTATIONS AMONG WOMEN WITH CARCINOMA OF THE FALLOPIAN TUBE

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Objective: The purpose of this study is to determine the prevalence of BRCA1 and BRCA2 mutations among unselected women with carcinoma of the fallopian tube.

Methods: Two series of women diagnosed with carcinoma of the fallopian tube were studied. Women identified from the Ontario Cancer Registry who were diagnosed with fallopian tube cancer between 1990 and 1998 and between 2002 and 2004. A second, hospital-based series was identified at Cedars Sinai Medical Centre, Los Angeles, California. These women were diagnosed between 1991 and 2007. Each subject was approached to provide her family history and ethnic background and to provide a blood sample for genetic testing for mutations in the BRCA1 and BRCA2 genes.

Results: In total, 108 patients with fallopian tube cancer were recruited (70 from Ontario and 38 from Los Angeles). Thirty-three patients (30.6%) were found to have a deleterious mutation; 23 in BRCA1 (21.3%) and 10 in BRCA2 (9.3%). The prevalence of mutations was 55.6% in Jewish women and was 26.4% in non-Jewish women. A family history of ovarian or breast cancer was positive for 24 women (23.3%); of these, 14 had a mutation (58.3%). Fourteen (14.4%) of the patients had a previous history of breast cancer; of these, 10 (71.4%) had a mutation. 40.3% of the women who were diagnosed with fallopian tube cancer before age 60 had a mutation, compared with 17.4% of the women diagnosed at age 60 and above.

Conclusions: Approximately 30% of women with fallopian tube cancer have a mutation in BRCA1 or BRCA2. The highest frequencies of BRCA mutations were seen in women with fallopian tube cancer diagnosed under age 60, in Jewish women, in women with a family history of breast or ovarian cancer, and in women with a personal history of breast cancer. All patients diagnosed with invasive fallopian tube cancer should be considered candidates for genetic testing.

ABSTRACT #P-A6

VEPH1 IS A NOVEL REGULATOR OF TGF-ß SIGNALING IN OVARIAN CANCER CELLS.

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ABSTRACT AVAILABLE IN HARDCOPY VERSION ONLY

ABSTRACT #P-B1

GLYCOGEN SYNTHESIS EVALUATION IN LIVERS OF C57BL/6J FETAL MALE MICE SUBJECTED TO ANTENATAL DIETARY RESTRICTION

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Our lab has previously developed a mouse model of antenatal dietary restriction (ADR) by imposing a 30% caloric restriction from day 6.5 to 18.5 of gestation on pregnant C57BL/6J (B6) mice. At day 18.5 of gestation, B6 fetuses from ADR mothers possess increased expression of gluconeogenic enzymes and elevated blood glucose. At as early as 3 months of age, the male offspring show signs of insulin resistance and glucose intolerance in response to ADR.

Objectives:
B6 male fetuses subjected to ADR show signs of increased glucose production prior to birth. Does the insulin resistance they develop in later life also begin prior to birth?

1) To develop a protocol evaluating glycogen production in mouse fetal livers at day 18.5 of gestation.
2) To use this protocol as an indication of insulin sensitivity in C57BL/6J fetal male mice subjected to ADR.

Methods: Pregnant B6 mice will be either fed *ad libitum* throughout gestation or subjected to an antenatal dietary restriction of 30% caloric reduction from day 6.5 to 18.5 of gestation. On day 18.5 of gestation between 0900-1100 hours, the B6 mouse is administered an intraperitoneal injection of 30 μCi of [U-^{14}C]D-glucose in isotonic NaCl solution. After equilibration of [U-^{14}C]D-glucose for 6 hours allowing entry into maternal blood circulation, transfer across the placentae and uptake by fetuses, the animals were sacrificed. The fetal livers will be extracted, solubilized in 10N KOH and subsequently boiled with cold glycogen carrier. Glycogen will be precipitated with two volumes of ethanol overnight on ice. Precipitated glycogen will be centrifuged at 10,000*g* for 10 min. The pellets will be washed with 66% ethanol, resuspended in 0.5ml water and counted by scintillation counting. All procedures involving laboratory animals were approved by the Toronto Centre for Phenogenomics Animal Care Committee.

Results: We have established an *in vivo* protocol to measure differences in glycogen synthesis in mouse fetal livers at day 18.5 of gestation.

Conclusions: Our glycogen synthesis evaluation protocol was used to measure glycogen production in B6 mice that were either subjected to antenatal dietary restriction or fed *ad libitum* during pregnancy.

Funded by: CIHR

ABSTRACT #P-B2

PLACENTAL LOCATION AND NEWBORN WEIGHT

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Introduction: While the uterus receives the majority of its blood from the uterine artery, blood flow to various sites of the uterus is not uniformly distributed. During pregnancy, the site of placental implantation (lateral vs. central) within the uterus is an important determinant of placental blood flow. Abnormal uterine artery flow velocity is more common among women with a lateral placental location as a central placenta receives equal blood flow from both the left and right uterine arteries. In theory, this could account for a lower birth weight among neonates with laterally implanted placentas compared to those with centrally implants placentas. Previous studies have implicated placental location in fetal growth, preterm birth, fetal malposition and the development of preeclampsia, however these have been small in size and have yielded conflicting results.

Objective: Using a large retrospective cohort design, we are investigating whether placental location at the time of the second trimester ultrasound is a predictor of birth weight among singleton infants. Secondary outcome measures include the development of maternal preeclampsia or gestational hypertension and NICU admission.

Methods: We are reviewing the charts of all the women who delivered at St. Michael's Hospital between July and October 2009 (n=800). Multiples and infants with a major congenital anomaly or chromosomal disorder were excluded. Measured covariates included maternal age, parity, place of birth, weight, hypertension, smoking/drug use and pre-gestational and gestational diabetes.

Conclusions: This is a work in progress. We hope that placental implantation site at the time of the second trimester ultrasound may prove to be useful as a simple and routine predictor of a pregnancy at risk for IUGR. This additional information would allow for more frequent ultrasonography and fetal surveillance to potentially optimize pregnancy outcome.

ABSTRACT #P-B3

PERINATAL OUTCOMES IN A PRENATALLY DIAGNOSED GASTROSCHISIS COMPLICATED WITH GASTRIC DILATATION

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Objective: To determine the prognostic significance of gastric dilation in fetuses with isolated gastroschisis.

Methods: Retrospective study with REB approval of 98 singleton pregnancies with a prenatal diagnosis of gastroschisis delivering at Mount Sinai Hospital and receiving postnatal care at the Hospital for Sick Children Jan 2001-Feb 2010. All maternal and fetal prenatal records, newborn postnatal records, and pediatric surgical ward admission records were reviewed. Gestational age at delivery, mode of delivery, indication for Cesarean section, presence or absence of meconium-stained amniotic fluid, birth weight centile, Apgar scores at 1 and 5 mins, Umbilical artery PH, time to full enteral feeding, length of hospital stay, need for bowel resection, bowel atresia, and bowel necrosis were recorded. Individual adverse outcomes and a composite adverse score will be compared by presence or absence of gastric dilation prior to delivery. Gastric dilation was determined based on a gestational age-specific normogram.

Results: Thirty-two of 98 (32.7%) patients exhibited gastric dilation prior to delivery. There were no significance differences between the two groups with respect to gestational age at delivery, birth weight, Apgar scores at 1 and 5 minutes. The remaining analysis of the significance of gastric dilation will be presented at the meeting.

Conclusion: Pending

ABSTRACT #P-B4

COMBINATIONS OF FIRST AND SECOND TRIMESTER MATERNAL SERUM BIOCHEMICAL MARKERS AND PREDICTION OF ADVERSE PREGNANCY OUTCOMES: SYSTEMATIC REVIEW AND META-ANALYSIS

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Objective: To review pregnancy outcomes associated with combinations of abnormal first and/or second trimester maternal serum biochemical markers used in routine prenatal testing for Down syndrome and open neural tube defects.

Methods: Medline, EMBASE, Cochrane library, and reference lists of included articles were searched for English language articles published between 1970-2009 with reference to combinations of maternal serum markers and adverse pregnancy outcomes. Two authors independently selected relevant articles in which combinations of maternal serum biochemical markers were analyzed in terms of their ability to predict adverse pregnancy outcomes; specifically pre-eclampsia, intrauterine growth restriction, and stillbirth >24 weeks gestation, in unselected populations. Study characteristics, quality assessments, and results were extracted independently by two authors.

Results: Combinations of any 2 or more of 6 possible serum markers were evaluated. 8 studies testing 106,324 pregnant women for the outcome of pre-eclampsia, 10 studies testing 156,554 women for development of intrauterine growth restriction as an adverse pregnancy outcome, and 9 studies testing 231,783 women for the outcome of stillbirth >24 weeks gestation met the selection criteria. Final statistical analysis is pending.

Conclusions: In spite of the significant volume of literature on this topic, methodological factors severely limit the ability to systematically review this data. Combinations of serum markers afford better prediction for adverse perinatal outcome than do single markers, and there is a positive relationship between the numbers of abnormal markers and the risk for adverse outcome. Large unselected cohorts, with standardized definitions of both the index tests (serum markers) and outcomes would provide much more reliable data on which to base the performance of these screening parameters.

ABSTRACT #P-B5

CONGENITAL MEGALOURETHRA: PRENATAL DIAGNOSIS AND POSTNATAL/ AUTOPSY FINDINGS. REPORT ON 9 CASES

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Objective: Congenital Megalourethra is a rare urogenital malformation characterized by dilation and elongation of the penile urethra associated with distended bladder and in some cases renal damage. Postnatal complications include voiding and erectile dysfunction, as well as renal insufficiency. To date there are only a few case reports of this condition diagnosed antenatally. We report our experience in the prenatal diagnosis and postnatal findings in 9 cases of congenital megalourethra.

Methods: Over a five-year period, 9 cases of megalourethra were diagnosed prenatally, between 13 and 24 weeks' gestation, 4 in one centre and 5 in other centres. The key prenatal ultrasound findings were megacystis and dilated/elongated urethra. Severe oligohydramnios was diagnosed only in two cases. The findings on fetal autopsies on the interrupted pregnancies and the clinical follow-up on the liveborns were reviewed.

Results: Nine fetuses with megalourethra were identified at a median gestational age of 19 weeks (range 13-24 weeks' gestation) and confirmed at follow-up/autopsy. Pregnancy was terminated electively in 3 cases and continued in 6. All cases presented with distended bladder, elongated and distended urethra. Some had echogenic kidneys. Of the 6 liveborn cases, one died neonatally due to pulmonary hypoplasia. Of the 5 cases currently alive, all have dysfunctional urethra, requiring vesicostomy, 3 had undescended testes, two suffer from end stage renal disease and one has impaired renal function. Of the 3 pregnancies that were interrupted, two had oligohydramnios, all cases had megaurethra and hypoplasia of both corpora cavernosa and spongiosa. All cases had a normal male karyotype.

Conclusion: Congenital megaurethra is caused by abnormal development of the corpora spongiosa and cavernosa. When amniotic fluid volume is normal, survival is possible. However, all liveborn fetuses suffer urethral dysfunction and half have renal dysfunction/failure. The prognosis regarding sexual function is guarded. Megalourethra should be considered in all cases presenting prenatally with megacystis and normal amniotic fluid volume. Detailed ultrasound of the external genitalia should be done in these cases for accurate diagnosis.

ABSTRACT #P-B6

THE ROLE OF TRANVAGINAL EARLY ANATOMY ULTRASOUND IN THE MANAGEMENT OF FETUSES WITH LARGE NUCHAL TRANSLUCENCY

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Objective: Large nuchal translucency (NT) is associated with a higher risk for chromosomal aberrations and fetal structural anomalies. Our objective was to examine the role of transvaginal early anatomy ultrasound (TEAU) in the management of pregnancies with large fetal nuchal translucency.

Methods: The study was conducted at Sunnybrook Health Sciences Centre and included data from women who had a TEAU due to a large fetal nuchal translucency (>3.0 mm) between April 2007 and August 2009. TEAU was performed at 13-15 weeks of gestation by a single experienced physician and included detailed fetal anatomy examination comparable to the standard ultrasound at 19-21 weeks. Information from the routine anatomy ultrasound, chromosomal testing, autopsy findings, and pregnancy outcome was collected prospectively.

Results: 68 fetuses with a large NT were examined during the study period. In 18 cases (26%), anomalies were detected during the TEAU. 13 of the 18 women were found to have fetuses with major cranial and/or cardiac anomalies and the pregnancies were terminated before the standard 19 weeks ultrasound was completed. In 5/13 who terminated, there were no chromosomal aberrations; in 8/13, chromosomal aberrations including T22, T18, 45XO, and other deletions and unbalanced translocations were found; and interestingly, FISH was normal in 9/13 cases. Of the 5 women with fetal anomalies who continued with the pregnancy, there were 2 cases of IUFD, 1 case of Down Syndrome, and 2 cases with mild, non-lethal abnormalities. In total, 9 cases of chromosomal aberrations were diagnosed and all were found to have fetal structural anomalies. Three cases had an abnormal outcome which was not detected by TEAU or by the routine anatomy scan. These fetuses had late onset or ultrasound undetectable anomalies such as Milroy's Syndrome, congenital nephrotic syndrome and dysmorphic features. TEAU had a 95% detection rate of ultrasound-detectable fetal anomalies when compared to pregnancy outcome.

Conclusions: The current study shows that a first trimester targeted TEAU of fetuses with an increased NT is highly effective in detecting fetal anomalies and can contribute to timely parental decision regarding the future of the pregnancy, especially when a structural anomaly is clearly diagnosed. Fetal anomalies are common in fetuses with large NT and TEAU is suggested as the first line investigation in the management of the condition.

ABSTRACT #P-C1

GENERATION OF SOLUBLE FORMS OF ENDOGLIN FOR STRUCTURAL STUDIES

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ABSTRACT AVAILABLE IN HARDCOPY VERSION ONLY

ABSTRACT #P-C2

ANGIOGENIC RESPONSE OF THE HUMAN PLACENTA TO HEPARIN: IMPLICATIONS FOR THE PREVENTION OF PRE-ECLAMPSIA?

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ABSTRACT AVAILABLE IN HARDCOPY VERSION ONLY

ABSTRACT #P-C3

ROLE OF FACTOR INHIBITING HIF-1 (FIH-1) IN THE SELECTIVE REGULATION OF HIF-1 TARGET GENES IN PREECLAMPSIA

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Objectives: Preeclampsia is a pregnancy-associated disorder characterized by placental hypoxia and increased expression of hypoxia inducible factor-1α (HIF-1α). HIF-1 is a major regulator of oxygen homeostasis, which we have previously reported plays an important role in the placental development and function. Factor inhibiting HIF (FIH) is an asparaginyl hydroxylase that regulates HIF-1 transcriptional activity. Additionally, FIH was found to selectively regulate the expression of specific HIF-1 targets, including vascular endothelial growth factor (VEGF) and prolyl hydroxylase domain 3 (PHD3), both of which have active roles in the human placenta. However, whether FIH selectively regulates VEGF and PHD3 in the placenta and whether this rheostat is altered in PE is unknown. Herein, we investigated FIH, PHD3 and VEGF expression in early-onset PE and examined the role of FIH in regulating PHD3 and VEGF expression.

Methods: Early-onset PE (n=26), age-matched preterm control (PTC) (n=16) and term control (TC) (n=11) placentae were used. FIH, PHD3 and VEGF mRNA and protein levels were assessed by qRT-PCR and Western blot (WB) analyses, respectively. FIH, PHD3 and VEGF spatial localization was also examined by immunofluorescence (IF) staining of placental sections. To establish the direct effect of FIH on PHD3 and VEGF, siRNA technology was used to silence FIH gene expression in JEG-3 choriocarcinoma cells. siRNA duplexes (30 nM) were transfected using a liposome-based reagent; FIH, PHD3 and VEGF expression levels were then examined by qRT-PCR and WB analyses.

Results: In early-onset PE, FIH mRNA and protein levels were significantly decreased relative to controls. Conversely, PHD3 mRNA and protein levels were dramatically increased in PE relative to controls. However, while VEGF mRNA levels were also increased in PE relative to PTC, VEGF protein levels were decreased. Hence, in PE, PHD3 and VEGF mRNA exhibited an inverse pattern of expression relative to FIH. IF staining indicated that, in PTC, FIH was expressed in the cytotrophoblast (CT) cells where it co-localized with both PHD3 and VEGF. In PE, while FIH expression was weaker than in PTC, its localization was not greatly altered. Conversely, in PE, PHD3 was minimally expressed in the trophoblast cells and was mainly expressed around the vasculature, while VEGF expression in the trophoblast was weak and diffuse. Importantly, minimal co-expression of FIH with PHD3 and VEGF was observed in PE. In addition, FIH gene silencing in JEG-3 cells resulted in increased expression of PHD3 and VEGF mRNA levels, suggesting a role for FIH in mediating the expression of these genes.

Conclusion: Our data introduces FIH as a novel player in the regulation of HIF-1 in the human placenta. In particular, our data provide the first evidence that FIH regulates PHD3 and VEGF expression in trophoblast cells. Moreover we show that FIH expression is decreased in PE, necessarily contributing to the aberrant PHD3 and VEGF expression observed in this pathology.

Funded by: CIHR/IGH and Genesis Research Foundation

ABSTRACT #P-C4

HEPARIN ELEVATES SFLT-1 SECRETION AND IMPAIRS VILLOUS TROPHOBLAST PHYSIOLOGY IN A DOSE-DEPENDENT MANNER: IMPLICATIONS FOR THE PREVENTION OF SEVERE PREECLAMPSIA?

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ABSTRACT AVAILABLE IN HARDCOPY VERSION ONLY

ABSTRACT #P-C5

A STORY OF LIFE AND DEATH: THE DUAL ROLE OF MTD/BOK IN TROPHOBLAST CELL FATE

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Objective: We have previously reported on the expression and function of Mtd/Bok (Mtd) in the human placenta and shown that levels of Mtd are significantly increased in preeclampsia, a serious disorder of pregnancy associated with both increased trophoblast cell proliferation and death. Mtd is a proapoptotic molecule however; we have recently reported on Mtd regulation of cell cycle in trophoblast cells. The **objective** of this study was to examine the dual role of Mtd with respect to trophoblast cell proliferation and apoptosis in physiological and pathological conditions, including preeclampsia and molar pregnancies.

Methods: Human placental samples were collected from normal pregnancies throughout gestation (n=60), severe early onset preeclamptic (n=40), molar pregnancy with a co-existing twin (n=2), and age-matched control placentae (n=40). Fluorescence immunohistochemistry was used to assess co-localization of Mtd with markers of proliferation (Ki67, cyclinE1) and apoptosis (nuclear morphology). Subcellular localization of the Mtd isoforms was evaluated by subcellular fractionation and co-localization of Mtd with mitotracker, a mitochondrial marker, in choriocarcinoma JEG3 cells. The effect of Mtd-L on cyclin E1 was assessed by knockdown studies in first trimester explants (antisense strategy) and in the HEK cell line (siRNA), and by overexpression using a doxycyline inducible Mtd-L HEK cell line.

Results: Mtd was expressed in both proliferative and apoptotic trophoblast cells across gestation and in the preeclamptic and molar pathologies. The main isoform of Mtd associated with trophoblast proliferation was Mtd-L, the full length isoform, which preferentially localized to the nuclear compartment in proliferating cells while during apoptosis it switched localization to the cytoplasm where it associated with mitochondria. Mtd expression in proliferating cells co-localized with cyclin E1, a G_1/S phase cell cycle regulator. Furthermore, functional assessment of Mtd-L, by induced Mtd-L expression or Mtd-L specific knock-down in early first trimester villous explants or HEK cell line, revealed a direct effect of Mtd-L on cyclin E1 expression. During normal placentation expression of cyclin E1 and the cell cycle inhibitor p27 were found to display opposing patterns of expression, whereby cyclin E1 expression decreased with gestational age as levels of p27 increased. Preeclampsia and molar pregnancy displayed increased levels of both Mtd and cyclin E1. In addition both pathologies were associated with altered expression of p27. In preeclampsia the p27 protein was upregulated but localized to the cytoplasm, whereas in molar pregnancy the levels of p27 were decreased.

Conclusion: Mtd is involved in the regulation of both proliferation and cell death in the human placenta. The increased expression of Mtd and cyclin E1 as well as the altered regulation of p27 seen in preeclampsia and molar pregnancy may contribute to both the increased apoptosis and hyperproliferative phenotype of these disorders.

Funded by: CIHR, OWH/IGH

ABSTRACT #P-D1

FLUORESCENT IN SITU HYBRIDIZATION (FISH) IS AN EFFECTIVE TOOL TO DETECT UNSUSPECTED TRISOMIES IN HYDROPIC GESTATIONS

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Objectives: Histological assessment of products of conception (POC) with hydropic changes has always been challenging for pathologists on a morphological basis since there is significant overlap between the features of partial moles (PM) and hydropic degeneration (HD). Hydropic changes can also be seen in gestations with trisomy and these cannot be diagnosed on morphology alone. These differential diagnoses have significant different implications for patient care and management. Therefore, the use of ancillary studies plays a major role in establishing a definitive diagnosis and flow cytometry has been considered the gold standard to identify triploidy and confirm the diagnosis of partial mole. However, flow cytometry is unable to detect cases of trisomy. FISH is a molecular cytogenetic technique that has been used more recently in the assessment of POCs with hydropic changes to identify triploidy in partial moles. Due to the use of chromosome-specific probes, FISH also allows the detection of specific trisomies. The aim of this study was to assess the efficacy of FISH as a novel ancillary technique in identifying trisomies and to determine the frequency of unsuspected trisomies in POCs with hydropic changes.

Methods: We identified 92 cases of POC accessioned in the Department of Pathology at Sunnybrook HSC between 2007 and 2010 for which FISH study was requested. 24 complete moles for which the diagnosis is based on morphology and immunostaining were excluded. The study cohort consists of 68 cases of POC for which the differential diagnoses included PM and HD. FISH analysis was performed on 4-μm formalin-fixed paraffin-embedded tissue sections using the Vysis AneuVysion Prenatal Test specific for chromosomes X, Y, 13, 18 and 21 as well as a probe specific for chromosome 16 (Abbott Molecular, Des Plaines, IL) using established protocols.

Results: FISH results revealed the presence of an unsuspected trisomy in 7/68 cases (10.3%). There were four cases of trisomy 16, two cases of trisomy 18 and one case of trisomy 21. FISH revealed triploidy in 32/72 cases which confirmed a diagnosis of PM. In the remaining 29 cases, the FISH results were within normal range and a diagnosis of HD was rendered based on morphology.

Conclusions: FISH is an effective tool in the detection of trisomies and the discernment of the cause of the miscarriage; an unsuspected trisomy was identified in a significant proportion (10.3%) of POCs with hydropic changes submitted for FISH analysis. A diagnosis of gestation with a trisomy can have significant clinical implications. Thus, FISH, rather than flow cytometry, should be considered as the new gold standard for the evaluation of hydropic POCs.

ABSTRACT #P-D2

UTILIZATION OF FLUORESCENCE IN SITU HYBRIDIZATION (FISH) METHODOLOGY FOR EVALUATION OF NUCLEAR ORGANIZATION IN HUMAN SPERMATOZOA

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Objective: Human spermatozoa have a unique, well-organized nuclear architecture that differs from somatic cells. Sperm nuclear organization is distinguished by telomeric dimerization, which results in a looped chromosomal configuration. Consequently, improperly packaged sperm chromatin will have a high probability of disrupting the extremely structured sequence of fertilization. The evaluation of nuclear organization in spermatozoa is difficult due to the compactness of their chromatin and the absolute necessity to perform chromatin decondensation, which itself could alter native sperm nuclear architecture. The purpose of our study was to identify a sperm nuclear decondensation (ND) protocol that results in minimal disturbance of sperm architecture. We have also evaluated the telomere-telomere interaction of chromosome 1 in men with proven fertility as compared to infertile men.

Methods: Three previously described ND protocols for FISH on human spermatozoa were evaluated in this study: 1) 0.5N NaOH; 2) 10mM dithiothreitol (DTT) and lithium diiodosalicylate (LIS); and 3) 2.5mM DTT and heparin. These ND methods were followed by direct labeling with sub-telolomeric (ST) arm specific Aquarius 1q (green) -1p (red) probes. Images were acquired using unbiased sampling and were analyzed employing a Visiopharm Integrator System. The distance between ST probes was calculated after taking into account the effect of decondensation and was compared to a normalized ST distance. The results are expressed as mean ± SD.

Results: Spontaneous Comparison of ND protocols is provided in the table:

Protocol	Head area (μm²)	Fold increase	ND (%)	Mild ND (%)
Neat sample	12.8 ± 2.7*	n/a	n/a	n/a
1. NaOH	30.2.± 7.6	2.3 ± 0.6**	94	83**
2. DTT-LIS	50.2 ± 25.7	3.9 ± 2.0	95	28
3. DTT-heparin	48.1 ± 13.3	3.8 ± 1.0	95	15

* P <.001 between neat and all protocols; ** P <.001 between NaOH vs. DTT-LIS and DTT-heparin protocols. Mild decondensation was defined as 1.5-3 fold increase in nuclear area. Only 40% of cells in fertile and infertile groups had the expected normalized ST distance of <0.6μm between two arms of chromosome 1, representing a looped chromosome configuration.

Conclusions: Combined denaturation and decondensation with 0.5N NaOH demonstrated superior results, providing uniform mild ND with over 80% of nuclei suitable for FISH sperm architecture assessment. There was no difference in telomere-telomere dimerization of chromosome 1 in men with fertility and infertility. This may be related to the nature of this particular chromosome as it has the largest amount of chromatin mass.

ABSTRACT #P-D3

MODULATION OF NUCLEAR FACTOR KAPPA β SIGNALING BY NALP5 (Work in Progress)

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Objective: A specific pathway for NALP5 in germ cells has not been determined. Based on what is known about other NALPs and their effect on transcription factor Nuclear Factor Kappa β (NFκB) regulation, we hypothesize that NALP5 may be involved in facilitating the activation of NFκB, essential for total embryonic genome activation. We anticipate that NALP5 deficient zygotes will have a decrease in NFκB nuclear translocation and therefore, DNA binding activity. This can be due to insufficient caspase activity, which is required for efficient translocation and transcriptional response of NFκB. Thus, we propose to interrogate this pathway and confirm key findings in wild-type (WT) and NALP5 deficient (KO) zygotes. Whether NALP5 acts independently or in association with other players downstream of the pathway will be examined.

Methods: We will investigate whether NALP5 facilitates caspase activation, which results in NFκB nuclear translocation and gene activation. Initially, we will create a profile of caspase activity during normal development to determine if the activity in oocytes, early (20-22hr) or late (26-28hr) zygotes and two-cell embryos differentially modulates preimplantation embryo development using fluorescent caspase substrate assays. We will evaluate the extent of nuclear translocation of total NFκB in early and late zygotes by immunocytochemistry. More importantly, phosphorylation of NFκB is required for its translocation to the nucleus; therefore, we will first analyze phosphorylated NFκB translocation in wild-type zygotes to then compare to NALP5 deficient zygotes.

Results: Immunocytochemistry showed altered NFκB subunit, RelA (p65) expression levels between WT and KO oocytes and zygotes. This indicates that kinetics of NFκB translocation may be altered in NALP5 KO zygotes. In addition, we have established pattern of phosphorylated p65 in early zygotes showing pronuclear membrane staining and progressive accumulation in pronuclei at later zygote stages Also, total caspase activity in WT conditions showed similar values for oocytes and zygotes, but reduction in two-cell embryos, indicating need for caspases during transition from oocyte to embryos. Further work is in progress to compare these to KO models.

Conclusions: These studies will help clarify the molecular pathways involved in NALP5 activity including its role in embryo development, as well as, embryo loss in humans. They will form a foundation for future endeavors that could lead to clinically applicable rescue approaches for patients with repeated failures of assisted reproductive technology.

Funded by: Supported by grants from the Canadian Institute for Health Research (CIHR) and Genesis Research Foundation.

ABSTRACT #P-D4

CHARACTERIZATION OF THE OOCYTE PHENOTYPE CAUSED BY MCL-1 DEFICIENCY

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Objective: To uncover the role of Mcl-1 in germ cell survival and mitochondrial function.

Methods: We have confirmed Mcl-1 expression in mouse ovaries using immunohistochemistry and Western blot analyses. A floxed Mcl-1 mouse line was crossed with a transgenic mouse line containing Cre under the control of the zona pellucida (ZP-3) promoter to ensure oocyte specific excision of Mcl-1. At 3 weeks of age, superovulated oocytes from females with complete germ cell-excision of Mcl-1 were compared to wild type controls to assess mitochondrial functionality. Oocytes were stained with Mitotracker and Depsifer to determine functional mitochondrial number. Mitochondrial copy number was measured using qPCR to gauge amounts of mtDNA and number of replicating mitochondria. Total levels of Reactive Oxygen Species (ROS) were determined in addition to those produced specifically by mitochondria (Mitosox). Levels of NAD(P)H and FAD were also measured as markers of mitochondrial function. Mcl-1-deficient and wild type control oocytes were also sent for Electron Micoroscopy imaging to determine ultrastructural differences.

Results: At 3-4 weeks of age, Mcl-1 germ cell-deficient females show little difference in number of oocytes ovulated when compared to controls. Mitotracker and DePsifer stains confirmed a reduction in number of functional mitochondria in Mcl-1-excised germ cells, yet qPCR revealed no difference in mitochondrial copy number. ROS and Mitosox levels were increased in Mcl-1-deficient oocytes and NAD(P)H levels were reduced. EM images also revealed an increase in lysosomal formation in the Mcl-1-deficient oocytes and this was confirmed with an increase in Lysotracker staining.

Conclusions: Our data suggests a role for Mcl-1 as a pro-survival factor in the regulation of oocyte fate and mitochondrial function. The absence of Mcl-1 results in an overall reduction in mitochondrial functionality as indicated by the increase in ROS levels and reduction in Mitotracker and DePsifer quantitation. These impaired mitochondria would most likely be cleared using the cell's own autophagic machinery, in a process termed mitophagy, which can be explained by the apparent increase in lysosomal formation.

ABSTRACT #P-D5

TAP73 KNOCKOUT MICE AS A MODEL OF REPRODUCTIVE AGING

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ABSTRACT AVAILABLE IN HARDCOPY VERSION ONLY

ABSTRACT #P-E1

IN VITRO DERIVATION OF FUNCTIONAL INSULIN-PRODUCING CELLS FROM HUMAN FIRST TRIMESTER UMBILICAL CORD STEM CELLS

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ABSTRACT AVAILABLE IN HARDCOPY VERSION ONLY

ABSTRACT #P-E2

ENHANCED FUNCTION OF PREFRONTAL SEROTONIN 5-HT$_{2A/C}$ RECEPTORS IN A RAT MODEL OF PSYCHIATRIC VULNERABILITY

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Prefrontal serotonin 5-HT$_{2A/C}$ receptors have been linked to the pathogenesis and treatment of affective disorders. However, it remains controversial whether enhanced 5-HT$_{2A/C}$ receptor binding in the prefrontal cortex predicts the presence of a psychiatric illness and whether a change in this measure predicts the effectiveness of treatment. Instead, it has been hypothesized that 5-HT$_{2A/C}$ signaling efficiency may be a better predictor of vulnerability to psychiatric illness.

Objective and Methods. Here, we examine the effects of 5-HT$_{2A/C}$ receptors in a rat model of psychiatric vulnerability using electrophysiology, gene expression, and behavior.

Results. Following the early stress model of chronic maternal separation, we found that serotonin has atypical 5-HT$_{2A/C}$ receptor-mediated excitatory effects in the adult prefrontal cortex, and that direct stimulation of 5-HT$_{2A/C}$ receptors evokes larger excitatory inward currents in layer V neurons. Further, in response to 5-HT$_{2A/C}$ receptor stimulation, adult animals with a history of early stress exhibit heightened prefrontal network activity, enhanced immediate early gene expression, and potentiated head shake behavior. These changes arise in the absence of any major alteration of prefrontal 5-HT$_{2A/C}$ receptor binding or mRNA expression. Our microarray and quantitative PCR results provide insight into the molecular changes that accompany such enhanced 5-HT$_{2A/C}$ receptor function in adult animals following early stress. We observed persistent prefrontal transcriptome changes, including significant alterations in genes associated with cellular excitability, G-protein signaling and neuroplasticity.

Conclusions. Together, our results demonstrate enhanced prefrontal 5-HT$_{2A/C}$ receptor function and persistent alterations in prefrontal gene expression in a rat model of psychiatric vulnerability.

ABSTRACT #P-E3

THE EFFECT OF CHRONIC MATERNAL ADVERSITY (CMA) ON ACTIVITY AND ATTENTION IN JUVENILE GUINEA PIG OFFSPRING

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Objectives: Human studies of maternal adversity have demonstrated attentional and behavioural problems in children as well as elevated cortisol levels. Previously we have demonstrated that male and female juvenile offspring of mothers exposed to chronic maternal adversity (CMA) display elevated basal cortisol levels. Additionally we found that adult male offspring exhibit increased locomotor activity and adult female offspring exhibit decreased attention. As this altered behaviour (hyperactivity and inattention) typically emerge in childhood in humans, we hypothesized that CMA will lead to increased activity in juvenile male offspring and decrease attention in juvenile female offspring.

Methods: Pregnant guinea pigs were exposed to a random stressor every other day over the second half of gestation and from postnatal day (pnd) 1 until weaning on pnd25. A group of control animals remained undisturbed throughout. At weaning, male and female offspring were pair housed by sex. On pnd28, offspring were tested on the Elevated Plus Maze (EPM) to assess locomotor activity and anxiety-like behaviour. On pnd29 and pnd 35 offspring underwent Prepulse Inhibition (PPI) testing to assess attention.

Results: *Elevated Plus Maze*: In juvenile male offspring, CMA resulted in elevated activity ($P<0.01$) as indicated by an increase in total arm entries. There was no effect of CMA on activity levels in female offspring. Additionally, CMA had no effect on the percent of movement that occurred in the open arms in either sex indicating no difference in anxiety-induced behaviour. *Prepulse Inhibition*: There was no effect of CMA on attention in either male or female offspring.

Conclusion: CMA increased activity in juvenile male offspring but not in juvenile female offspring. CMA did not alter attention in either sex. These results, together with previous results in adult offspring demonstrate that adversity during both the pre- and post-natal period can lead to hyperactivity throughout life in male offspring however decreased attention does not emerge in female offspring until later in life.

ABSTRACT #P-E4

ALTERED mGLUR1/5 SIGNALING IN MICE LACKING THE GENERAL TRANSCRIPTION FACTOR GTF2IRD1

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Objective: The general transcription factor GTF2IRD1, along with its family member GTF2I, is commonly deleted in Williams-Beuren syndrome. While the function of GTF2IRD1 remains unclear, GTF2I has been shown to play a role outside the nucleus where it interferes with TRPC3 translocation to the plasma membrane (Caraveo et al., 2006). Since TRPC channels are thought to contribute to the excitatory effects of the group 1 metabotropic glutamate receptors (mGluR1/5), we sought to determine whether the excitatory effects of mGluR1/5 are altered in transgenic mice deleted for *Gtf2ird1*.

Materials and Methods: Since*Gtf2ird1* is expressed in layer V pyramidal cells of the medial prefrontal cortex (Proulx et al., 2010), we performed whole cell recordings in this population of neurons in adult *Gtf2ird1*$^{-/-}$ mice and their wildtype (WT) siblings. Peak currents in response to bath application of the mGluR1/5 agonist DHPG were measured in voltage-clamp. To appreciate the cellular mechanism(s) underlying these currents, we measured the current-voltage (I-V) relationship of the currents elicited by mGluR1/5 stimulation. Such currents could be a result of this Galphaq-coupled receptor (1) opening excitatory non-selective cation channels, (2) closing inhibitory leak potassium ion channels, or (3) having a combination of these channel-mediated effects.

Results: Cell properties did not vary between genotypes as assessed by resting membrane potential, input resistance, capacitance and spike amplitude. Bath application of mGluR1/5 agonist DHPG (30 μM, 15s) elicited prolonged excitatory inward currents in both groups. Examining the IV curve of the DHPG current in each neuron, we found that neurons from WT mice (n=9 cells) yielded an IV relationship with a negative slope and a reversal potential near -100 mV, while neurons from *Gtf2ird1*$^{-/-}$ mice yielded an IV relationship with a positive slope and an extrapolated reversal potential near -10 mV. These data suggest that mGluR1/5 signaling may function via different mechanisms in the WT and *Gtf2ird1*$^{-/-}$mice, potentially involving a greater potassium channel component in the WT neurons and a greater cation channel component in the *Gtf2ird1*$^{-/-}$ neurons.

Conclusions. We conclude that mGluR1/5 receptors may operate via different mechanisms WT and *Gtf2ird1*$^{-/-}$ mice to affect pyramidal cell excitability in the medial prefrontal cortex. Future work will further test the contribution of TRPC channels in both genotypes. In addition, we will examine mGluR1/5 signaling in transgenic mice that are deleted heterozygously for *Gtf2i* and *Gtf2ird1* together.

Funded by: This work was supported by th Scottish Rite Charitable Foundation (EKL), NSERC (EKL) and CIHR (EP).

ABSTRACT #P-E5

TRANSGENERATIONAL EFFECTS OF SYNTHETIC GLUCOCORTICOIDS ON THE EXPRESSION OF KEY REGULATORY GENES IN THE HIPPOCAMPUS

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Objective: Approximately 10% of pregnant women are at risk of preterm delivery. The majority of these women receive treatment with synthetic glucocorticoids (sGCs) to mature the fetal lungs, and therefore reduce the risk of infant respiratory distress syndrome. Prenatal exposure to sGCs has been associated with modification of hypothalamic-pituitary-adrenal (HPA) function in first (F1) and second (F2) generation offspring. In the present study, we hypothesized that the reduction in HPA (stress) responsiveness in F2 offspring results from increased central feedback inhibition through an elevation of glucocorticoid (GR) and mineralocorticoid (MR) receptor expression.

Methods: The left hemispheres of female and male offspring (F2) of mothers (F1) that had themselves been prenatally exposed to Betamethasone (BETA; 1 mg/kg), Dexamethasone (DEX ; 1 mg/kg) or saline (VEH) were cryosectioned at 10μm with focus on the hippocampus and the hypothalamic paraventricular nucleus (PVN). Hippocampal expression of GR, MR and synaptophysin, and hypothalamic expression of GR, corticotropin releasing hormone (CRH) and arginine vasopressin (AVP) mRNA were analyzed using *in situ* hybridization.

Results: Grandmaternal exposure to sGC resulted in a significant increase in GR mRNA expression in the CA1/2 region of the hippocampus in DEX females ($P<0.01$) and males ($P<0.001$), and in BETA males ($P<0.001$); sGCs also caused increased GR in the dentate gyrus in DEX ($P<0.01$) males. Female BETA F2 offspring exhibited a significant decrease of synaptophysin mRNA in hippocampal CA3 ($P<0.01$), and a trend towards decrease in the dentate gyrus. There were no significant differences in hypothalamic GR, CRH or AVP mRNA between treatment groups, or with MR in the hippocampus.

Conclusions: Adult male and female F2 offspring exhibit an overall reduction in HPA function as a result of grandmaternal antenatal sGC treatment. In these F2 animals, treatment with either BETA or DEX caused increased expression of hippocampal GR mRNA. We believe that this increase in GR mRNA is at least in part responsible for the reduced HPA activity observed in these animals (from behavioural and endocrine data), and that it does so by increasing hippocampally-mediated negative-feedback of the PVN. Grandmaternal antenatal sGC may also affect synaptic transmission, as evidenced by the decrease in hippocampal synaptophysin. We are currently undertaking additional studies to further investigate the mechanisms that underlie this transgenerational programming of HPA function following antenatal exposure to sGC.

Funded by: Canadian Institutes for Health Research

ABSTRACT #P-E6

ACUTE AROMATASE INHIBITION DECREASES BREAST PARENCHYMAL ENHANCEMENT OBSERVED ON MAGNETIC RESONANCE IMAGING IN POSTMENOPAUSAL WOMEN

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Objectives: To investigate the effect of acute estrogen deprivation on the breast background parenchymal enhancement in magnetic imaging resonance (MRI). The breast is highly hormonally sensitive especially to the sex steroid hormone estrogen. Both physiologic and iatrogenic steroid hormone modifications could affect how the breast tissue may appear with breast imaging techniques.

Methods: A prospective pilot phase II clinical trial. Baseline breast MRI was followed one month later by administration of aromatase inhibitor (letrozole 12.5 mg/day) for 3 days prior to a 2nd breast MRI. Background breast parenchymal enhancement was compared between the pre and post treatment studies for each patient.

Results: There was a significant reduction of background breast enhancement after treatment. No adverse effects were recorded by the subjects.

Conclusion: This study is a preliminary indication that aromatase inhibitors could improve the positive predictive value of the breast MRI by decreasing the overall nonspecific enhancement of the breast tissue.

Funded by: Canadian Foundation for Women's Health (CFWH) and CIHR Health Professional Fellowship.

ABSTRACT #P-F1

COST COMPARISON OF THE LAPAROSCOPIC BURCH COLPOSUSPENSION, LAPAROSCOPIC TWO-TEAM SLING AND THE TRANS-OBTURATOR TAPE PROCEDURE FOR THE TREATMENT OF STRESS URINARY INCONTINENCE

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Objective: To compare health care costs of three surgical procedures for the treatment of primary stress urinary incontinence: the laparoscopic Burch colposuspension procedure, laparoscopic 2-team slingand the trans-obturator tape (TOT) procedure.

Methods: This retrospective observational study includes patients who underwent an isolated surgical intervention (no concomitant surgery) for primary stress incontinence between December 2003 and December 2009. All procedures were performed by specialists of urogynecology at the Women's College Hospital site of Sunnybrook Health Science Centre. Six patients underwent a laparoscopic Burch colposuspension procedure, six underwent a laparoscopic 2-team sling, and six underwent a trans-obturator tape (TOT) procedure. A detailed review of the patients' medical records was conducted to establish the direct costs related to their surgery. Itemized calculations were made for: (a) equipment costs;(b) surgeon, surgical assistant andanaesthesiologist reimbursements;(c) nursing costs; (d) operating and recovery room costs;and (e) length of stay in hospital. Costs were estimated based on 2010 dollars. The main outcome measure was the mean aggregated cost per patient treated.

Results: The mean cost per patient undergoing a TOT procedure was $2,547 (95% CI: $2,260 - $2,833), a laparoscopic Burch colposuspension procedure was $4,354 (95% CI: $3,465 - $5,244), and the mean cost per patient undergoing a laparoscopic 2-team sling was $5393 (95% CI: $4959 - $5826). Significant differences were found across procedures using a one-way ANOVA. A TOT was lower in cost than both a Burch ($p=0.0008$) with a mean cost difference of $1807.88, and sling ($p<0.0001$) with a mean cost difference of $2834.73.

Conclusion: The preliminary results show that a trans-obturator tape procedure is less costly than a laparoscopic Burch colposuspension or a laparoscopic two-team sling in the surgical treatment of stress urinary incontinence.

ABSTRACT #P-F2

USE OF THE INTRAUTERINE SYSTEM FOR MENSTRUAL SUPPRESSION IN THE DEVELOPMENTALLY DELAYED ADOLESCENT

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ABSTRACT AVAILABLE IN HARDCOPY VERSION ONLY

ABSTRACT #P-F3

A HIERARCHICAL ANALYSIS OF TRIAL OF LABOUR IN ONTARIO: DO WOMEN, DOCTORS, OR HOSPITALS CHOOSE?

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Objective: In Ontario in 2006, the caesarean birth rate was 28%, the main contributor being the repeat caesarean rate. Canadian guidelines recommend that women with one previous caesarean be offered a Trial of Labour (TOL) providing there are no contraindications. The objectives of this study were (1) to determine the TOL rate (proportion of women with previous CS who had TOL), and the successful TOL rate (proportion of women who had TOL who had a vaginal birth), and (2) to determine the association between provider and hospital factors and rates of TOL and successful TOL.

Methods: This is a population-based retrospective cohort study of women who gave birth in Ontario in 2007, with history of previous CS, eligible for TOL based on clinical characteristics. Administrative databases were directly linked in order to determine characteristics of the women (age, income, gestational age at delivery, history of previous vaginal birth), their physician providers (gender, years of experience, specialty, country of medical education), and the hospitals in which they gave birth (annual obstetric volume, NICU level, teaching status).
For the data analysis, two-level generalized hierarchical linear models were developed to adjust for maternal characteristics, and to take into account the hierarchical nature of the data (women clustered into providers, and women clustered into hospitals).

Results: The study population comprised 12,170 women. The TOL rate was 23% and the successful TOL rate was 75%. Women whose prenatal care provider was a family doctor or a female doctor were more likely to have a TOL. Characteristics of the intrapartum care provider were not significantly associated with successful TOL. Women who gave birth in teaching hospitals were more likely to have a TOL and a successful TOL.

Conclusions: Multi-faceted interventions aimed at patients, prenatal care providers and hospitals would be necessary to impact the low TOL rate in this province.

Funded by: The POWER study, **P**roject for an **O**ntario **W**omen's Health **E**vidence-based **R**eport

ABSTRACT #P-F4

CHARACTERISTICS OF WOMEN REQUIRING HOSPITAL ADMISSION FOR TREATMENT OF PELVIC INFLAMMATORY DISEASE (PID) AT AN INNER CITY HOSPITAL

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Objective: To determine patient characteristics of women requiring hospital admission for treatment of pelvic inflammatory disease (PID) at a tertiary care inner city hospital.

Methods: A retrospective chart review was performed on all women seen in our Emergency Room (ER) from 2000-2006 with a diagnosis of PID, tubo-ovarian abscess (TOA), salpingitis, or pelvic infection. Data were collected regarding patient characteristics and management strategies.

Results: During the study period, 137 women were seen, with a mean age of 30 years and 78% having never been married. Most women (74%) had no identified risk factors for PID. Among women with ER visits, 35% were admitted to the hospital. The mean length of stay was 3 days, with 80% admitted for 4 days or fewer. Of women admitted, 20% had TOA, 76% were treated with medical management, and 24% had surgery. The most common presenting symptoms were pelvic/abdominal pain (100%), cervical motion tenderness (96%), vaginal discharge (82%), elevated white blood cell count (75%), and fever (57%). Bacterial vaginosis was the most commonly identified (28%) documented infection, with chlamydia (15%) and gonorrhea (7%) found less commonly. Higher patient age was a significant predictor of being admitted to hospital ($p<.001$), being diagnosed with TOA ($p=.0012$), and requiring surgical management ($p=.0002$). Marital status was also predictive, with married women significantly more likely to have TOA (OR=2.92, $p=.0219$) and to require surgery (OR=3.68, $p=.0109$). Using logistic regression, increasing patient age remained statistically significantly associated with hospital admission, having a TOA, and requiring surgery.

Conclusions: Approximately two thirds of women seen in the ER of an inner city hospital with PID were managed as outpatients. Among women admitted to the hospital, most were treated with medical therapy and had short admissions. Married and/or older women were significantly more likely to require hospital admission and have TOA.

ABSTRACT #P-F5

CONSISTENCY OF ANTIBIOTIC REGIMENS FOR SUSPECTEDCHORIOAMNIONITIS IN A DOWNTOWN TORONTO TEACHING HOSPITAL

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Objective: To determine the number and types of antibiotic regimens used in a downtown Toronto teaching hospital for cases of suspected chorioamnionitis.

Methods: A retrospective chart review of 617 deliveries during a 3-month period (March – May 2007) at St. Michael's Hospital was performed to identify patients with suspected chorioamnionitis. Chorioamnionitis was defined as intrapartum fever > 38.0^{o}C and one or more fo the following: maternal tachycardia, fetal tachycardia, uterine tenderness, foul smelling amniotic fluid, and maternal leukocytosis. A descriptive analysis of the number and types of antibiotic regimens was carried out.

Results: The incidence of chorioamnionitis during the study period was 8.1% (50/617). Nine different antibiotic regimens were used in the intrapartum period and ten different regimens were used in the postpartum period, with a variation in drug dose, frequency, and duration of use involving either clindamycin, gentamycin, and/or ampicillin.

Conclusions: There is no clear consensus regarding the treatment of suspected chorioamnionitis within a single downtown Toronto teaching hospital. This marked variation in the choice of antibiotic regimens parallels what is observed in the literature, and is likely seen at other institutions as well. Future research should be directed at duplicating this study in other centres, and at the development of universally accepted protocols for antibiotic regimens for chorioamnionitis in order to help guide resident teaching and provide consistent care.

ABSTRACT #P-G1

THE ROLE OF ANDROGEN SUPPLEMENTATION IN OVULATION INDUCTION IN OLDER WOMEN

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Objectives: The aim of the study was to determine the efficacy of DHEA in a subset of women 40 years and older with diminished ovarian reserve who were treated with controlled ovarian hyperstimulation and intrauterine insemination (COH-IUI).

Methods: A retrospective cohort study was conducted in a private, university affiliated fertility center. Between February 2006 and September 2009, 214 patients 40 years and older were treated with 25 mg of pharmaceutical grade micronized DHEA 3 times daily for at least 3 months. Diminished ovarian reserve was evidenced by basal FSH level, AMH level and antral follicle counts. Of these 214 patients, 79 were lost to follow up, 2 stopped treatment due to side effects (acne; hair loss), and 133 continued with treatment. Of these 133, 29 patients who did not get pregnant on DHEA alone or both DHEA and aromatase inhibitors were treated with COH-IUI. This consisted of an age appropriate dose of HMG starting on cycle day 3, midcycle HCG (10,000 TO 20,000 IU), 2 consecutive inseminations and luteal phase progesterone support.

Results: In the 29 patients treated with DHEA and COH-IUI, 14 patients conceived (48%), pregnancy rate per cycle 18.6%.Excluding the 2 patients with the most cycles without achieving a pregnancy (7 and 11 cycles), the pregnancy rate per cycle is 24.4%. The average patient age was 41.7 years. The average time to conception was 2.6 cycles. The outcome in these 14 patients included 8 early losses (57%), and 6 singleton pregnancies, that resulted in 4 full term deliveries and 2 pregnancies still ongoing.

Conclusion: Casson et all (2000) reported a beneficial effect of exogenous administered Androgens in a small group of patients in improving fertility in older women. Since 2005 Gleicher et all have been reporting similar results in older women doing IVF. Our center has been using DHEA since February 2006 and now has over 140 total pregnancies from DHEA alone or DHEA combine with aromatase inhibitors, COH with IUI and IVF. Pretreatment with DHEA followed by COH-IUI in our study resulted in an excellent pregnancy rate in an otherwise poor prognosis group of patients. This study failed to demonstrate the reduction in pregnancy loss with DHEA that we had previously reported in all age groups. However, we propose that COH-IUI remains an efficient and affordable therapeutic modality for older women who have taken DHEA for at least 3 months.

ABSTRACT #P-G2

EFFECT OF VERY HIGH SERUM ESTRADIOL LEVELS ON IMPLANTATION IN HIGH-RESPONDERS UNDERGOING IN VITRO FERTILIZATION (IVF)

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Objective: To evaluate the effect of high E2 levels on the day of hCG on implantation in high responders undergoing IVF.

Methods: Retrospective Cohort. 94 women (47 <35y and 47 ≥35y; FSH<10 mIU/mL and AMH>15 pmol/L) were studied. 21 patients <35y had peak E2 levels <10000 pmol/L (2730 pg/mL) (Group 1) and 26 patients <35y had peak E2 levels >10000 pmol/L (Group 2). 21 patients ≥35y had peak E2 levels <10000 pmol/L (Group 3) and 26 patients ≥35y had peak E2 levels >10000 pmol/L (Group 4). Patients underwent a GnRH-a downregulation or GnRH-ant protocol with rFSH/LH (150-450 IU/day) for 10-12 days. Once the majority of follicles were 18 mm, hCG (5-10,000 U) was given followed by oocyte retrieval at 35h and day 3 or 5 ET. Primary outcomes studied were peak E2 levels, #oocytes retrieved, #embryos transferred, implantation, ongoing pregnancy, and OHSS.

Results: The mean age in the <35 and ≥35 groups was 29.7y and 36.6y. The total gonadotropins used was 3907(sd=1947.5) IU in the <35y group and 4422 (sd=1975.1) IU in the ≥35y group. Peak E2 levels were 6302.3 (sd=2625.7) pmol/L (1982.2 ±825.7 pg/mL) in Group 1, 16752.0 (sd=6274.9) pmol/L (5267.9 ±1973.2 pg/mL) in Group 2, 6270.5 (sd=2599.8) pmol/L (1971.9 ±817.5 pg/mL) in Group 3, and 16709.5 (sd=4589.3) pmol/L (5254.6 ±1210.1 pg/mL) in Group 4. The #oocytes retrieved in the <35y group was 17 (sd=8.20) *vs.* 16 (sd=8.29) in the ≥35y group. The #embryos transferred was 4.96(sd=2.95) in the <35y group *vs.* 6.27(sd=3.72*)* in the ≥35y group. The implantation rate (IR: #pts with positive b-hCG 14d after ET/total # pts) in Group 1 was 12/21, or 57% and 5/26, or 19% in Group 2. The IR in Group 3 was 9/21, or 43%, and 9/26, or 35% in Group 4. Ongoing pregnancy rates were 9/21 (43%), 4/26 (15%), 8/21 (38%), and 8/26 (31%) in Groups 1-4, respectively. No OHSS was reported.

Conclusions: High peak E2 levels during IVF may adversely affect implantation, especially in women <35y. This may be due to overmaturation of the endometrium. Lower gonadotropin dosing in high responders may yield a more receptive endometrium during IVF.

Funded by: N/A

ABSTRACT #P-G3

FUNCTIONAL ANALYSIS OF NALP5 AND ITS POSSIBLE INTERPLAY WITH SPINDLE ASSEMBLY CHECKPOINT PROTEINS DURING OOGENESIS

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Objective: Nalp5, a Maternal Lethal Effect gene, is essential for proper progression of development beyond the two-cell stage of embryogenesis. Thus far, only three interacting partners of NALP5 have been identified. One of which, FILIA, has recently been found to maintain euploidy during preimplantation embryo development, more specifically at the two-cell stage of development (Zheng & Dean, 2009). We propose that Nalp5 may facilitate activation of the Spindle Assembly Checkpoint (SAC) and may play a role in preventing aneuploidy, possibly via interaction with Filia.

Methods: Wildtype & Nalp5 knockout female mice were super-ovulated with gonadotropins and mature (metaphase II stage) oocytes were collected from oviducts. To see whether protein localization and protein levels differed in wildtype and Nalp5 knockouts, immunocytochemistry was performed using anti-α-Tubulin, anti-Nalp5, anti-Bub1, anti-Pericentrin, anti-Aurora A, anti-p73, anti-BRCA1, and anti-Dynactin p50 antibodies and DAPI. Oocytes were imaged using a deconvolution microscope and levels of fluorescence were quantified using DeltaVision software.

Results: We observed a greater frequency of pronuclear number abnormalities in Nalp5 knockout zygotes (35%) than in wildtype zygotes (8%) and found that the extra pronuclei were of maternal origin, indicating that this defect was likely maternally-derived. Subsequent analysis of ovulated oocytes revealed an increased frequency of spindle morphology abnormalities were present in Nalp5-deficient oocytes (38%) compared to wildtype oocytes (3%). In Nalp5 knockout oocytes, spindles showed decreased levels of α-Tubulin staining. Additionally, oocytes from Nalp5 knockouts displayed a greater frequency of misaligned chromosomes at metaphase II (54%) than in wildtypes (3%). Moreover, when looking at the SAC-associated protein kinase, BUB1, a lower level of protein expression was observed in Nalp5-deficient oocytes relative to wildtype oocytes. The additional proteins are under analysis.

Conclusions: An increased number of Nalp5 knockout zygotes displayed abnormalities in pronuclear number, which is caused by improper segregation of parental DNA and is likely attributable to abnormal spindle structure. Our data suggests that Nalp5 plays a significant role in maintaining euploidy during oogenesis. Furthermore, Nalp5 may be upstream of Filia since spindle defects are observed at the oocyte stage of development in Nalp5-deficient females.

Funded by: Canadian Institutes of Health Research

ABSTRACT #P-G4

EXPRESSION OF SURVIVIN IN HUMAN OOCYTES AND EMBRYOS

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Objective: Survivin is a structurally unique member of the inhibitor of apoptosis protein family. It is a multifunctional molecule which acts as a suppressor of apoptosis and plays a crucial role in cell division. High levels of survivin expression have been detected in cancer cells and human fetal cells but no studies have been done on human embryos. The purpose of our study was to determine if: 1) survivin is expressed in human oocytes and early preimplantation embryos; 2) embryos secret survivin protein into culture medium; and 3) survivin expression is correlated with embryo quality and embryo cleavage rate.

Methods: Survivin mRNA and protein expression were investigated in human/mouse oocytes and embryos using nested RT-PCR and immunocytochemistry techniques, respectively. Materials used in the study consisted of oocytes and embryos that were not suitable for uterine transfer or cryopreversation. The concentration of survivin protein in culture conditioned media from Day 3-5 human embryos was measured using a commercial survivin ELISA assay. Similar experiments were also performed on mouse oocytes and embryos to compare expression of survivin between human and mouse species. Ethics (REB) approval was obtained for this study.

Results: Survivin mRNA was detected in human oocytes, zygotes, cleaved and blastocyst stage embryos. The main survivin transcript (339bp), two variants survivin-2B(414bp) and survivin-ΔE3(229bp) were detected in all human oocytes, zygotes and blastocysts studied. Interestingly, 2-8 cell embryos differed in that they had no detectable survivin-2B. Survivin protein was localized within cytoplasm of all human oocytes and embryos studied, located predominantly in the cell cortex. In human blastocysts stage, strong survivin protein staining was detected in both the inner cell mass and trophectoderm. Similar results were seen in the mouse oocytes and embryos. Culture media samples from 147 patients undergoing IVF procedures were tested for this protein. A positive correlation was found between embryo cleavage rate and survivin secretion by human embryos.

Conclusions: This study has demonstrated for the first time that survivin mRNA and protein are expressed in all developmental stages of human oocytes and embryos. Survivin protein is also secreted into culture media by human and mouse embryos. A positive correlation was found between embryo cleavage rate and survivin secretion by human embryos. Survivin is a possible marker of embryo quality that potentially could be utilized for embryo selection during IVF.

Funded by: Unrestricted research grant.

ABSTRACT #P-G5

NON-INVASIVE GENOMIC ANALYSIS OF HUMAN ENDOMETRIAL RECEPTIVITY

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Objective: Impaired endometrial receptivity is a major barrier to fertility and contributes to poor Assisted Reproductive Technology (ART) outcomes. The endometrium is receptive to the embryo only during a temporally-restricted window of receptivity, during the mid-luteal phase. The molecular mechanisms underlying this process are still poorly understood and there are no robust clinical tests for endometrial receptivity. The objectives of this study are to develop a minimally-invasive technique to sample the endometrium and to identify genes involved in endometrial receptivity by examining the endometrial gene expression profile during the early luteal (pre-receptive) phase compared to the mid-luteal (receptive) phase.

Methods: Institutional ethics approval has been obtained from Mt. Sinai Hospital. Women with regular menstrual cycles, normal hormonal profiles, and no history of female factor infertility will be recruited. Endometrial cells will be obtained from each subject by uterine aspiration with an intrauterine insemination (IUI) catheter during the early luteal phase (2 days post-LH surge) and the mid-luteal phase (7 days post-LH surge). RNA will be extracted, reverse transcribed to cDNA, amplified and hybridized to human genome microarrays. Data will be subjected to extensive statistical analysis to identify differentially expressed genes. Differential expression of selected genes will be verified by RT-qPCR or nanostring arrays and immunohistochemistry. As uterine aspiration is a novel technique, we will also obtain endometrial biopsies only during the mid-luteal phase in the same subjects in order to compare microarray results from the few cells obtained by uterine aspiration to those obtained by biopsy.

Results: This is a work-in-progress project. We are currently in the enrollment phase. Preliminary work has been done on developing uterine aspiration as a technique for sampling the endometrium. We have shown that uterine aspirations contain a sufficient quantity and proportion of endometrial cells for microarray analysis. Our aim is to enroll 25 women in one year's time. This pilot study may form the basis for future studies on candidate genes identified as potential markers of endometrial receptivity and may direct research towards development of assays of proteins present in uterine aspirates that could assess endometrial receptivity.

Conclusions: Pending.

ABSTRACT #P-G6

DIFFERENTIAL EXPRESSION OF MAESTRO (MRO) GENE SPLICE VARIANTS IN CULTURED GRANULOSA AND CUMULUS CELLS IN RESPONSE TO HORMONAL STIMULATION

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Objective: We previously showed that MAESTRO is over expressed in granulosa cells from lean patients with Polycystic Ovarian Syndrome (PCOS). Our group also described the presence of unique splice variants in granulosa cells from PCOS as compared to non-PCOS patients. These differences disappear when PCOS patients are treated with Metformin. The purpose of our study was to determine the influence of testosterone, estrogen and hCG on the level and ratio of the different MRO gene isoforms in vitro.

Methods: After obtaining informed consent, granulosa and cumulus cells were collected from the follicular fluid of five patients undergoing IVF procedures. Two samples were included from PCOS patients, who were taking Metformin, and three from patients with male factor infertility. Cells were first cultured for 48h in DMEM/F12 media followed by incubation in medium supplemented with either: testosterone, estrogen or hCG. The cells were further incubated for 20min, 3h, 6h, 12h and 24h in hormone supplemented medium and collected for RNA extraction. Total RNA of 200ng was reverse-transcribed to cDNA and used for PCR. Various MRO primers and different culture conditions were analyzed and compared. These experiments were reviewed and approved by the Sunnybrook Health Sciences Centre REB.

Results: In granulosa cells differential expression was observed at different time points between the control group (no hormone added) and the testosterone and hCG, but not with estrogen. In cumulus cells a differential expression pattern was detected with testosterone but not with estrogen or hCG. As expected, there was no difference in results between PCOS patients taking Metformin and male factor infertility cases either with or without exposure to hormones.

Conclusions: Hormones such as testosterone, estrogen and hCG have distinct influences on different types of cells. We found differential splice variant expression between cultured granulosa and cumulus cells. We also observed differential regulation by testosterone and/or hCG, but not with estrogen,.in these cell types.

Funded by: Unrestricted research grant from Ferring Canada Inc.

ABSTRACT #P-H1

THE EFFECTS OF SELECTIVE SEROTONIN REUPTAKE INHIBITORS (SSRIs) ON PLACENTAL P-GLYCOPROTEIN

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Objective: P-glycoprotein (P-gp) is a member of the ATP-binding cassette (ABC) superfamily. P-gp, located in the cell membrane, extrudes a wide variety of compounds, including cardiac glycosides, antiviral and anticancer drugs. It is expressed at high levels in the placental syncytiotrophoblast, and prevents xenobiotics present in the maternal circulation from entering the fetus. In cancer cells, P-gp is potently inhibited by the selective serotonin reuptake inhibitors (SSRIs), resulting in increased cellular accumulation of P-gp substrates. We have previously shown that SSRIs, particularly sertraline, can have profound influences on P-gp mediated drug transport in endothelial cells in the brain microvasculature. In the present study, we hypothesized that sertraline would decrease placental P-gp activity and increase drug transfer from the mother to the fetus.

Methods: At embryonic day 15.5, pregnant FVB mice were injected simultaneously with: 1) sertraline (10 mg/kg) and [^{3}H]digoxin (1 μCi/30 g), or 2) vehicle and [^{3}H]digoxin (1 μCi/30 g). Digoxin is a stable P-gp substrate and serves as an effective marker of P-gp activity. Animals were either killed 5 min or 240 minutes after the injections in order to determine the time course of effect. After euthanasia, litters were split; half of the fetuses were left intact with fetal membranes and amniotic fluid (to assess total transplacental transfer), and half were dissected to allow determination of drug transfer into the fetal brain. Maternal blood and maternal brains were also collected. Drug ratios in the fetal unit and the fetus were then determined.

Results: In animals that were euthanized 240 minutes after injection, there was a significant decrease in fetal digoxin accumulation in the sertraline-treated group ($P < 0.05$). There was no difference in drug transfer between the sertraline- and vehicle-treated groups at the 5 minute time-point. There was also no significant effect of sertraline on brain accumulation of digoxin in fetuses or mothers at either the 5- or 240-minute time-points.

Conclusions: The results indicate that sertraline has no effect on placental drug transfer at 5 minutes, but increases P-gp activity at 240 minutes, resulting in decreased drug transfer to the fetus. This is the opposite of what we had hypothesized. We are currently investigating additional time courses to determine how the effects of sertraline on placental P-gp change as a function of time. The fact that placental P-gp can be effectively modulated by SSRIs and that this modulation is different from effects in other tissues, indicates that it may be possible to develop treatment strategies that maximize the effects of therapeutic drugs in the mother but protect the fetus.

Funded by: Canadian Institutes for Health Research

ABSTRACT #P-H2

DIFFERENTIAL PLACENTAL PATTERNING BY FETAL SEX IN HIGH-RISK PREGNANCIES WITH PLACENTAL DYSFUNCTION

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Objective: To test the hypothesis that a differential pattern of chorion regression (concentric/ symmetrical vs. random/asymmetrical) exists between male and female placentas from high-risk pregnant women following delivery due to placental complications of pregnancy.

Methods: Retrospective study of deliveries at MSH from 2000-10. Singleton pregnancies were identified from our database delivering between 22^{+0} and 32^{+6} weeks' gestation with one or more of the following inclusion criteria: small-for-gestational age (SGA) infant (birth weight < 10^{th} centile), intrauterine growth restriction (IUGR; absent or reversed umbilical artery flow by Doppler), major placental abruption, intrauterine fetal death (IUFD), or severe pre-eclampsia (as per ACOG definition). All cases had placental pathology examination at MSH following delivery. Maternal demographics were recorded and measures of placental patterning [placental weight and weight centile, maximum length/width ratio, site of umbilical cord insertion and number of umbilical cord vessels] were compared between male and female fetuses to determine the incidence of random vs. concentric chorionic regression.

Results: From the first 124 pregnancies with one or more inclusion criteria; the results are presented in Table 1. Eighty-five women (68.5%) were delivered by C/Section illustrating disease severity. A 4-5-fold excess of SGA placentas was observed in each sex, but this proportion did not differ between the sexes. Male placentas were significantly more likely to show asymmetric chorion regression compared to females.

	Males (n=71; 57.3%)	**Females** (n=53; 42.7%)	**Sign**
Placental weight <10th centile	29 (40.8%)	26 (49.1%)	NS
Cord insertion <2cm from edge	34 (47.9%)	19 (35.8)	NS
Cord insertion:			
Central	37 (52.1%)	34 (64.2%)	NS
Marginal	26 (36.6%)	19 (35.8%)	NS
Velamentous	8 (11.3%)	0 (0.0%)*	*p=0.034
Mean length:width ratio (±SE)	1.25 ± 0.026	1.21 ± 0.017	NS
2 vessel cord	3 (4.2%)	5 (9.4%)	NS

Conclusions: Male pregnancies exhibit an asymmetrical pattern of placental insufficiency (asymmetric chorion regression) compared to females, suggesting that different underlying molecular mechanisms operate in these diseases between the sexes.

ABSTRACT #P-H3

ANTENATAL DEXAMETHASONE TREATMENT DOWN-REGULATES SYSTEM A TRANSPORT AT TERM IN THE MURINE PLACENTA.

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Objective: Synthetic glucocorticoids (sGCs) are administered to women with threatened preterm labour (PTL) to enhance fetal lung maturation. Repeat courses of sGCs have shown to reduce infant birth weight, although this is no longer a recommended clinical practice. A possible mechanism for birthweight reduction is through alteration of placental nutrient transport, a critical determinant of fetal growth. Paradoxically, the neutral amino acid system A transporter in term placental explants is stimulated by 48 hour *in vitro* treatment with dexamethasone (DEX) but there are no comparable *in vivo* data. DEX also regulates long term changes of gene expression in the murine placenta and these may alter nutrient transport. Since 70% of pregnant women who receive sGCs for threatened PTL do not deliver within 7 days, and almost 30% carry to term, our objective was to examine the short and long term effects of a single course of sGC treatment at mid-gestation on placental system A transport using a mouse model. We hypothesized that DEX treatment would lead to an acute (after 24 hours) stimulation of placental system A activity, but that with time this effect would reverse leading to reduced placental system A transport in late gestation.

Methods: Pregnant C57BL/6 mice were treated with either vehicle (saline) or DEX (0.1mg/kg) on days E13.5 and E14.5. Twenty-four hours later at E15.5 and prior to term (E18.5) fetal and placental weights were recorded and the transplacental transfer of ^{14}C-methylaminoisobuytyric acid (specific system A substrate) was measured in fetal-compartments (fetus, amniotic sac, yolk-sac and amniotic fluid) (E15.5-VEH n=5, DEX n=7; E18.5 VEH n=7, DEX n=7). A ratio of radioactivity present in the fetal-compartment relative to maternal plasma was calculated per weight. To verify specificity, the transfer of ^{14}C-mannitol (passive permeability marker) was measured. Fetal sex was determined.

Results: System A activity increased from E15.5 to E18.5 in placentae from male and female fetuses ($p<0.01$). No significant differences in system A activity, fetal weight or placental weight due to DEX treatment occurred at E15.5. At E18.5 placenta from fetuses of either sex had significantly reduced system A transport due to DEX treatment ($p<0.05$). DEX treatment did not cause any significant differences in passive permeability of the placenta. At E18.5 DEX treatment significantly reduced total weight in compartments containing female fetuses ($p<0.05$) and placental weight of female fetuses compared to vehicle treatment. Placentae of female fetuses were significantly smaller than those of male fetuses ($p<0.05$), irrespective of treatment.

Conclusion: sGC administration does not appear to have short term effects on system A transport *in vivo*. However, if gestation persists to term, sGC treatment leads to a substantial reduction in system A mediated transport in placentae of both male and female fetuses. Furthermore, female foetuses appear more susceptible to reduced placental growth and fetal growth when treated with a single course of sGCs.

ABSTRACT #P-H4

DECIDUAL LEUKOCYTE POPULATIONS EXHIBIT DRAMATIC CHANGES ACROSS GESTATION IN HEALTHY HUMAN PREGNANCY.

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Objective: The unique leukocytes that populate the uterine lining have proposed roles in regulating trophoblast invasion, angiogenesis, decidual spiral artery remodeling, and immune tolerance. The primary aim of this study was to characterize the maternal leukocyte populations (T cells, macrophages, uNK cells, dendritic cells & neutrophils) in the decidua across gestation by detailed immunophenotyping. A further aim is to identify changes in decidual leukocyte subtypes of patients with IUGR or preeclampsia that may contribute to the impaired vascular remodeling associated with these pathologies.

Methods: First (n=9), second (n=8), and third (n=5) trimester decidual tissues were collected from patients of the Morgentaler clinic, the Mount Sinai Hospital ST Interruption of Pregnancy Clinic, or the Mount Sinai Hospital Special Pregnancy Program following informed consent. Leukocytes were derived from decidua by mechanical isolation, passed through a series of microfilters and incubated with erythrocyte lysis buffer before being labeled with fluorescence-conjugated antibodies (anti- CD45, CD3, CD4, CD8, CD11b, CD14, CD15, CD25, CD56, CD83, CD163, CD205, CD206, CD209 & HLA-DR,) for analysis by flow cytometry.

Results: Flow cytometric analysis identified several changes in leukocyte profiles between trimesters. Of particular interest were the decline in proportion of both uterine Natural Killer cells and decidual macrophages after the first trimester and the large accumulation of neutrophils and T helper cells in the second trimester. There is a decline in immature dendritic cells and markers of both antigen uptake and presentation with advancing gestation. As well, the consistently low levels of both cytotoxic T lymphocytes and mature dendritic cells suggest a suppressed maternal immune environment at this time which may be altered in pathologies with a presumed immunological component, such as preeclampsia. Preliminary data collected from patients with utero-placental vascular insufficiency indicate that an increase in NK cells and NKT cells may be associated with IUGR.

Conclusions: These data suggest a differential role for immune cells between the first, second and third trimesters and provide evidence of unique and complex immune interactions in the decidual microenvironment during healthy human pregnancy. In particular, neutrophils, uterine NK cells and macrophages may mediate tissue and artery remodeling since they are abundant when this process is occurring. The immature dendritic cells may stimulate induction of maternal tolerance of the fetally-derived cells. Comparisons of these data with leukocyte profiles from pathologies will help to identify abnormal leukocyte presentation or activation in these cases.

Funded by: The Canadian Institute for Health Research IHD-86232, a Bernard Ludwig OSOTF Fellowship, an Ontario Graduate Scholarship Master's Award, and a CIHR Master's Award.

ABSTRACT #P-H5

EXPANSION OF THE PLACENTAL VASCULATURE IN LATE GESTATION IS STRAIN DEPENDENT IN MICE

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Objective: In the mouse rapid capillarization occurs in late gestation to meet the demands of the growing fetus. Whether such changes are associated with elaboration of the larger vessels of the fetoplacental tree and the uteroplacental vasculature is unknown. As various mouse strains are used to study placental insufficiencies, inter-strain differences in the vascular growth that occurs in late gestation need to be assessed. This study aimed to quantify vascular growth in C57Bl/6J (B6) mice, a common background strain for transgenic mouse models, and in CD-1 mice which are strong breeders and the most common strain used in pregnancy research.

Methods: Fetoplacental arterial trees from B6 and CD-1 mice were infused with X-ray contrast agent at embryonic day (E)15.5 and 17.5 (N = 8 to 13 /group), uteroplacental arterial trees were perfused at E17.5 (N = 5 to 7 /group), and 3-D micro-CT images were obtained. Volume measurements of uteroplacental spiral arteries and maternal canals were obtained by segmenting 3D images using the Amira visualization program, whereas novel, purpose-designed image processing software was used to determine length, diameter, and connectivity of each vessel greater than 50 μm in diameter in the fetoplacental trees. Fetoplacental arterial resistance was calculated (N = 3-9 /group) using blood flow modeling software written in house.

Results: At E15.5, B6 fetal weight was 13% smaller than that of CD-1 ($p< 0.05$) but placental weights were similar (~0.15 g). Nevertheless, umbilical artery diameter was larger (0.52 ± 0.01 mm vs. 0.47 ± 0.01 mm, $p < 0.01$) and there were 40% more vessel segments of large diameter (>100 μm, $p < 0.05$) in the fetoplacental tree of B6 placentas. From E15.5 to 17.5 the number of these large diameter vessels increased by 80% in the B6 tree, while the number of segments of arteriolar size (ie. 50-100 μm in diameter) stayed constant. The vascular depth of this tree increased by 30% ($p < 0.01$), suggesting an increase in labyrinthine thickness in late gestation in B6. These changes led to a 33% decrease in B6 arterial resistance from E15.5 to 17.5. The increase in CD-1 fetal weight (+126%) was greater than for B6 (84%) augmenting the fetal weight discrepancy at E17.5. Additionally, CD-1 placental weight at E17.5 was significantly higher than B6, yet the number of fetoplacental vessels did not increase significantly in CD-1 placentas in either size range, nor were there changes in vascular depth and arterial vascular resistance. The volume of uteroplacental spiral arteries (1.3 ± 0.2 mm^3 vs. 0.7 ± 0.1 mm^3, $p < 0.02$) and maternal canals (1.0 ± 0.1 mm^3 vs. 0.5 ± 0.1 mm^3) at E17.5 was double in CD-1 mice as compared to B6.

Conclusions: Quantification of the placental vasculature using micro-CT image analysis revealed significant strain-dependent differences in late gestational expansion of the fetoplacental vascular tree with prominent expansion of larger diameter vessels and a decrease in vascular resistance in B6 placentas only. CD-1 placentas demonstrated much greater uteroplacental vasculature, which may reduce their need for fetoplacental expansion in late gestation

Funded by: Heart and Stroke Foundation of Ontario

ABSTRACT #P-H6

THE ROLE OF VHL IN REGULATING THE EXPRESSION OF CCND1 IN PHYSIOLOGICAL AND PATHOLOGICAL PLACENTAL CONDITIONS

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Objective: We have previously demonstrated that Von Hippel Lindau (VHL) tumour suppressor protein plays a critical role in placental development as a master regulator of hypoxia inducible factor (HIF)-1α. Apart from this canonical function, emerging evidence has underscored a novel role for VHL in cell cycle exit through an inhibitory effect on cyclin D1 (CCND1). Classically, D-type cyclins associate with cyclin dependent kinases (Cdks) and promote cell cycle progression. Cell cycle and CCND1 activity/stability is inhibited by the INK4 and Cip/Kip family of proteins including p15 and p27 respectively. As events guiding trophoblast proliferation and differentiation are important to proper placental development and are impaired in placental pathologies such as preeclampsia, the aim of this study was to determine the relative contribution of VHL to trophoblast cell cycle regulation in normal and pathological conditions.

Methods: Placental tissue were collected from first trimester placentae (5-8wks; n=8, 10-13wks; n=11), preeclamptic placentae (PE; n=25) and age-mated control (AMC; n=21). VHL and CCND1 mRNA was assessed by qRT-PCR analysis and VHL, CCND1, p27 , p15, HIF-2α protein expression was assessed by immunoblot analysis. To study the direct role of VHL on CCND1 expression siRNA strategy was used to knock-down VHL in choriocarcinoma JEG-3 cells using a liposome-based reagent. To assess the proliferative status of JEG-3 cells following VHL silencing we performed BrdU incorporation assays and immunoblotted for proliferating nuclear antigen (PCNA). To assess spatial localization of VHL, CCND1 and p15 we performed dual labeled immunofluorescent staining of first trimester, PE and AMC placental sections.

Results: In this study we report that VHL and CCND1 exhibit an inverse pattern on expression in first trimester placentae, whereby high VHL expression at 6-9 weeks of gestation is associated with a decrease in CCND1 levels. This is further corroborated by our immunofluorescence data which demonstrates that in the trophoblastic layer, cells expressing high levels of VHL, express minimal amounts of CCND1. Moreover, VHL silencing in JEG-3 cells resulted in an approximately 2-fold increase in CCND1 transcript and 1.4-fold increase in CCND1 protein. Notably this was associated with a 1.5 fold increase in HIF-2α, a transcriptional activator of CCND1 which similarly to HIF-1α is also regulated by VHL. Moreover, while we observed no differences in p27 expression following VHL knockdown, we report a novel role for VHL in regulating the expression of p15. Lastly, we observed decreased VHL mRNA and protein expression in PE relative to AMC and this was associated with altered expression of cell cycle regulators CCND1 and p15.

Conclusions: Our findings suggest a novel role for VHL in cell cycle regulation during normal placental development. In particular, we provide evidence that VHL negatively regulates the expression of CCND1 via its action on HIF-2α and p15 and that this regulation is altered in PE.

Funded by CIHR/IGH/Margaret J. Santalo, University of Toronto Scholarship

ABSTRACT #P-I1

THE IMPACT OF ETHNICITY ON AWARENESS, KNOWLEDGE, AND ATTITUDES OF THE HPV VACCINE IN ADULT WOMEN

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Objective: Widespread HPV vaccination has the potential to decrease discrepancies in cancer risk based on ethnicity and socioeconomic status. The objective of the current study was to determine whether ethnicity affects HPV vaccine awareness, knowledge, and attitudes.

Methods: English-speaking women (n = 172) over 18 years old were recruited from the St. Michael's Hospital gynaecology clinic from January 2009 to March 2009. Subjects completed a self-administered cross-sectional quantitative questionnaire. The questionnaire was divided into three domains: (1) vaccine awareness/knowledge (2) vaccine attitudes, and (3) demographic information. Responses to questions generated a vaccine knowledge score from 0-10 and an attitudes score from 8-40, with higher scores indicating more positive vaccine attitudes.

Results: Vaccine awareness was significantly higher in Caucasians vs. Non-Caucasians (94% vs. 64%, $p < 0.0001$). In a multivariate logistic regression model, Caucasian ethnicity, higher education status, and greater number of years in Canada each emerged as unique predictors of vaccine awareness. Vaccine knowledge scores were higher in Caucasians vs. Non-Caucasians (7.2 vs. 6.4, $p = 0.0418$). Caucasians had more positive vaccine attitudes than Non-Caucasians (31.4 vs. 29.2, $p = 0.0211$). Greater vaccine knowledge was positively associated with interest in vaccination ($r2 = 0.26$, $p < 0.01$) and higher vaccine attitude scores ($r2 = .40$, $p < 0.0001$).

Conclusions: Ethnicity was a predictor of vaccine knowledge and attitudes, with Caucasian women having higher knowledge and attitudes scores. Improving HPV vaccination knowledge has the potential to improve attitudes and, ultimately, vaccine uptake. Culturally sensitive vaccination promotion may increase vaccine knowledge across ethnic groups.

ABSTRACT #P-I2

MOVING BEYOND PRIMARY SCREENING FOR CERVICAL CANCER IN LOW-RESOURCE SETTINGS

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The burden of cervical cancer is highest in the developing world where resources are limited. Screening programs using visual inspection with acetic acid (VIA) and see and treat programs with cryotherapy are well described in the literature and have been shown to be effective. In developed countries, follow-up of a positive screen is well established utilizing cytology, colposcopy and even HPV testing. However in the low-resource setting repeated cytology is not feasible. Cost effective, resource appropriate strategies need to be developed to address follow-up after treatment for a positive screen.

Objective: To develop a protocol for follow-up screening of women treated with cryotherapy for cervical dysplasia in resource poor settings.

Methods: Systematic literature review.

Results: Eight studies were found that address follow-up screening after treatment for cervical dysplasia and six looked at re-screening with VIA after cryotherapy. Sensitivities and specificities for detecting ≥CIN2 were 60% and 87-94% respectively. Based on the evidence, screening with VIA should commence at age ≥30. Following an initial negative screen, a second screen after age 40 is recommended. A positive screen should be treated with immediate cryotherapy and repeat VIA screen in one year. LEEP is recommended after a positive follow-up screen. If the squamocolumnar junction is not visible at any stage of the screening process, cytology is recommended followed by possible colposcopy and endocervical curettage.

Conclusions: An evidence-based, comprehensive follow-up strategy was developed for resource poor settings that addresses the ongoing management of women who have been treated for cervical dysplasia.

ABSTRACT #P-I3

INTERNATIONAL COOPERATION FOR CANCER CARE: PROTOCOL DEVELOPMENT FOR OVARIAN CANCER TREATMENT IN KENYA

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Objective: The proportion of affected women dying of gynecological cancers is unequally distributed around the world. There are several ways in which Canadian physicians may contribute to solving this problem. We aimed to develop and introduce a protocol for the treatment of advanced stage ovarian carcinoma in resource constrained conditions. In addition we explored the possibilities and barriers of international cooperation between physicians in Kenya and Toronto.

Methods: This project combines the knowledge, experience and effort of a group of Kenyan and Canadian medical students and gynecologists. In Moi Teaching and Referral Hospital in Eldoret, Kenya, the need for a protocol to treat women with advance stage ovarian carcinoma was identified. The standard chemotherapy treatment for this disease, a combination of Paclitaxel and Carboplatin, is not available in Kenya due to resource constraints. A systematic literature review was performed to identify papers describing experience with chemotherapy in low and middle income countries. Secondly randomised controlled trials comparing the different chemotherapy regimes that are attainable within the budget constraints in Moi Hospital were identified. Data extraction from the papers was performed by two medical students in Kenya and Toronto using e-mail and Skype for communication and data sharing.

Results: Developing a protocol is challenging. There is a paucity of data that are applicable to the Kenyan situation. Literature originating from low and middle income countries was insufficient to guide protocol development. The guideline was subsequently based on the results from older RCT's performed before the introduction of Paclitaxel. However establishing the proper tradeoff between effectiveness, toxicity, cost and acceptability can only occur through intensive discussion with all parties involved. Before introduction local financial sustainability will have to be examined. Introduction of the final guideline in Moi Hospital requires discussion of the content with staff and management involved in treating women with ovarian cancer. Barriers that might hamper clinical application of this guideline will have to be explored during introduction. Potential barriers include: acceptability to patients and support staff and logistical and organizational factors. Specific interventions will need to target such barriers.

Conclusions: International cooperation to develop a protocol is feasible in a timely matter with current communication methods. International cooperation is rewarding for all parties involved. Developing a protocol is but only the first step in successful quality improvement. Future evaluation will have to establish whether the new guideline will lead to a change in practice and improved outcome for women with ovarian cancer.

ABSTRACT #P-I4

Withdrawn

ABSTRACT #P-I5

TRAJECTORY OF CARE OF WOMEN REFERRED TO A TERTIARY HEALTH CARE CENTRE WITH ATYPICAL SQUAMOUS CELLS OF UNDETERMINED SIGNIFICANCE (ASCUS) OR LOW-GRADE SQUAMOUS INTRAEPITHELIAL LESIONS (LSIL) ON CERVICAL CYTOLOGY: A CROSS-SECTIONAL ANALYSIS

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Objective: To describe the trajectory of care of women referred to a tertiary care centre for evaluation of ASCUS or LSIL on cervical cytology with respect to the number of visits, cytology result from the initial consultation at the colposcopy clinic, final diagnosis (histology), and treatment.

Methods: Data from patients >18y with referral to the Princess Margaret Hospital/University Health Network for further evaluation of ASCUS or LSIL cervical cytology between January 1, 1998 – July 1, 2009, were drawn from the eCancerCare Colposcopy Clinic Information System (CCIS). The CCIS is a web-based application that allows prospective capture and management of sequential data on colposcopy clinic patients. A descriptive analysis of the characteristics and trajectory of care of these patients was performed.

Results: There were 830 complete records of women who met inclusion criteria, comprising 25% of the population in the database. Of these, 305 (37%) were referred for ASCUS (mean age 37.3 $\pm$ 12.1y) and 522 (63%) were referred for LSIL (mean age 33.6 $\pm$ 11.0y). Among those referred for ASCUS, the mean number of visits was 2.3 (range: 1-21), with 25% eventually requiring treatment. Among those referred for LSIL, the mean number of visits was 2.4 (range: 1-16), with 35% eventually requiring treatment. The role of HPV status will be correlated with the trajectory of care.

Conclusion: By retrospectively evaluating the management of patients referred with low-grade cytologic changes, we hope to add to the literature that informs management, including referral to colposcopy, among these patients, and the trajectory of care once referred.

ABSTRACT #P-J1

SPATIAL AND TEMPORAL EXPRESSION OF ACID CERAMIDASE IN PLACENTAL DEVELOPMENT AND IN PREECLAMPSIA

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ABSTRACT AVAILABLE IN HARDCOPY VERSION ONLY

ABSTRACT #P-J2

DUAL SPECIFICITY PHOSPHATASE 9 (DUSP9): A CANDIDATE GENE TO EXPLAIN THE MALE BIAS IN SEVERE PLACENTAL INSUFFICIENCY SYNDROMES

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Background and Objectives: Severe forms of early-onset preeclampsia (PE) and intrauterine growth restriction (IUGR) have a male predominance, suggesting a role for the X-chromosome in the pathogenesis of these placental diseases. Using micro-array and targeted Western expression studies, we identified abnormal expression of Dual Specificity Phosphatase-9 (DUSP9) in placentas from BPH/5 mice that develop hypertension and proteinuria late in pregnancy. DUSP9 negatively regulates mitogen activated protein kinases (MAPKs) which are involved in promoting cell proliferation and differentiation. The human DUSP9 gene is located on the X-chromosome and is thus a candidate to explain the male bias. Our specific objectives were first to create a subset of placentas from patients with well defined clinical characteristics of severe pure PE, pure IUGR, mixed PE+IUGR and normal age matched controls, and second to localize and quantify DUSP9 expression in these clearly defined groups.

Methods: Human placentas between 24^{+0} and 34^{+6} weeks gestation were obtained after delivery and classified into one of 4 gestational age-matched groups: 1) Pure IUGR (N=4, BW<10%ile, absent or reversed umbilical artery (UA) end diastolic flow (AREDF), no co-existing hypertension (HTN) and proteinuria); 2) Mixed PE+IUGR (N=20, BW<10%ile, AREDF, co-existing HTN±proteinuria); 3) Pure PE (N=11, BW>10%ile, ±AREDF, HTN (>140/90) and proteinuria (>300mg/24h, 1+ on dipstick)); 4) Controls (N=11, BW>10%ile, normal UA dopplers, no HTN or proteinuria, normal placental histology). We used immunohistochemistry and Western blotting to localize and quantify the DUSP9 protein across gestation and in the above groups.

Results: DUSP9 was localized to the villous trophoblast layer, predominantly in the cytoplasm of cytotrophoblasts (CT). Staining was most abundant in 1st trimester tissues where proliferating CTs predominate; staining was least in pathologic cases with villous CT depletion and syncytial knot formation. Western analysis revealed significantly decreased expression of DUSP9 in pure PE vs pure IUGR, with controls showing intermediate values.

Conclusions: Decreased DUSP9 expression is seen in cases of severe pure PE and may contribute to abnormalities in the structure and function of the villous trophoblast layer. We are presently testing 2 hypotheses: first, that the decreased DUSP-9 promotes mitotic activity of CTs in villous explants using knock down strategies; second that decreased DUSP9 in severe PE is regulated by methylation as it contains multiple CpG-rich sites.

Funded by: CIHR and the Department of Obstetrics and Gynaecology, Mount Sinai Hospital.

ABSTRACT #P-J3

THE ROLE OF PAR6 IN REGULATING TROPHOBLAST CELL POLARITY

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Objective: Human placental development is dependent upon the establishment and regulation of proper trophoblast cell differentiation events including fusion and invasion. Cell polarity plays an important role in cell differentiation shaping proper organogenesis via the generation of cell diversity and the execution of specific cellular functions. Accordingly, this study examines the contribution of cell polarity to trophoblast cell differentiation, an area of research which remains elusive, by examining Par6 (Partioning defective protein 6), a key regulator of cell polarity.

Methods: The temporal expression of Par6 was examined throughout placental development (5-39 weeks of gestation) by Western Blot analysis on placental lysates using Par6 (goat polyclonal, Santa Cruz) antibody. Spatial localization of Par6 was determined by immunofluorescence staining on placental sections. To establish a role for Par6 in trophoblast cell fusion, primary isolated trophoblast cells, known to spontaneously fuse in culture, were used and Par6 expression was examined spatially and temporally in conjunction with polarity marker, ZO-1. In addition, Par6 expression was assessed following forskolin (25uM) treatment in choriocarcinoma BeWo cells. To establish a role for Par6 in regulating trophoblast cell polarity Par6 siRNA strategy was employed in BeWo and JEG-3 cells and cell polarity markers including Zona occludin-1 (ZO-1) and E-cadherin were tested.

Results: Par6 expression was increased around 10-14wks of gestation. Furthermore, using immunofluorescence staining, early on in gestation, Par6 localized mainly to the nuclei of cytotrophoblast cells while, with advancing gestation, it shifted to the cytoplasm and it was found at the interface between cytotrophoblasts and syncytium. Silencing of Par6 in JEG3 and BeWo cells resulted in decreased expression of tight junction marker, ZO-1 and E-cadherin. Moreover, following forskolin treatment in BeWo cells, Par6 expression decreases compared to control vehicle-treated cells. In addition, preliminary data using primary isolated trophoblast cells revealed temporal changes in Par6 expression whereby Par6 was observed early on at tight junctions though following 48hrs in culture its expression became cytoplasmic and more diffused.

Conclusions: Par6 plays a role in regulating trophoblast cell differentiation via its effect on polarity during human placental development.

Funded by: CIHR/IGH and OGS.

ABSTRACT #P-J4

EXPRESSION OF VEGF-A IN THE PLACENTA, MATERNAL ORGANS, AND CIRCULATION DURING PREGNANCY IN MICE

Abhijeet Minhas [G], Shannon Bainbridge, Dawei Qu, Hoon-ki Sung, Andras Nagy, and S Lee Adamson. Samuel Lunenfeld Research Institute, Mount Sinai Hospital; Department of Obstetrics & Gynaecology, Mount Sinai Hospital and Department of Physiology, University of Toronto.

ABSTRACT AVAILABLE IN HARDCOPY VERSION ONLY

ABSTRACT #P-J5

HER-1 SIGNALING AND EXTRAVILLOUS TROPHOBLAST MIGRATION

Caroline E Dunk[O](1), JKWright(1,2), H Amsalem(1), C Maxwell(3), S Keating(4) & SJ Lye(1,2,3). (1)Women's and Infant's Health Research Centre, Samuel Lunenfeld Research Institute, Mount Sinai Hospital, (2)Depts of Physiology, (3)Obstetrics and Gynaecology and (4)Pathology, University of Toronto.

Objective: Decidua Conditioned Media (DCM), previously shown to induce differentiation of EVT in culture, contains many members of the Epidermal Growth Factor (EGF) family of ligands including EGF, Heparin Binding EGF (HBEGF), Amphiregulin and Transforming Growth Factor-α thạt are known to bind the HER-1 receptor. This study examines the role of HER-1 signaling and the differential effects of EGF and HBEGF in the differentiation of proliferative extravillous trophoblast (EVT) into invasive EVT.

Methods: JAR choriocarcinoma cell line and placental villous explants were used as experimental models and immunohistochemical and assessment of protein markers of EVT differentiation (HER-1, Cx40, α5 integrin downregulation, and HER-2 and α1 upregulation) was performed. Western blotting and multiplex phosphorylation assays were performed to assess activation of the downstream signaling resulting from HER1 phosphorylation.

Results: We show that the ability of DCM to induce invasive EVT differentiation was abrogated in the presence of the HER-1 antagonist, AG1478. Moreover, EGF (10ng/mL) treatment of JAR cells resulted in the downregulation of Cx40 and HER-1 expression and an upregulation of HER-2 expression whereas co-incubation with AG1478 inhibited this response. Similarly EGF treatment of placental villous explants downregulated HER-1 expression and upregulated HER-2 and α1 integrin expression, effects that were also inhibited with AG1478, However, EGF treatment did not downregulate Cx40 or induce migration, separation and invasion of either Jar cells or EVT. In contrast HBEGF induces a potent dose dependant migration of Jar cells that was inhibited by AG1478 or AG825 (HER2 inhibitor). Western blot analysis of HER-1 activation demonstrated DCM induced a rapid phosphorylation of HER1 at the Tyr^{1068} site, and subsequent activation of the PI3 Kinase and Akt signaling pathway. Moreover migration assays using a panel of signaling pathway inhibitors also demonstrated that DCM mediated migration was dependent on the PI3K pathway. Further investigation of the differential effects of EGF and HBEGF on the activation of the Map Kinase ERK pathway vs the PI3Kinase, Akt pathway is currently under investigation.

Conclusions: These results demonstrate that HBEGF activation of the HER-1 signaling through PI3 Kinase and Akt pathway is an important component of the invasive EVT differentiation cascade.

Funded by: CIHR 165436

ABSTRACT #P-K1

HUMAN AMNION TIGHT JUNCTIONS AND INTRAUTERINE INFECTION AND INFLAMMATION

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Objective: To determine the effects of *Lactobacillus rhamnosus* GR-1 (GR-1) supernatant on lipopolysaccharide (LPS) and lipoteichoic acid (LTA) stimulated cultured human amnion cell tight junction protein expression.

Methods: Placentae were collected from women undergoing elective cesarean section showing no signs of labour or clinical infection at Mount Sinai Hospital (Toronto, Ontario). The amnion was stripped and digested in 0.1% trypsin and 1% collagenase. Cells were plated until confluency was reached and serum starved followed by incubation with GR-1 supernatant for 12 hours. Cells were stimulated with either LTA or LPS and sampled after 12 hours. Including control, there were 6 treatment groups: GR-1, LTA, LTA+GR-1, LPS, LPS+GR-1. Proteins were extracted from cell lysates and analyzed by Western Blot. Cells were also fixed after 96 hours incubation using a 1:1 acetone and ethanol solution and immunocytochemistry was performed.

Results: E-cadherin and occludin were localized along cellular borders whereas zo-1 and claudin-1 were localized within cells after 96 hours of incubation.

Protein	Treatment Group				
	GR-1	**LTA**	**LTA+GR-1**	**LPS**	**LPS+GR-1**
Zo-1	2.00	1.16	0.91	2.35	1.48
E-cadherin	1.17	0.99	1.36	0.88	1.28
Occludin	1.36	1.16	1.38	1.30	1.29
Claudin-1	1.22	0.74	0.98	0.49	0.90

Table One: Ratios of tight junction protein expression relative to control across all treatment groups. Zo-1 (n=5), e-cadherin (n=5), occludin (n=4), claudin-1 (n=4). No statistical significance was observed across all treatment groups.

Conclusions: Preliminary results show no changes in e-cadherin, zo-1, occludin, and claudin-1 protein expression across all treatments. We are currently increasing the number of experiments in each group.

Funding: This study was funded by the Canadian Institutes of Health Research SGM:MOP-82799.

ABSTRACT #P-K2

PREVALENCE AND CHARACTERISTICS OF GROUP B STREPTOCOCCUS POSITIVE PREGNANT WOMEN IN AN INNER CITY TERTIARY CARE CENTRE

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Objective: To assess the prevalence and patient characteristics of Group B Streptococcus (GBS) positive pregnant women in an inner city tertiary care center.

Methods: A retrospective chart review was performed for all patients who delivered at our institution from Jan 1, 2008 to December 31, 2008. All GBS positive patients were identified, and data were abstracted regarding demographic characteristics, method of GBS detection (rectovaginal or urine culture), and GBS prevalence.

Results: During the study period 628 (22%) of 2879 patients who delivered at St Michael's Hospital in Toronto were identified as having GBS positive cultures. The mean age was 31 years. 25% were Canadian born, 44% were foreign born and country of birth was unknown for 30% of patients. Among GBS positive women, 575 (92%) of patients had a positive rectovaginal culture, 40 (6%) of patients had a positive urine culture, and 13 (2%) of patients had both a positive rectovaginal and urine culture. 481 (93%) of patients were treated with penicillin, 30 (6%) were treated with clindamycin, 3 with cefazolin, 2 with vancomycin, and 1 with erythromycin. 109 women who were GBS positive did not receive antibiotics. The treatment was unknown for 2 patients.

Conclusions: The GBS positivity rate among pregnant patients was similar in our inner city tertiary care centre to rates quoted in the literature among women in other populations.

ABSTRACT #P-K3

H1N1 IN PREGNANCY: A TERTIARY CARE CENTRE EXPERIENCE

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Objective: To elucidate factors pertinent to the course and outcome of H1N1 in pregnancy.

Methods: This is a retrospective chart review of H1N1 afflicted pregnant patients conducted at Mount Sinai Hospital (MSH) in Toronto, Ontario. All women who tested positive for H1N1 between June 1, 2009 and December 5, 2009 were identified. Records were reviewed to determine pregnancy status. Clinic and hospital charts of pregnant patients were reviewed and information gathered according to data collection forms previously approved by the Mount Sinai Hospital Research Ethics Board.

Results: Of 42 patients, there were 12 inpatients and 30 outpatients. 25/42 (60%) presented in the third trimester. 14/42 (33%) had co-morbidities. 61% were afebrile, thus not meeting the CDC's definition of Influenza-Like-Illness. Antivirals were administered promptly in most, yet delays resulted in 1/3 being treated past 48 hours from symptom onset. 30/42 (71%) did not require hospitalization. 7/12 (58%) of hospitalized patients were admitted for reasons unrelated to H1N1. While 3/12 (25%) of patients were delivered at the time of discharge, no deliveries occurred because of H1N1. 88% delivered at term. 55% of deliveries occurred by cesarean section, but none were necessitated by H1N1. 85% of infants were appropriately grown, and none were admitted to the NICU secondary to H1N1.

Conclusions: H1N1 in pregnancy has the propensity to result in significant maternal and fetal morbidity and mortality, and requires vigilance in assessment and prompt treatment. In contrast to reports published to date, our cohort experienced a largely uncomplicated course of illness with minimal fetal and maternal impact in most instances.

ABSTRACT #P-K4

A NOVEL ROLE OF NUCB2 IN REGULATING THE INITIATION OF LABOUR

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Labour is a multi-factorial process associated with inflammatory responses and steroid hormone regulation. Nucleobindin (Nucb2), an ubiquitously expressed protein with DNA and Ca2+- binding capabilities, can regulate COX-mediated prostaglandin (PG) synthesis and Ca2+ homeostasis. Uterine PG expression is regulated by progesterone (P4) and estrogen (E2). We hypothesize that Nucb2 contributes to the initiation of term and preterm labour (PTL) and that its expression is modulated by P4 and/or E2.

Objective: Study the expression of Nucb2 in rat myometrium during gestation and under steroid hormone modulation.

Methods: We used animal models of (1) term labour; (2) P4-delayed labour: rats [n=4/group] randomized to receive daily injections of either MPA (P4, 16 mg/kg, s.c.) or vehicle (V) starting on day 20 of gestation, samples collected on days 21, 22, or 23 (during labour) in the V group or days 21, 22, 23, or 24 in the P4 group; (3) RU486-induced PTL: on day 19 of gestation, two groups of rats [n=4/group] treated with either P4 antagonist (RU486, 10 mg/kg, s.c.) or V, samples collected on day 20 from non-labouring (V) and during PTL in RU486-treated animals; and (4) hormone treated ovariectomized (OVX) non-pregnant rats: adult OVX rats [n=5/group] treated with E2 (10μg/kg, s.c.) followed 12 hours later by a second treatment of E2 with/without P4 (16 mg/kg, s.c.). mRNA expression of Nucb2 in myometrial samples was measured by Real-time PCR.

Results: Experiments revealed that (1) Nucb2 mRNA level decreased by 75% during labour (d23) compared to day 21 of pregnancy (P=0.018); (2) P4 administration prevented both the decrease in Nucb2 expression and the onset of term labour (d23V vs d23P, P=0.004; d23V vs d24P, P=0.008); (3) Blockage of P4 signaling repressed Nucb2 gene expression (P=0.047) and induced PTL; (4) P4 administration induced a 3 fold increase in Nucb2 expression in OVX rats after priming with E2 ($P<0.001$).

Conclusions: Nucb2 mRNA expression decreased at both term and PTL and was subject to the regulation by P4 signaling, implying a putative role for Nucb2 in the regulation of labour initiation. Additional studies are in progress to confirm myometrial protein expression by means of western blot analysis throughout gestation.

Funded by: CIHR# 42378

ABSTRACT #P-K5

IMMUNOPHENOTYPING OF PERIPHERAL BLOOD LEUKOCYTES IN PREGNANT AND TERM LABOURING WOMEN

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Objective: There is limited information about maternal immunophenotypic profile in normal and pathological pregnancies. We hypothesized that activation of peripheral blood leukocytes (PBL) is important early in parturition and sought to analyze peripheral blood from women in active term labour (TIL) and preterm labour (PTL), compared to those not in labour undergoing elective Caesarean section (TNIL) and age-matched mid-pregnant control women (PTNIL). Activated peripheral immune cells target the myometrium, inducing an inflammatory response contributing to labour. We analyzed the infiltration of immune cells to the myometrium before and during labour.
Methods: 1. We used six-color flow cytometry and a panel of specific monoclonal antibodies to characterize subpopulations of peripheral leukocytes and their activation status in four patient groups. The FACSAria Flow Cytometer (Becton Dickinson) and BD Diva software were used for data collection. FlowJo software (Tree Star) was used for flow cytometry data analysis. Leukocytes (lymphocytes, granulocytes, and monocytes) were gated using forward and side scatter plots and a common leukocyte marker (CD45-APC-Cy7) in order to eliminate doublets and all other cell types from subsequent analysis. Anti-CD3, -CD4, -CD8 antibodies were used for the T lymphocyte subgroup, whereas anti-CD14, anti-CD15 and anti-CD19 were used to detect monocytes, granulocytes and B cells, respectively. Three surface activation markers (CD55, CD44 and CD11b) were chosen to identify the activation status of the leukocyte subpopulations. CD55, a complement regulatory protein, is a multifunctional cell-surface receptor present on leukocytes and all tissues exposed to maternal serum. CD44 is the receptor for hyaluronic acid and multi-function cell surface adhesion molecule involved in cell-cell and cell-matrix interactions. CD11b mediates leukocyte adhesion to endothelial cells and subsequent migration out of the vasculature. 2. The myometrial tissue biopsies were collected from term patients (TIL, n=2 and TNIL, n=15) and used to detect whether activated immune cells target the myometrium before or during labour. Two different antibodies, anti-CD45 (pan-leukocyte marker) and anti-CD68 antibody (macrophage specific marker) were used for immunohistological analysis.
Results: 1. Peripheral leukocytes contain typical subpopulations, identified by specific markers; there were immune alterations in blood samples of labouring (both TIL, n=18 and PTL, n=7) vs non-labouring women (both TNIL, n=11 and PTNIL, n=7). Activation status of all the leukocyte subpopulation was increased at term. The majority of granulocytes (90-95%) were CD15+CD14- neutrophils, all expressing activation markers CD44, CD55 and CD11b. Monocytes subpopulation showed the high expression of all three markers studied (CD11b,CD44 and CD55). B cells were CD55+ and CD44+, but CD11b was low. The majority of T cells were CD11b negative but CD55+ and CD44+. Analysis of the Mean Fluorescent Intensity (MFI) of the surface activation markers showed increased levels of CD55 expressed on granulocytes and monocytes in both labouring groups(PTL,TIL).MFI levels of CD44 were high on the monocytes, granulocytes and B-cells in the PTL group compared to other groups. 2. Visual observation of the myometrial tissues showed the number of CD45+ cells was readily detectable. The number of CD68+ macrophages was higher in labouring vs non-labouring myometrial biopsies.

ABSTRACT #P-L1

PPROM in Twins: From A to B, In Progress

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Objective: The majority of studies to date regarding PPROM have not included twin pregnancy. Though physiologically plausible, none of the studies on PPROM and twins have specified any difference in the clinical outcome of PPROM in Twin A versus B. The purpose of our study is to seek out any clinically significant differences with regard to pregnancy latency or neonatal outcome in PPROM of Twin A versus Twin B that may be relevant to future patient counseling and care.

Methods: A chart review of all patients admitted to Sunnybrook Health Sciences Centre with PPROM and twin gestation from Jan 1, 2004 to Dec 31, 2008 is in progress. Patient demographic characteristics and risk factors for preterm labour and PPROM were collected. Measures applied to the management of PPROM were recorded. Labour and delivery details were collected. Postpartum complications, both fetal and maternal, were determined where possible.

Results: In total, 25 of 166 maternal charts have been reviewed. The average maternal age was 31.5. 18 primiparas and 7 multiparas were included in the analysis. Of the twin gestations, 18 were dichorionic and 7 were monochorionic. The average gestational age at PPROM was 32.2 weeks. Only one Twin B PPROM has been identified to date. 14/25 delivered less than 24 hours after confirmed rupture of membranes, 10/25 within 1 day and only 1/25 within 2 days. 11/25 received antibiotic therapy. 13/25 received prophylactic steroids at some point in the pregnancy. Only 4/25 were delivered for indications other than labour: 2/25 for non-reassuring fetal heart rate status, 1 for cord prolapse and 1 for malpresentation of Twin A at 35 weeks gestation. The mode of delivery differed slightly between Twin A and B: 7 vs 5 SVD, 4 vs 4 AVD and 14 vs 16 CS. Overall, more Twin B than Twin A had low (<7) 1 minute (11/25 vs 7/25) and 5 minute (4/25 vs 1/25) Apgar scores. Of known risk factors for PPROM and preterm delivery, 11/25 had infertility treatments with either intrauterine insemination or in vitro fertilization. 3/25 underwent fetal reduction, 1/25 had an amniocentesis and 1/25 underwent laser therapy for twin-to-twin transfusion at 18 weeks. 3/25 had previous cervical LEEP procedures, 5/25 had either a short (<2.5 cm) or dilated cervix at ultrasound and 2/25 had a cerclage placed in the second trimester.

Conclusions: Unfortunately, the few charts of the series reviewed to date limits our conclusions to observation alone. However, some interesting trends may be emerging. In our case series, there are a significant number of pregnancies affected with known (and some modifiable) PPROM risk factors. There is some suggestion of greater obstetrical intervention required for the safe delivery of Twin B (more operative birth) and poorer overall outcomes of Twin B (seen by lower 1 and 5 minute Apgar scores) even in the case of Twin A PPROM. Ultimately, however, greater numbers of subjects are required to draw definitive conclusions.

ABSTRACT #P-L2

SURVEY OF MENSTRUAL CYCLES: THE PREVALENCE OF MENORRHAGIA AND ITS IMPACT IN THE WORKFORCE

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Objective: To investigate the prevalence of heavy menstrual bleeding in reproductive aged women, and to examine its impact on women in the workforce.

Methods: From June 2008 to November 2009 surveys were distributed at four general practitioners' offices in Toronto, Ontario to women between the ages of 18 and 55 who had at least one menstrual period in the last year. The questionnaire included twenty-three questions related to demographics, occupation, menstrual cycles and possible impact of menorrhagia at home and at work.

Results: Three hundred and three surveys were completed and returned. Forty-six percent of the respondents (138/303) considered their menstrual periods to be heavy and of these women, 46% reported a history of anemia. Forty-seven percent of women with heavy menses also reported having missed work or school with 80% (48/60) of these women missing at least one day in the last year (mean =8.35 days/year). There was no difference between employed and self-employed women. Interestingly, some of these women felt that dysmenorrhea was a bigger contributing factor to their absenteeism.

Conclusions: Approximately half of the women surveyed considered their periods to be heavy and almost half of these women had a history of anemia. Almost 50% of the women with heavy menses also reported missing days off work or school. These numbers appear to be higher than those reported in the literature and may suggest that the prevalence of menorrhagia and its impact on women is much more significant than previously thought.

ABSTRACT #P-L3

HOW DOES THE UNIVERSITY OF TORONTO HYSTERECTOMY TRAINING COMPARE TO THE REST OF CANADA?

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Objectives: This study aims to compare the hysterectomy surgical training and comfort level acquired by recent Canadian obstetrics and gynaecology graduates with University of Toronto graduates.

Methods: A national survey was designed to determine the hysterectomy surgical training received by obstetrics and gynaecology residents across Canada and their practice plans. It was electronically distributed to 289 recent graduates (2005-2010).

Results: The response rate was 37% across Canada (107 respondents), with a 62.5% response rate from University of Toronto (UofT) graduates. Fewer UofT graduates performed zero laparoscopic-assisted vaginal hysterectomies (LAVH), laparoscopic sub-total hysterectomies (LSTH) and total laparoscopic hysterectomies (TLH) compared to other Canadian graduates (11.5%, 10.7% and 17.9% respectively vs 19.2%, 37% and 57.5%). Despite this, there was no significant difference in comfort level between UofT and other Canadian graduates in performing LSTH (42.9% vs 41.7%) and LAVH (71.4% vs 60.3%). However, there was a significant difference in comfort level of performing TLH, with 48.1% of UofT graduates comfortable compared to 17.8% of other Canadians ($p < 0.002$) and statistically more UofT graduates perform/plan to perform TLH (80.8% vs 52.5%, $p< 0.013$). Conversely, 53.5% of UofT graduates performed at least 50 abdominal hysterectomies (AH) and 42.9% performed at least 21 vaginal hysterectomies (VH) compared to 74% and 68% of other Canadian graduates, respectively. Despite this, the majority of all Canadian residents reported they were comfortable performing AH (100% UofT vs 98.6% other Canadian) and VH (85.7% UofT vs 87.5% other Canadian).

Conclusions: The significantly higher level of laparoscopic hysterectomy surgical training at UofT translates into increased comfort level and future performance plans amongst graduating residents, without impacting the comfort level of executing AH and VH. Given the substantial benefits of vaginal and laparoscopic hysterectomy compared to laparotomy, we should continue to strive for further educational initiatives to ensure graduates across Canada are capable of performing all types of hysterectomy.

ABSTRACT #P-L4

DO PODCASTS MEASURE UP? MEDICAL STUDENTS' PERCEPTIONS OF THE UTILITY OF PODCASTS IN MEDICAL EDUCATION (WORK IN PROGRESS)

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Introduction: There has been an increasing interest in web-based tools, such as podcasts, for dissemination of research and education. Podcasts are digital media files available in both audio and audio-visual formats. Podcasts have the advantages of being inexpensive, easily accessible, and can be conveniently listened to almost anywhere with a portable MP3 player or laptop computer. Many educational institutions use technologies such as podcasts to supplement curriculum. Medical student have limited time, particularly in their clinical years, requiring them to multitask. Podcasts can deliver a digestible amount of information on demand, making them an attractive tool for medical education.

Objective: To assess the perceived utility of Obstetrical and Gynaecological podcasts among undergraduate medical students during their Obstetrics and Gynaecology rotation at the University of Toronto.

Methods: We have created 6 podcasts, 15-35 minutes in length, covering Obstetrics and Gynaecology topics. The podcasts review relevant course material as outlined by the course objectives and are posted on the internet for free download. During this academic year, approximately 240 third-year medical students will rotate through a 6-week Obstetrics and Gynaecology block and will have access to these podcasts. At the end of each of the rotations, the medical students will be provided with a questionnaire assessing the use of digital study tools and the perceived utility of the obstetrics and gynecology podcasts for their learning of the material and preparation for their examination.

Results: The questionnaires will be collated as each group of students completes their rotations for further analysis.

Conclusions: In progress.

ABSTRACT #P-L5

THE EFFECT OF REIKI ON PAIN IN WOMEN AFTER ELECTIVE CAESAREAN SECTION – A DOUBLE-BLINDED RANDOMIZED CONTROLLED TRIAL

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Objective: Reiki is an ancient Japanese form of healing where the practitioners transfer healing energy through light touch and positive healing intention. The objective of this study was to assess the effectiveness of Reiki in reducing pain following elective Caesarean section.

Methods: In this randomized, double-blinded study, women who underwent an elective Caesarean section were allocated to receive either usual care (control, n=40) or three sessions of remote Reiki in addition to usual care (n=40). Pain was assessed using a visual analogue scale (VAS). The primary endpoint was the Area Under the VAS-time Curve (AUC) for days 1 to 3. Secondary measures of pain included: proportion of women who required opioid medications, dose of opioid medication consumed, rate of healing and vital signs (heart rate, respiration rate and blood pressure).

Results: AUC for pain was not significantly different for the Reiki group compared to the control group (mean ± SD; 212.1 ± 104.7 vs. 223.1 ± 117.8; p=0.96). There were no significant differences in opioid consumption or rate of healing. Compared to the control group, the Reiki group had a small but significantly lower heart rate 4 hours post surgery (74.3 ± 8.1 bpm vs. 79.8 ±7.9 bpm, p=0.003) and systolic blood pressure on day 3 (106.4 ± 9.7 mmHg vs. 111.9 ± 11.0 mmHg, p=0.02).

Conclusions: Remote Reiki had no significant effect on pain following an elective Caesarean section.

Funded by: *No funding source*

INDEX
Presenters by Last Name

Presenters by Last Name cont'd

O7	Kalkat, Manpreet	G	Isabella Caniggia
G6	Kenigsberg, Shlomit	O	Clifford Librach
O9	Kirkham, Yolanda	R	Lisa Allen
K1	Koscik, Rebecca	G	Alan Bocking
L3	Kroft, Jamie	R	Patricia Lee
K4	Li. Yunqing	G	Stephen Lye
K3	Malinowski, Ann	F	Cynthia Maxwell
O13	Mansour, Fady	F	Guylaine Lefebvre
O10	McGee, Jacob	F	Steven Narod
O12	Menzies, Rebecca	M	Wendy Wolfman
J4	Minhas, Abhijeet	G	Lee Adamson
E5	Moisiadis, Vasilis	O	Stephen Matthews
I4	Morton, Matthew	F	Sari Kives
D2	Moskovtsev, Sergey	O	Clifford Librach
E6	Mousa, Noha	F	Robert Casper
G2	Mussani, Fareen	M	Prati Sharma
D3	Naranian, Taline	G	Andrea Jurisicova
O14	Nedd-Roderique, Tamara	G	Stephen Lye
D4	Omari, Shakib	G	Andrea Jurisicova
L1	Packenham, Susan	R	Ori Nevo
F5	Pham, Alice	R	Mark Yudin
E4	Proulx, Eliane	G	Evelyn Lambe
C3	Racano, Antonella	G	Isabella Caniggia
C5	Ray, Jocelyn	G	Isabella Caniggia
H5	Rennie, Monique	G	Lee Adamson
O11	Robertson, Julie	F	Mathew Sermer
K5	Sabra, Sally	G	Stephen Lye
I1	Sadry, Sharon	M	Mark Yudin
A6	Shathasivam, Premalatha	G	Ted Brown
K2	Shore, Eliane	R	Mark Yudin
D1	Siddiqui, Raheela	R	Valerie Dube
I5	Simpson, Andrea	M	Joan Murphy
L4	Sisca, Lyana	R	Michele Farrugia
J3	Sivasubramaniyam, Tharina	G	Isabella Caniggia
C2	Sobel, Mara	R	John Kingdom
L2	Sutandar, Marilyn	R	Wusun Paek

Presenters by Last Name cont'd

J1	Tal, Reshef	PD	Isabella Caniggia
O5	Tone, Alicia	PD	Ted Brown
I3	van Lonkhuijzen, Luc	F,G	Barry Rosen
L5	vanderVaart, Sondra	G	Howard Berger
G3	Velummailum, Russanthy	G	Andrea Jurisicova
A5	Vicus, Danielle	F	Steven Narod
H2	Walker, Melissa	O	John Kingdom
O6	Wegener, Marie	M	Sarah Ferguson
F3	Wise, Michelle	G	Geoff Anderson
E1	Xiao, Rong	PD	Clifford Librach
D5	Yavorska, Tetyana	G	Andrea Jurisicova
O15	Yeganegi, Maryam	PD	Alan Bocking
G4	Zaver, Shirin	G	Clifford Librach

*F=Clinical Fellow; G=Graduate Student; M=Medical Student; PD=Post-Doctoral Fellow; R=Resident; O=Other (non-eligible for awards; eg. non-trainee, non U of T)

Presenters by Abstract # and Session

ORALS (O)

Morning

Oral Session I (8:30-9:45 a.m.)

O1 Bainbridge, Shannon
O2 Iqbal, Majid
O3 Chu, Kelly
O4 Bortolini, Maria
O5 Tone, Alicia

Oral Session II (11:10 a.m. -12:10 p.m.)

O6 Wegener, Marie
O7 Kalkat, Manpreet
O8 Baczyk, Dora
O9 Kirkham, Yolanda

Afternoon

Oral Session III (1:20-2:50 p.m.)

O10 McGee, Jacob
O11 Robertson, Julie
O12 Menzies, Rebecca
O13 Mansour, Fady
O14 Nedd-Roderique, Tamara
O15 Yeganegi, Maryam

Presenters by Abstract # and Session cont'd

POSTERS (P)

SESSION I (MORNING) (Groups A-F) (9:45-11:05 a.m.)

Poster Group A
A1 Durbin, Joshua
A2 Greenaway, James
A3 Abrol, Kaajal
A4 Chiu, Hannah
A5 Vicus, Danielle
A6 Shathasivam, Premalatha

Poster Group B
B1 Chun, Lauren
B2 Devarajan, Karthika
B3 Alfaraj, Malikah
B4 Hui, Dini
B5 Amsalem, Hagai
B6 Frasca, Erika

Poster Group C
C1 Gregory, Allison
C2 Sobel, Mara
C3 Racano, Antonella
C4 Drewlo, Sascha
C5 Ray, Jocelyn

Poster Group D
D1 Siddiqui, Raheela
D2 Moskovtsev, Sergey
D3 Naranian, Taline
D4 Omari, Shakib
D5 Yavorska, Tetyana

Poster Group E
E1 Xiao, Rong
E2 Goodfellow, Nathalie
E3 Emack, Jeff
E4 Proulx, Eliane
E5 Moisiadis, Vasilis
E6 Mousa, Noha

Poster Group F
F1 Grossman, Stacey
F2 Barlow, Erin
F3 Wise, Michelle
F4 Estanbul, Fatuma
F5 Pham, Alice

Presenters by Abstract # and Session cont'd

POSTERS (P)

SESSION II (AFTERNOON) (Groups G-L) (2:50-4:10 p.m.)

Poster Group G
G1 Blanco Mejia, Sonia
G2 Mussani, Fareen
G3 Velummailum, Russanthy
G4 Zaver, Shirin
G5 Chan, Crystal
G6 Kenigsberg, Shlomit

Poster Group H
H1 Bhuiyan, Manzerul
H2 Walker, Melissa
H3 Audette, Melanie
H4 Hazan, Aleah
H5 Rennie, Monique
H6 Deda, Livia

Poster Group I
I1 Sadry, Sharon
I2 Jumah, Naana
I3 van Lonkhuijzen, Luc
~~I4 Morton, Matthew~~ (Withdrawn)
I5 Simpson, Andrea

Poster Group J
J1 Tal, Reshef
J2 Czikk, Marie
J3 Sivasubramaniyam, Tharina
J4 Minhas, Abhijeet
J5 Dunk, Caroline

Poster Group K
K1 Koscik, Rebecca
K2 Shore, Eliane
K3 Malinowski, Ann
K4 Li, Yunqing
K5 Sabra, Sally

Poster Group L
L1 Packenham, Susan
L2 Sutandar, Marilyn
L3 Kroft, Jamie
L4 Sisca, Lyana
L5 vanderVaart, Sondra

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Faculty of Medicine, University of Toronto
92 College St.
Toronto, Ontario M5G 1L4

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Dr. Jane Norman

Jane Norman graduated in Medicine from the University of Edinburgh in 1986. The early part of her clinical and academic training in obstetrics and gynaecology was in Edinburgh, under the supervision of Professors David Baird, Andrew Calder and Rodney Kelly. She was awarded the degree of MD by the University of Edinburgh in 1992. Jane moved to a Clinical Lecturer post at the University of Glasgow in 1993 and progressed to the Regius Chair of Obstetrics and Gynaecology at the University of Glasgow in 2007. In 2008, she was appointed to the Chair of Maternal and Fetal Health at the University of Edinburgh. She is currently the Director of the Edinburgh Tommy's Centre for Maternal and Fetal Health, and the Research Director for the Jennifer Brown Research Laboratory.

Her current research activity centres on the issues of obesity in pregnancy and preterm birth – both of which are major contributors to maternal and neonatal mortality and morbidity. She is Chief Investigator of the clinical trial "OPPTIMUM" – a UK multicentre study (estimated over 30 centres), funded by the Medical Research Council (£2.7 million) and which will determine if progesterone prevents preterm labour and improves neonatal outcome in high risk singleton pregnancy.

In addition, Jane has a clinical role of Consultant Obstetrician at the Royal Infirmary of Edinburgh. Her national and international roles include membership of The Maternal and Perinatal Health Research and Epidemiology Advisory Group at the WHO and the UK Confidential Enquiry on Maternal Health.

Jane is a former Royal College of Obstetricians and Gynaecologists Blair Bell Lecturer and Sims Black Travelling Professor. She has also been Visiting Professor at the Universities of Loma Linda (USA), Calgary and Edmonton (Canada) and Newcastle and Sydney (Australia). She was recently awarded the 2009 Society for Gynecologic Investigation (SGI) President's Achievement Award, an annual award made to a member of the society whose "record in scientific investigation is outstanding and assures a continued productive career in research", and awarded to a UK investigator only once previously in its 25 year history.

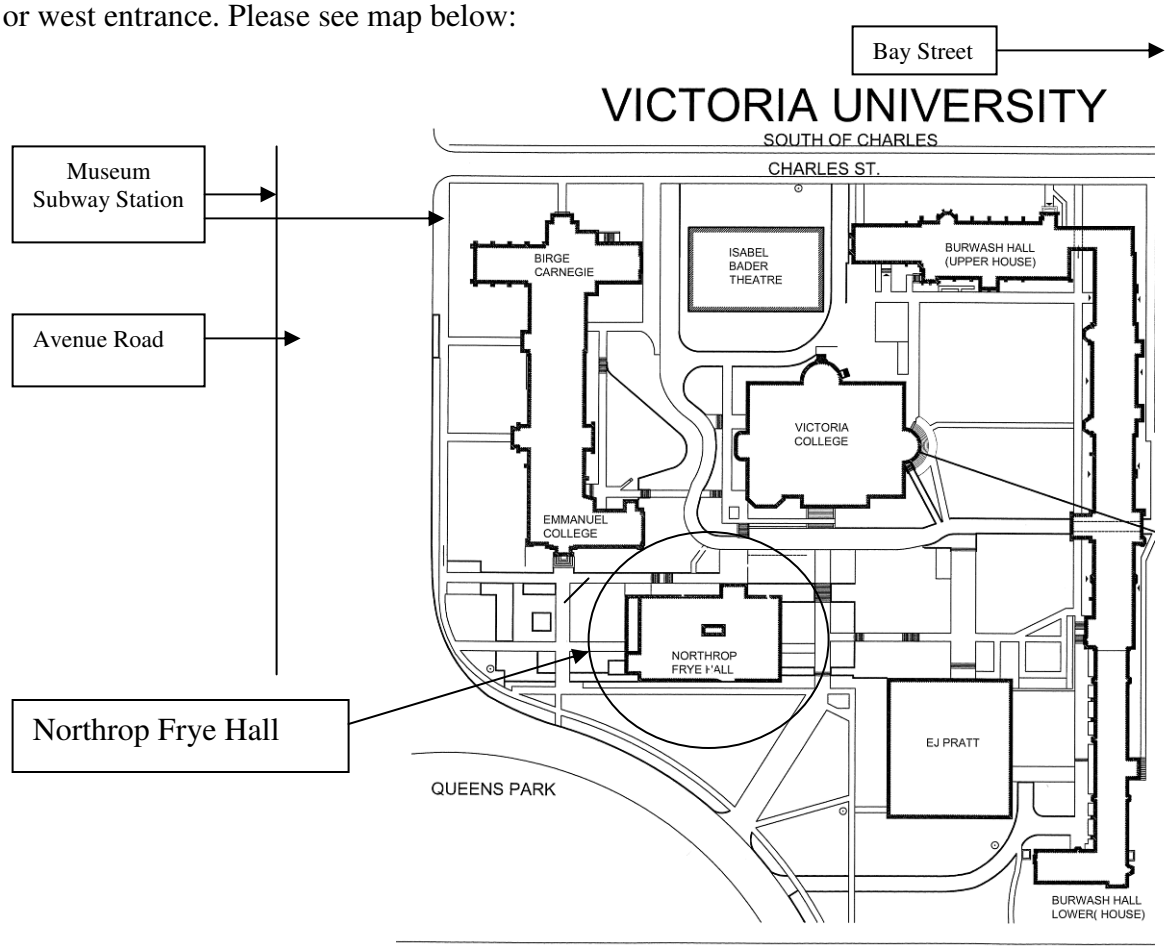
Jane's hobbies include spending time with her husband and two children (aged 10 and 11); and playing bridge and the piano.

Obstetrics & Gynaecology
UNIVERSITY OF TORONTO

Research Day, Friday, May 7, 2010

Location:
Northrop Frye Hall, Ground Floor
Victoria University, 73 Queen's Park Crescent East
University of Toronto M5S 1K7

Northrop Frye Hall is part of Victoria University, University of Toronto, and is located at 73 Queen's Park Crescent East, northeast of Queen's Park and east of Avenue Road, south of Charles St. and near the Museum subway station. You may use either the north or west entrance. Please see map below:



By subway: Go to the Museum subway stop and cross underground to the east side of Queen's Park/Avenue Road. Proceed south along Queen's Park/Avenue Road past the south end of Emmanuel College, where you will see walkways leading east to the Hall. You may enter from the west or north doorways.

* Parking is available on the street at Charles St. West (one way and accessible from Bay Street) and at the southwest corner of Charles Street West and Bay St. in a public parking lot.

27th Annual Research Day & Henderson Lecture

The Department of Obstetrics & Gynaecology 27th Annual Research Day took place Friday, May 7, 2010.

There were 15 oral and 61 poster presentations by trainees from the department, covering a range of excellent research, both basic and clinical. We also heard an excellent Henderson Lecture, delivered by Dr. Jane Norman, University of Edinburgh, UK, on the topic of "Being born too soon – do obstetricians have anything to offer?" Dr Norman is a specialist in preterm birth and inflammation.

Dr. Jane Norman
Henderson Lecturer

I would like to thank everyone who participated and extend special thanks to all those faculty members who acted as Chairs and Judges.

The **2010 JW Knox Ritchie Research Awards** for best abstract/presentation by trainee category were awarded during the celebratory wine and cheese reception at the end of the day. I am pleased to announce the following winners:

Clinical Fellow: Dini Hui (Supervisor: N. Okun) for Poster Presentation, B4, Combinations of First and Second Trimester Maternal Serum Biochemical Markers and Prediction of Adverse Pregnancy Outcomes: Systematic Review and Meta-Analysis. Dini Hui, Prakeshkumar Shah, Kellie Murphy, Elizabeth Uleryk, John Kingdom, Nan Okun.

Graduate Student: Jocelyn Ray (Supervisor: I. Caniggia) for Poster Presentation, C5, A Story of Life and Death: The Dual Role of Mtd/Bok in Trophoblast Cell Fate. Jocelyn Ray, Julia Garcia, Yuan Wu, Tullia Todros, Andrea Jurisicova, Isabella Caniggia.

Post-Graduate Fellow: Alicia Tone (Supervisor: T.J. Brown) for Oral Presentation, 05, DAB2 Enhances Glucocorticoid Receptor-Mediated Anti-Inflammatory Signalling in ES2 Ovarian Cancer Cells. Alicia Tone, Carl Virtanen, Patricia Shaw, Theodore J. Brown.

Resident: Mara Sobel (Supervisor: J. Kingdom) for Poster Presentation, C2, Angiogenic response of the human placenta to heparin: Implications for the prevention of pre-eclampsia? Mara Sobel, Sascha Drewlo, John Kingdom.

Medical Student: Marie Wegener (Supervisor: Sarah Ferguson) for Oral Presentation, O6, Validation of the Modified Sexual Adjustment and Body Image Scale in Women with a Diagnosis of Gynaecologic Cancer (SABIS-G) Marie Wegener, Sara Urowitz, Catherine Classen, David Wiljer, Christine Massey, Sarah E. Ferguson.

In addition, the **Papsin Award** for postgraduate resident in final year of training, based on teaching ability, mentorship activities and leadership, as chosen by peers, was awarded to**Rebecca Cash**.

Please join me in congratulating the winners in each of these categories, as well as Dr. Cash for this recognition by her peers. I would also like to commend all the participants, both oral and poster presenters, for their valuable contribution to the continued success of Research Day.

Alan D. Bocking, MD
Gordon C. Leitch Chair
Department of Obstetrics and Gynaecology

Call for Abstracts
27th Annual University of Toronto Department of Obstetrics and Gynaecology Research Day, Friday, May 7, 2010

Dear Faculty, Staff, Trainees and Guests:

The 27th Annual Research Day of the University of Toronto Department of Obstetrics and Gynaecology will take place on **Friday, May 7, 2010** at Northrop Frye Hall, Ground Floor, Victoria University, 73 Queen's Park Crescent East, in the University of Toronto.

We are very pleased to have **Dr. Jane Norman**, Professor of Maternal and Fetal Health, University of Edinburgh, UK, and Co-Director, Edinburgh Tommy's Centre for Maternal and Fetal Health Research, as our **2010 Henderson Lecturer**. Dr. Norman is a clinician-scientist specializing in preterm birth and inflammation and her topic is, "Being born too soon – do obstetricians have anything to offer?"

This year's **abstracts** for oral and poster presentations are **due on Friday, March 5, 2010.** Please note that those submitting abstracts are asked for their preference of oral or poster presentation**. Please confer with your supervisor to ensure only one request for an oral comes from each supervisor.**

Please go to our website at http://www.obgyn.utoronto.ca/Research/ResearchDay.htm for the following information and links for **abstract submission**:

Abstract Requirements and Template (required for submission)
Contact Information Form (required for submission)
Instructions for Oral Presenters (coming soon)
Instructions for Poster Presenters (coming soon)
Awards Criteria
Research Day Poster
Research Day Booklet (coming soon)
Research Day Programme (coming soon)
Location

If you have any questions with regard to these documents or the process, please contact me at helen.robson@utoronto.ca.

We look forward to another excellent Research Day, an opportunity to exhibit and share all the cutting-edge research in the department!

Best regards,

Helen Robson
Consultant; Research Coordinator

INSTRUCTIONS FOR ORAL PRESENTERS

University of Toronto Department of Obstetrics and Gynaecology
Research Day, Friday, May 7, 2010

Thank you for submitting your abstract for Research Day. Your abstract has been chosen for an oral presentation. Please see the details below.

Location: Northrop Frye Hall at Victoria University in the University of Toronto. Northrop Frye Hall is located at 73 Queen's Park Crescent East, northeast of Queen's Park and east of Avenue Road, south of Charles St. and near the Museum subway station. You may use either the north or west entrance. A map is available on our website.

Time: Research Day will begin with breakfast at 8:00 a.m. on Friday, May 7, 2010 and end with a wine and cheese reception and award presentation from 5:25 to 6:30 p.m.

Registration: Please register on the ground floor level of Northrop Frye Hall between 8:00 and 8:25 a.m. to receive your nametag, abstract booklet and any further instructions.

Oral Session Times: There will be three oral sessions, two in the morning (8:30-9:45 a.m. and 11:10-12:10 a.m.) and one in the afternoon (1:20-2:50 p.m.).

Presentation: Each oral presentation is allowed 15 minutes: 10 minutes for the presentation and 5 minutes for questions**. Please note that we will adhere strictly to these time limits!**

Audiovisual Support: All presentations will be placed on the Department's laptop. Please bring a **CD** of your presentation or a **USB key** to the Department at 92 College St, **as early as Monday, May 3, 2010, but NO LATER THAN WEDNESDAY, May 5, 2010.** Your presentation must be PC-compatible. If you foresee any difficulties, please contact the Departmental Assistant ahead of time (416-978-2668). **Please provide your own backup for Research Day.**

Awards: In order to be eligible for an award, you must be a U of T trainee and present your own work. Your work and presentation will be judged by the Chair of your oral session and two other Judges for the JW Knox Ritchie Research Awards. There will be 5 awards, with a monetary component, based on level of training (Graduate Student, Resident, Clinical Fellow, Post-Doctoral Fellow, Medical Student), rather than type of presentation. (Please see judging criteria on our website.) These awards will be presented at the wine and cheese reception between 5:25 and 6:30 p.m.

***Please see http://www.obgyn.utoronto.ca/Research/ResearchDay.htm for information or contact Helen Robson at* helen.robson@utoronto.ca**

FINAL INSTRUCTIONS FOR POSTER PRESENTERS

University of Toronto Department of Obstetrics and Gynaecology
Research Day, Friday, May 7, 2010

Thank you for submitting your abstract for Research Day. Your abstract has been chosen for a poster presentation. Please see the details below.

Location: Northrop Frye Hall at Victoria University in the University of Toronto. Northrop Frye Hall is located at 73 Queen's Park Crescent East, northeast of Queen's Park and east of Avenue Road, south of Charles St. and near the Museum subway station. You may use either the north or west entrance. A map is available on our website.

Time: Research Day will begin with breakfast at 8:00 a.m. on Friday, May 7, 2010 and end with a wine and cheese reception and award presentation from 5:25 to 6:30 p.m.

Registration: Please register on the ground floor level of Northrop Frye Hall between 8:00 and 8:25 a.m. to receive your nametag, abstract booklet and any further instructions.

Poster boards: We will provide each presenter with a 3' high by 6' wide board and velcro for attaching the poster. Boards will be numbered to correspond to the poster numbers published in the abstract book distributed at the meeting.

Set-up for Morning Poster Session: Between 7:30 and 9:45 a.m., Friday, May 7, 2010
Set-up for Afternoon Poster Session: Between 11:05 and 2:50 p.m.
Tear-down: Directly after your poster session.

Poster Session I: The morning session will start with a general walkabout from 9:45 to 10:05 a.m., followed by a Poster Tour from 10:05 to 11:05 a.m..
Poster Session II: The afternoon session will start with a general walkabout from 2:50-3:10 p.m., followed by a Poster Tour from 3:10-4:10 p.m.
Poster Tours: Each group will be led by two Chairs, one a basic scientist and the other a clinician. We ask that you be present the entire time and join the others in your group in the tour. Please be prepared to give a 3-5 minute presentation, with 5 minutes for discussion/questions. **Please note that strict adherence to timing is essential.**

Awards: In order to be eligible for an award, you must be a U of T trainee and present your own work. Your work and presentation will be judged by the Chairs of your poster tour and one other Judge for the JW Knox Ritchie Research Awards. There will be 5 awards, with a monetary component, based on level of training (Graduate Student, Resident, Clinical Fellow, Post-Doctoral Fellow, Medical Student) rather than type of presentation. Please see judging criteria on our website. These awards will be presented at the wine and cheese reception between 5:25 and 6:30 p.m.

***Please see http://www.obgyn.utoronto.ca/Research/ResearchDay.htm for information or contact Helen Robson at* helen.robson@utoronto.ca**

JW Knox Ritchie Research Award Criteria

Principles:

1. Only a trainee (clinical fellow, graduate student, post-doctoral fellow, medical student or resident affiliated with the University of Toronto Department of Obstetrics & Gynaecology) is eligible for an award.
2. The submitter must be a trainee at the time of submission and at Research Day.
3. If the trainee is in more than one category, i.e. resident and graduate student, both those categories should be noted in the submission.
4. Each trainee may submit only **one** abstract.
5. Work-in-progress is welcomed and **is eligible** for an award.
6. Trainees are required to be present during the poster session for the entire period.
7. The trainee is eligible for an award only if he/she presents his/her own work.
8. Those in the **Other** category are not eligible for an award, but are marked for feedback purposes.
9. There will be 5 awards, with a monetary component, based on level of training (Graduate Student, Resident, Clinical Fellow, Post-Doctoral Fellow, Medical Student), rather than type of presentation.
10. Oral = Poster for evaluation purposes.
11. Each presentation is judged by at least 2 people, one or two chairs and one or two judges.
12. Any judge with a conflict in his/her session will cede to an alternate judge for marking.

Judging Criteria:

The following information will be provided on the judging form. Each criterion is marked from 1-10 with 1 being the *lowest* and 10 being the *highest* for a maximum total of 50 marks.

Originality = New topic or novel approach? (**Mark out of 10)**

Scientific Merit = Is the topic important and worth studying? Is this a useful examination? (**Mark out of 10)**

Study Design and Analysis = Is the design and are tests appropriate and done properly? **(Mark out of 10)**

Interpretation and Conclusions = Does the candidate examine all data and inferences? Are there realistic and practical conclusions? **(Mark out of 10)**

Presentation = Is the presentation, oral or poster**,** well developed and explained? Is the poster easy to read? Is the candidate able to respond well to questions? **(Mark out of 10)**

Total = addition of criteria columns to a maximum of 50 marks