Hypertension and Beyond

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Hilton Toronto Airport
Hotel and Suites
Toronto, ON

Congrès Hypertension Canada

Canadian Hypertension Congress

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PROGRAM

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GENERAL INFORMATION

CONGRESS CHAIRS' WELCOME
MOT DE BIENVENUE DU PRÉSIDENT DU CONGRÈS

Welcome to the fifth annual Canadian Hypertension Congress!

This congress is Canada's only national conference focused solely on hypertension and cardio-metabolic disease. You are among more than 300 of Canada's leading cardiologists, nephrologists, general practitioners, pharmacists, nurses, allied health professionals, population health experts and public health officials, converging to share and hear the latest research and thinking in hypertension. By its nature, the Congress represents our community's pursuit of excellence.

It is in this spirit that our conference begins, with a provocative opening panel titled "*Evidence and Influence – What role do clinical practice guidelines play in everyday practice?*" With this bold topic, addressed on the evening following our clinical practice guidelines consensus conference, we aim to draw out varying perspectives and experience, and challenge ourselves to continually learn and improve. We hope you'll join us to add to the discussion.

Over the following two days, through presentations, debate, and discussions, we address known gaps head on and inspire innovation through open exchange. The Canadian Hypertension Congress is filled with presentations of the latest research and advances in hypertension, provided in three programming streams to meet your educational needs. We are pleased to offer for the second year a defined stream in Primary Care – and extend a warm welcome to family physicians from the Greater Toronto Area.

In our pursuit of excellence, our community is known also for challenging the status quo. We are very pleased that Dr. Norm Campbell, the HSF-CIHR Chair in Hypertension Prevention and Control, has chosen our Congress for the release of the "Hypertension Framework" progress update. Collaboratively created in 2011, the Hypertension Framework called for extensive collaboration among decision-makers in governments, corporations, and health care to meet prevention and control goals and markers. We look forward to the insights the progress report will bring.

And, we look forward to seeing and hearing from you. From all of our Planning Committee, welcome – and enjoy!

Bienvenue au Congrès Hypertension Canada, organisé pour une cinquième année consécutive!

Le Congrès est la seule réunion nationale, tenue au Canada, qui porte uniquement sur l'hypertension et le syndrome métabolique. Vous êtes plus de 300 participants, parmi les meilleurs cardiologues, néphrologues, omnipraticiens, pharmaciens, infirmières et infirmiers, professionnels paramédicaux, experts en santé de la population et responsables de la santé publique au pays, à venir prendre connaissance des résultats les plus récents de la recherche sur l'hypertension et réfléchir sur divers aspects de la maladie. Le Congrès, de par sa nature, représente bien la poursuite de l'excellence par notre communauté.

C'est dans cet esprit que sera donné le coup d'envoi du Congrès; d'ailleurs, la table ronde inaugurale au titre provocateur, *Evidence and Influence – What role do clinical practice guidelines play in every day practice?*, donne le ton. Par ce débat sur un sujet si audacieux, qui aura lieu le soir suivant la conférence consensuelle sur les lignes directrices de pratique clinique, nous voulons faire ressortir divers points de vue et expériences, et en même temps nous mettre au défi de toujours apprendre et de toujours nous améliorer. Aussi espérons-nous que vous serez des nôtres pour participer au débat.

Au cours des deux journées suivantes, nous attaquerons de front des lacunes connues et susciterons de l'intérêt pour l'innovation dans des échanges directs de points de vue, dans le cadre de présentations, de débats et de discussions. Le Congrès Hypertension Canada offre un programme varié, composé de présentations qui font état des tout derniers résultats de la recherche sur l'hypertension et des progrès réalisés en la matière, selon trois parcours conçus pour répondre à vos besoins de formation. Nous avons également le plaisir d'offrir, pour la deuxième année, un parcours particulier en soins primaires. Nous profitons de l'occasion pour accueillir chaleureusement les médecins de famille de la région du Grand Toronto.

Dans sa quête de l'excellence, notre communauté est également connue pour ne pas se reposer sur ses lauriers. Nous avons donc le plaisir d'annoncer que le Dr Norm Campbell, président de la Chaire de la Fondation des maladies du cœur du Canada et des Instituts de recherche en santé du Canada sur la prévention et le contrôle de l'hypertension artérielle, a choisi le Congrès pour dévoiler la mise à jour du Cadre de lutte contre l'hypertension. Les auteurs du document sur le Cadre, fruit d'un effort collectif réalisé en 2011, plaident en faveur d'une collaboration élargie entre décisionnaires au sein des gouvernements ou d'entreprises et intervenants en soins de santé afin de rendre possible l'atteinte des objectifs fixés et des jalons posés en matière de prévention et de maîtrise de la pression artérielle. Nous avons bien hâte de connaître les observations qui se dégageront du rapport d'étape.

Au plaisir de vous voir et d'avoir de vos nouvelles! Au nom du comité de planification, je vous souhaite la bienvenue et profitez du Congrès.

Rob Gros (left) and Ross Feldman

Ross Feldman, MD and Robert Gros, PhD
Congress Co-Chairs
2015 Canadian Hypertension Congress
Hypertension Canada

PRESIDENT'S WELCOME
MOT DE BIENVENUE DE LA PRÉSIDENTE

It is my pleasure to welcome all 2015 Canadian Hypertension Congress attendees - members, volunteers, scientists and clinicians from across disciplines, sponsors and others - and extend our sincere thanks for your support of this Congress and of Hypertension Canada.

Canada is fortunate to lead the world in hypertension control rates, an accomplishment often credited to Hypertension Canada's CHEP clinical practice guidelines for the diagnosis, treatment, and control of hypertension. Our hypertension community powers these guidelines. Hundreds of scientists, clinicians and health care professionals dedicate over 15,000 hours each to their annual review, advancement and implementation. We are most grateful for their generosity.

We pursue our mission in Canada by fostering new research careers, developing the CHEP Guidelines, and helping professionals who treat hypertension to put them into practice. We are also dedicated to improving the environment for Canadians with hypertension and those who treat them. We strive to raise awareness and collaborate with governments, public policy makers and peer organizations to create environments that enable and encourage the best possible choices for Canadians and their health.

Collaboration is our core strength - collaboration among dedicated professionals who cross disciplines, sectors, and also international borders. As world leaders in hypertension control rates, Canadians are conscious global citizens and Hypertension Canada members are sought out by other nations as they prepare to address the globe's number one risk for death and disability. In the last year, we worked with groups in Saudi Arabia and in the United Kingdom to lay the foundations of primary health care education and of broad plans to improve control rates in their own nations. At this Congress, we are pleased to welcome international delegates to our community, and wish them a most enriching experience.

On behalf of the Board of Directors of Hypertension Canada, welcome to the Canadian Hypertension Congress, and thank you for being part of our community.

J'ai le plaisir de souhaiter la bienvenue à vous tous ici présents au Congrès Hypertension Canada de 2015 : membres, bénévoles, scientifiques et cliniciens de toutes disciplines, commanditaires et autres intervenants, et de vous remercier sincèrement de votre appui au Congrès et à Hypertension Canada.

Nous avons la chance, au Canada, d'avoir les meilleurs taux de maîtrise de l'hypertension artérielle dans le monde, une réussite que l'on attribue souvent aux recommandations du Programme éducatif canadien sur l'hypertension (PECH), rattaché à Hypertension Canada; ces recommandations sont le guide national de pratique clinique concernant le diagnostic, le traitement et la maîtrise de l'hypertension. Et c'est notre communauté vouée à l'hypertension qui « alimente » ces lignes directrices. Des centaines de scientifiques, de cliniciens et de professionnels de la santé consacrent plus de 15 000 heures chacun à leur examen annuel, à leur mise à jour et à leur mise en œuvre. Aussi méritent-ils tous notre reconnaissance pour leur générosité.

Nous remplissons notre mission au Canada en suscitant de nouvelles carrières de chercheur, en élaborant les lignes directrices du PECH et en aidant les professionnels de la santé qui traitent l'hypertension à les mettre en pratique. Nous sommes également déterminés à améliorer l'environnement dans lequel vivent les personnes atteintes d'hypertension et les professionnels qui les traitent. En effet, nous nous efforçons de sensibiliser les gouvernements, les décisionnaires publics et des organisations du domaine, et de collaborer avec eux afin de créer un environnement qui sensibilise les Canadiens et Canadiennes aux meilleurs choix possible pour eux-mêmes et pour leur santé, et qui leur donne les moyens de le faire.

La collaboration, voilà notre FORCE. En effet, des professionnels de la santé tout dévoués qu'ils sont franchissent les frontières entre disciplines, entre secteurs, et franchissent même les frontières internationales. En tant que chef de file mondial dans la maîtrise de l'hypertension, nous sommes une organisation citoyenne consciente, et des pays font appel à nos services pour les aider à faire face au principal risque d'incapacité et de mortalité dans le monde. Ainsi, l'année dernière, nous avons travaillé avec des groupes en Arabie saoudite et au Royaume-Uni pour jeter les bases de formation en soins de santé primaires et de plans généraux visant à améliorer les taux de maîtrise de l'hypertension dans ces pays. Nous avons donc le plaisir d'accueillir en notre sein, au Congrès, des représentants étrangers; nous leur souhaitons une expérience des plus enrichissantes.

Au nom du conseil d'administration d'Hypertension Canada, je vous souhaite la bienvenue au Congrès Hypertension Canada, et vous remercie de faire partie de notre communauté.

Ernesto L Schiffrin, C.M., MD, PhD
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Hypertension CANADA

WHO WE ARE

MISSION: Advancing health through the prevention and control of high blood pressure and its complications.

Hypertension Canada is Canada's only national non-profit organization dedicated solely to the prevention and control of hypertension and its complications. Powered by a professional volunteer network of the nation's leading multidisciplinary experts, Hypertension Canada pursues its mission through research, professional and public education, and advocacy for healthy environments.

WHAT WE DO

INNOVATE

Hypertension Canada aims to enhance the quantity and quality of hypertension and vascular biology research in Canada by attracting, supporting and mentoring new researchers to the field. We support current and future investigators through investment partnerships with the Canadian Institutes of Health Research (CIHR), we foster career mentoring and succession planning, and we collaboratively set hypertension research priorities in partnership with clinicians and patients.

EDUCATE

Hypertension Canada creates and publishes the world-renown CHEP clinical practice guidelines for hypertension diagnosis, treatment, and control, and supports health care professionals in their application. The CHEP Guidelines are developed by a multi-disciplinary task force and are rigorously reviewed and annually updated. We provide a suite of accredited educational programs for health care professionals based on the CHEP Guidelines, and create educational resources for professionals to use with their patients.

INFLUENCE

Hypertension Canada strives to influence the external environment to implement policies and strategies to improve awareness, prevention, and treatment of hypertension and its complications through the engagement of stakeholders and governments. Working with the Heart & Stroke Foundation-Canadian Institutes of Health Research Chair (HSFC/CIHR) in Hypertension Prevention and Control, and as a conscious organizational citizen of the health care community, we keep abreast of issues that need our attention, and opportunities to make positive changes that bring us closer to achieving our mission.

ENGAGE

Hypertension Canada's membership works with stakeholders and governments, to improve awareness, prevention, and treatment of hypertension and its complications. We make it our business to keep abreast of issues affecting people with hypertension and the professionals who treat them, and drive innovation in public policy.

These services measurably improve health outcomes, toward our vision:

Canadians will have the healthiest and best managed blood pressure in the world.

We are the health care professional's community for excellence in hypertension research, education and care.
Learn more at **www.hypertension.ca**.

À PROPOS D'HYPERTENSION CANADA

SA MISSION: Promouvoir la santé par la prévention et le contrôle de l'hypertension artérielle et de ses complications

Hypertension Canada est la seule organisation nationale, sans but lucratif, vouée uniquement à la prévention et à la maîtrise de l'hypertension artérielle et de ses complications. Animée par la force d'un réseau de professionnels bénévoles, eux-mêmes éminents experts dans diverses disciplines au pays, Hypertension Canada remplit sa mission par la recherche, la formation du public et des professionnels de la santé ainsi que la promotion d'environnements favorables à la santé.

SES ACTIVITÉS

L'INNOVATION

Hypertension Canada vise à accroître la recherche sur l'hypertension artérielle et en biologie vasculaire au Canada, tant en quantité qu'en qualité, en attirant de nouveaux chercheurs dans le domaine et en leur apportant soutien et mentorat. L'organisation offre son appui aux chercheurs, présents et futurs, par l'établissement de partenariats d'investissement avec les Instituts de recherche en santé du Canada; favorise le mentorat professionnel et la planification de la relève; et établit les priorités de recherche sur l'hypertension en collaboration avec les cliniciens et les patients.

LA FORMATION

Hypertension Canada élabore et publie les guides de pratique clinique du Programme éducatif canadien sur l'hypertension (PECH), de renommée internationale, sur le diagnostic, le traitement et la maîtrise de l'hypertension artérielle, et aide les professionnels de la santé à les mettre en pratique. Les lignes directrices du PECH sont élaborées par un groupe de travail pluridisciplinaire, et font l'objet d'un examen rigoureux et d'une mise à jour annuelle. Enfin, l'organisation offre divers programmes de formation reconnue, fondés sur les lignes directrices du PECH, à l'intention des professionnels de la santé, et élabore du matériel didactique à utiliser par les professionnels de la santé à l'intention des patients.

L'INFLUENCE

Hypertension Canada travaille en collaboration avec divers intervenants et les gouvernements, et a établi un partenariat avec la Chaire de la Fondation des maladies du cœur du Canada et des Instituts de recherche en santé du Canada sur la prévention et le contrôle de l'hypertension artérielle dans le but d'améliorer la prévention et le traitement de l'hypertension et de ses complications, et de sensibiliser le public à la maladie. L'organisation reste à l'écoute des personnes hypertendues touchées par différents problèmes et des professionnels qui les traitent, et elle est à l'affût des possibilités prometteuses, qui la rapprochent encore davantage de sa mission.

LA MOBILISATION

Hypertension Canada prend au sérieux la place et le rôle de premier plan que lui confère son titre de seule organisation sans but lucratif au Canada, vouée au principal risque d'incapacité et de mortalité dans le monde : l'hypertension artérielle. Elle répond aux besoins de son vaste réseau de professionnels bénévoles, de membres dévoués et de partenaires de collaboration en tant que ressource la plus sûre et la plus digne de confiance en matière de prévention, de diagnostic, de traitement et de maîtrise de l'hypertension. L'organisation réagit rapidement aux nouveaux problèmes, et communique de l'information par l'intermédiaire du bulletin eINFO, du site Web, des médias sociaux et du Congrès Hypertension Canada. Elle cherche aussi à recueillir différents points de vue et à attirer des professionnels et des partenaires de divers horizons afin de cerner les démarches les plus efficaces lui permettant d'accomplir son travail, et ce, dans un esprit de collaboration. C'est ainsi, grâce à l'effort collectif, qu'Hypertension Canada fait la promotion de la santé par la prévention et la maîtrise de l'hypertension artérielle et de ses complications.

Les services offerts permettent d'améliorer, de manière mesurable, les résultats cliniques, et rapprochent ainsi l'organisation de sa vision.

Que les Canadiens aient la meilleure pression artérielle et la meilleure prise en charge au monde!

Hypertension Canada forme une communauté de professionnels de la santé en quête d'excellence dans les domaines de la recherche, de la formation et des soins relatifs à l'hypertension. Pour en apprendre davantage sur l'organisation, rendez-vous au **www.hypertension.ca**.

LEARNING OBJECTIVES AND ACCREDITATION

LEARNING OBJECTIVES

The purpose of the Canadian Hypertension Congress (CHC) is to encourage and promote the development, advancement and exchange of balanced and evidence-based information regarding the research, diagnosis and treatment of hypertension and related cardiovascular diseases towards the ultimate goal of improved patient care and health.

The scientific sessions are designed to:

- Present and examine new findings on the physiology, pathophysiology, epidemiology, diagnosis and management of hypertension, atherosclerosis and related diseases;
- Review current state-of-the-art advances in managing particular groups of patients; and
- Evaluate specific treatment modalities and pharmacological agents.

By attending the Canadian Hypertension Congress, participants will be able to:

- Familiarize themselves with exemplary collaborative clinical and research initiatives in the area of cardiovascular disease prevention;
- Integrate their knowledge of information derived from current Canadian cardiovascular research into their practice; and
- Foster discussion and debates that encourage innovation in cardiovascular health and research.

ACCREDITATION

This conference has been organized in collaboration with Schulich School of Medicine & Dentistry, Western University. This event is an Accredited Group Learning Activity (Section 1) as defined by the Maintenance of Certification program of The Royal College of Physicians and Surgeons of Canada and approved by Continuing Professional Development, Schulich School of Medicine & Dentistry, Western University (11.0 hours).

This program meets the accreditation criteria of The College of Family Physicians of Canada and has been accredited by Continuing Professional Development, Schulich School of Medicine & Dentistry, Western University, for up to 11.0 Mainpro-M1 credits.

Each participant should claim only those hours of credit that he/she actually spent participating in the educational program.

Schulich MEDICINE & DENTISTRY Western

CONFLICT OF INTEREST DISCLOSURE

Hypertension Canada strives to ensure balance, independence, objectivity and scientific rigor in all of its educational programs. All faculty members participating in this program have been required to disclose any real or apparent conflict(s) of interest that may have a direct bearing on the subject matter of the session in which they are participating. This includes relationships in place at the time of the meeting or in the twelve (12) months preceding the meeting, with pharmaceutical companies, biomedical device manufacturers, or other corporations whose products or services are related to the subject matter of the presentation topic. The intent of the policy is to identify openly any conflict of interest so that the listeners may form their own judgments about the presentations with the full disclosure of the facts.

NOTES

Congrès Hypertension Canada

Canadian Hypertension Congress

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SPEAKERS

SPEAKERS

THANK YOU TO OUR 2015 SPEAKERS

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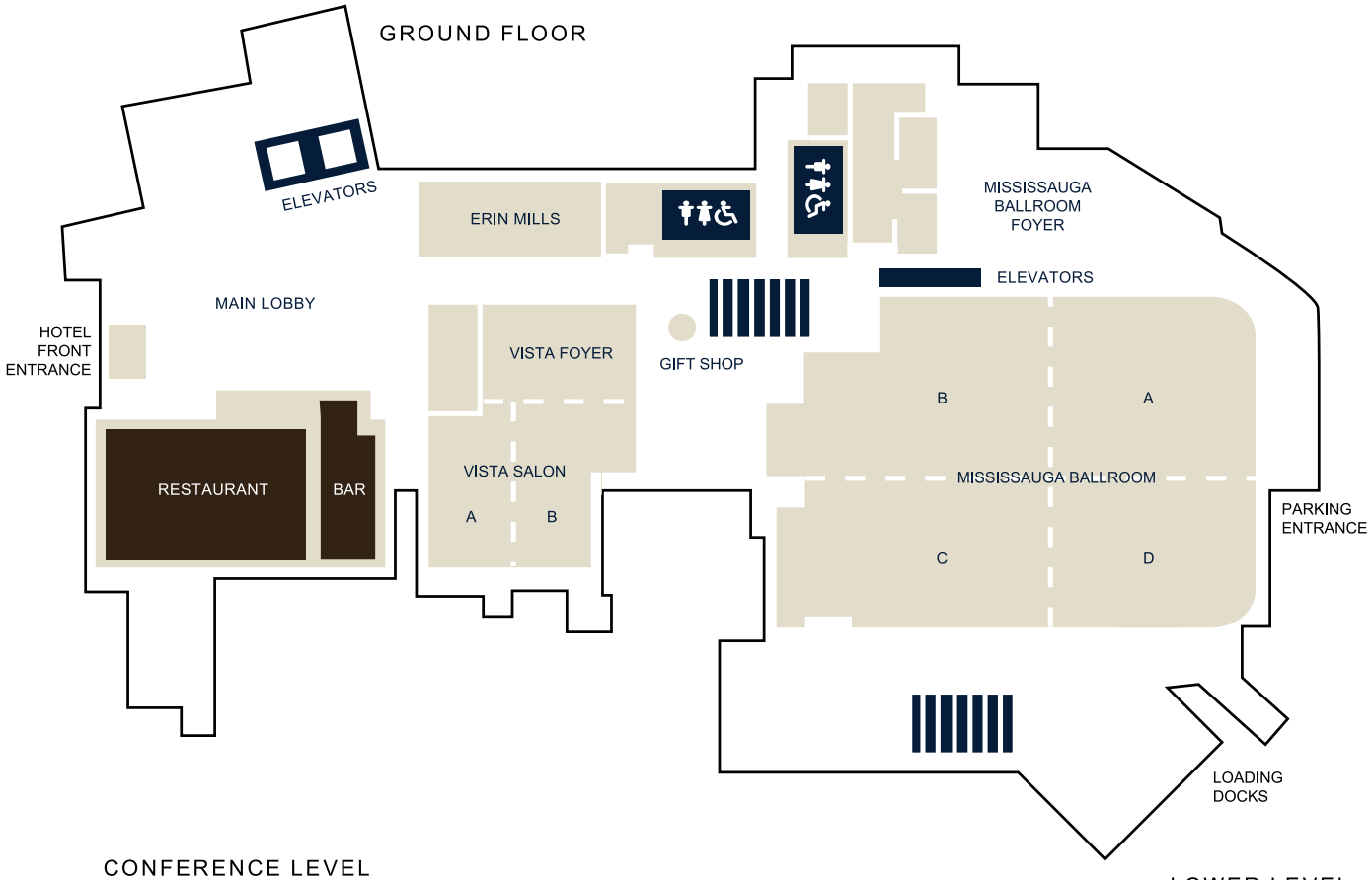
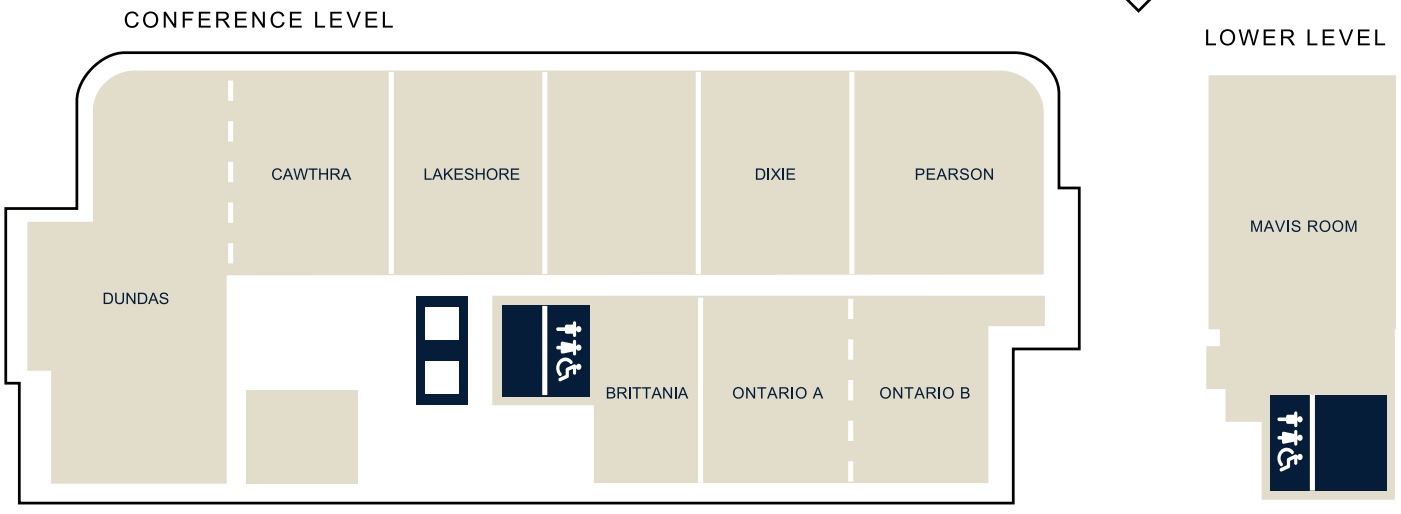
FLOOR PLAN





KEY

- Food & Beverages
- Meeting/Conference Rooms

Congrès Hypertension Canada

Canadian Hypertension Congress

Hypertension CANADA

SCIENTIFIC PROGRAM

SCIENTIFIC PROGRAM

THURSDAY, OCTOBER 22, 2015

18:00 - 18:10	**OPENING REMARKS** *Dr. Ross Feldman, Canadian Hypertension Congress Co-chair* Room: Mississauga B&C
18:10 - 19:00	**OPENING PANEL DISCUSSION** **Evidence and Influence - What role do clinical practice guidelines play in everyday practice?** *Moderator: Tom Blackwell* **CLOSING REMARKS** *Dr. Ernesto Schiffrin, President and Chair of the Board, Hypertension Canada* Room: Mississauga B&C
19:00 - 20:30	**POSTER SESSION # 1** Mississauga Foyer **Biomedical Research Track** **BMR1** - **Proteomic analysis of microparticles derived from human endothelial cells and podocytes** - *Shareef Akbari* **BMR2** - **Novel inhibitory effects of an oxazol-5-one (DI) compound on Rho-associated kinase II functions in human coronary artery smooth muscle cells** - *Abdulhameed Al-Ghabkari* **BMR3** - **Gamma/Delta T cells mediate angiotensin II-induced hypertension and vascular injury** - *Antoine Caillon* **BMR4** - **Induction of human endothelin-1 overexpression for 3 months causes blood pressure rise and small artery endothelial dysfunction and stiffening** - *Suellen Coelho* **BMR5** - **Divergent roles of GPER vs. ER in estrogen-mediated regulation of LDL cholesterol metabolism** - *Qingming Ding* **BMR6** - **Sodium nitroprusside (SNP) attenuates hypertension in spontaneously hypertensive rats: role of Gi proteins and oxidative stress** - *Ekhtear Hossain* **BMR7** - **ER stress inhibition decreases hypertensive proteinuria in a CKD mouse model** - *Zahraa Mohammed-Ali* **BMR8** - **A primitive somite-derived stem cell in the adult mouse is capable of myelopoiesis** - *Sarah Steinbach* **BMR9** - **Doxycycline hyclate administration differentially affects vascular mineral accrual in CKD based on vessel anatomical location** - *Bruno Svajger* **BMR10** - **The relationship between fibroblast growth factor 23 and kidney function** - *Emilie Ward* **Clinical/Outcomes/Population Research Track** **CR1** - **Are home blood pressure devices accurate? A systematic review of the evidence** - *Eva Bruketa* **CR2** - **Implementation of Canada's sodium reduction strategy: Evaluation of actions, barriers and facilitators to reducing sodium in public institutions** - *Sharon Chandra* **CR3** - **Is dietary intake of calcium associated with surrogate markers of cardiovascular disease in healthy postmenopausal women?** - *Shubhabrata Das* **CR4** - **Hypertension screening, management and follow-up by community pharmacists** - *Shelley Diamond* **CR5** - **Attenuation of arterial stiffness gradient with age and its impact on central pulse wave profile in dialysis patients** - *Catherine Fortier* **CR6** - **Nutritional consequences and benefits associated with dietary sodium reduction in individuals** - *Katherine Jefferson* **CR7** - **The effect of vitamin D ergocalciferol supplements on mild and moderate high blood pressure** - *Samia Rizk* **CR8** - **The metabolic acidosis among patients with high blood pressure in the Great Lakes Region** - *Jean Safari* **CR10** - **Prevalence of hypertension and associated factors among residents of Ibadan-North local government area (LGA) of Oyo State, Nigeria** - *Ibukan Sowemimo* **CR11** - **Evaluation of the Ontario Stroke Network's Hypertension Management Program: A model for stroke prevention in primary care settings** - *Pauline Therrien* **CR12** - **Effect of a workplace organizational intervention targeting adverse psychosocial work factors on blood pressure and hypertension** - *Xavier Trudel*
19:00 - 20:30	**WELCOME RECEPTION IN MISSISSAUGA FOYER**

SCIENTIFIC PROGRAM

FRIDAY, OCTOBER 23, 2015

	Biomedical Research Track Room: Mississauga B	Clinical/Outcomes/Population Research Track Room: Mississauga C	Primary Care Track Room: Vista Salon
	Each 15 min session includes a 5 min Q & A Each 30 min session includes a 10 min Q & A	*Each 15 min session includes a 5 min Q & A Each 30 min session includes a 10 min Q & A*	*Each 15 min session includes a 5 min Q & A Each 30 min session includes a 10 min Q & A*
07:00 - 08:00	**BREAKFAST SYMPOSIUM IN MISSISSAUGA A&D** **IN MY DEFENCE: The Case of Mrs. S. Death by Myocardial Infarction. Was it Preventable?**		
08:00 - 09:30	**BIOMEDICAL # 1** *Co-chairs: Dr. Grant Pierce & Dr. Michael Adams* 08:00 **Modeling type II diabetes-associated vasculopathies with skin-derived precursors (SKPs)** *Sarah Steinbach* 08:15 **Role of potassium-chloride cotransporter type 3 in the cardiometabolic physiology in mice** *Alexandre P Garneau* 08:30 **A conserved microRNA cluster as a potential master regulator in angiotensin II-induced vascular damage** *Kugeng Huo* 08:45 **Pudendal artery structure and function as early markers for elevated pulse wave velocity** *Paul Jeronimo* 09:00 **STATE OF THE ART** **We are what we eat: Emerging food effects on BP or CV disease** *Dr. Grant Pierce*	**CLINICAL # 1** *Co-chairs: Dr. Sheldon Tobe & Dr. Donna McLean* 08:00 **STATE OF THE ART** **How to Manage the Patient with Renovascular Disease after the CORAL Study** *Dr. Sheldon Tobe* 08:30 **Longterm exercise decreases von Willebrand Factor antigen levels in abdominally obese adults** *Cynthia Pruss* 08:45 **Impact on blood pressure from an interactive mobile based self-management system in patients with advanced chronic kidney disease** *Stephanie Ong* 09:00 **Next-generation cloud-based blood pressure devices in chronic disease management: A direct intra-arterial pressure calibration of an oscillometric wrist cuff device for clinically reliable and accurate blood pressure measurements** *Keith R Brunt* 09:15 **Cardiovascular risk and prevention strategies at the Great Lakes Regional Hospital** *Jean Safari*	**PRIMARY CARE # 1** *Co-chairs: Dr. Raj Padwal & Dr. Tavis Campbell* 08:00 **BP Thresholds and Targets: Consensus and Controversy** *Dr. Raj Padwal* 08:30 **First Line Drug Management of Hypertension: Role of Single Pill Combination Therapy** *Dr. George Dresser* 09:00 **Nutritional Therapies for Prevention and Management of Hypertension and CV Disease** *Dr. Debra Reid*
09:30 - 10:00	**BREAK**		

SCIENTIFIC PROGRAM

FRIDAY, OCTOBER 23, 2015

	Biomedical Research Track Room: Mississauga B	Clinical/Outcomes/Population Research Track Room: Mississauga C	Primary Care Track Room: Vista Salon
10:00 - 11:30	**BIOMEDICAL # 2** *Co-chairs: Dr. Pierre Paradis & Dr. Jeffrey Dickhout* 10:00 **Oxidative stress contributes to the enhanced expression of Gqα and PLCβ1 proteins and hypertrophy of VSMC from SHR: role of growth factor receptor transactivation** *Mohammed Emehdi Atef* 10:15 **Effect of 4-phenylbutyric acid treatment on hypertension development and pre-hypertensive tachycardia in the SHR** *Safaa Naiel* 10:30 **Handle region peptide induces adipogenesis in subcutaneous adipose tissue to promote healthy fat distribution** *Paul Tan* 10:45 **Preferential disposition of radiolabeled phosphate to the vasculature modifies calcium incorporation in experimental chronic kidney disease** *Jason Zelt* 11:00 **STATE OF THE ART** **microRNAs and Hypertensive Vascular Disease: What will the future bring?** *Dr. Pierre Paradis*	**CLINICAL # 2** *Co-chairs: Dr. Paul Timothy Pollak & Dr. Doreen Rabi* 10:00 **STATE OF THE ART** **Reducing Cardiovascular Risk in Patients with Hypertension: Moving Beyond Traditional Models** *Dr. Finlay McAlister* 10:30 **Differences in 24-h ambulatory blood pressure (ABPM) responses between differing nifedipine osmotic delivery formulations coincide with differences in dissolution profiles** *Paul Timothy Pollak* 10:45 **Effect of the double exposure to adverse psychosocial work factors and high family responsibilities on blood pressure among white-collar working women: A 5-year prospective study** *Mahée Gilbert-Ouimet* 11:00 **Antihypertensive drug initiation versus chronic use and its impact on fracture risk in the elderly: A systematic review and meta-analysis** *Debra Butt* 11:15 **Socioeconomic status and longitudinal change in aortic stiffness: the Whitehall II study** *Xavier Trudel*	**PRIMARY CARE # 2** *Co-chairs: Dr. Mark Gelfer & Dr. George Dresser* 10:00 **BP Measurement and Hypertension Diagnosis in 2015: Which Methods, Which Numbers** *Dr. Mark Gelfer* 10:30 **Better Communication for Helping Patients Take Action and Adhere with Health Behaviour Change** *Dr. Tavis Campbell* 11:00 **Global CV Risk Assessment: Art and/or Science** *Dr. Guy Tremblay*
11:30 - 13:30	**LUNCH SYMPOSIUM IN MISSISSAUGA A&D** **Evolving Role of Single-Pill Combination in Hypertension**		

SCIENTIFIC PROGRAM

FRIDAY, OCTOBER 23, 2015

	Biomedical Research Track Room: Mississauga B	Clinical/Outcomes/Population Research Track Room: Mississauga C	Primary Care Track Room: Vista Salon
13:30 - 15:00	**A RENAISSANCE OF VASCULAR BIOLOGY IN CANADA** *Chair: Dr. Mansoor Husain* 13:30 **Macrophage Responses in Cardiovascular Health and Disease** *Dr. Clinton Robbins* 13:45 **Engineering Intravascular Materials Towards Solutions to Atherothrombosis and Hemorrhage** *Dr. Christian Kastrup* 14:00 **microRNA Regulation of Vascular Disease: Small RNA With a Big Impact** *Dr. Katey Rayner* 14:15 **What is Endothelial Heterogeneity and Why Should We Care?** *Dr. William C Aird* 14:30 **Endothelial Cell Function in Health and Disease: The Environment and Epigenetics** *Dr. Philip Marsden* 14:45 **Panel Discussion** *Moderator: Dr. Mansoor Husain*	**HSF/CIHR CHAIR OF HYPERTENSION PREVENTION AND CONTROL SYMPOSIUM** **The Public Health Dimensions of Hypertension Prevention, Treatment and Control.** *Chair: Dr. Norm Campbell* 13:30 **Hypertension Surveillance: What's happening?** *Dr. Raj Padwal* 13:40 **Economics of Hypertension Prevention and Control** *Dr. Fiona Clement* 13:50 **An Updated 2015 Hypertension Framework: What's needed?** *Dr. Norm Campbell* 14:00 **Working with Governments on Hypertension Prevention and Control** *Dr. Perry Kendall & Dr. David Mowat* 14:20 **Panel discussion and questions**	**PRIMARY CARE # 3** *Co-chairs: Dr. Ross Feldman & Dr. Guy Tremblay* 13:30 **Coronary Artery Disease in Women: Under Treatment or Overkill** *Dr. Ross Feldman* 14:00 **Why Obesity Is A Disease** *Dr. Arya Sharma* 14:30 **Smoking Cessation in Patients with Mental Illness and Addictions: Adapting Evidence Based Interventions in Clinical Practice** *Dr. Peter Selby*
15:00 - 15:30	**BREAK**		

SCIENTIFIC PROGRAM

FRIDAY, OCTOBER 23, 2015

	Biomedical Research Track Room: Mississauga B	Clinical/Outcomes/Population Research Track Room: Mississauga C	Primary Care Track Room: Vista Salon
15:00 - 17:00	**POSTER SESSION # 2** Mississauga Foyer **Biomedical Research Track** **BMR11** - **4-phenylbutyric acid inhibited salt sensitive hypertension in the model of Dalh salt sensitive rat** - *Chao Lu* **BMR12** - **Targeted deletion of matrix metalloproteinase 2 prevents angiotensin II-induced vascular injury, mediated in part by inhibition of epidermal growth factor receptor phosphorylation in vascular smooth muscle cells** - *Tlili Barhoumi* **BMR13** - **Plasma oxylipins as potential diagnostic markers and therapeutic targets for cardiovascular and cerebrovascular events in patients with peripheral arterial disease** - *Stephanie Caligiuri* **BMR14** - **The extent of vascular remodeling following injury is dependent on the balance between ERα and GPER** - *Qingming Ding* **BMR15** - **Therapeutically targeting the cystic fibrosis transmembrane regulator (CFTR) in cerebrovascular dysfunction associated with subarachnoid hemorrhage (SAH)** - *Jessica Fares* **BMR16** - **Endothelin-1 overexpression exaggerates type 1 diabetes-induced endothelial dysfunction by altering oxidative stress balance** - *Sofiane Ouerd* **BMR17** - **Heme oxygenase and pancreatic regeneration** - *Manish Mishra* **BMR18** - **Stromal interaction molecule-1 mediates angiotensin-II-induced expression of early growth response protein-1 in vascular smooth muscle cells** - *Estelle Rolande Simo Cheyou* **BMR19** - **Origins of adventitial Sca1+ progenitor cells** - *Sarah Steinbach* **BMR20** - **The contribution of organ fibrosis to hypertension through the unfolded protein response pathway** - *Victor Tat* **Clinical/Outcomes/Population Research Track** **CR13** - **The expanding role of Microbiota: from gastrointestinal disorders to blood pressure control** - *Tawfik Albassam* **CR15** - **The effect of short-versus long-acting antihypertensives on blood pressure variability** - *Jessica Gorgui* **CR16** - **Effect of antihypertensive medications on bone mineral density: a systematic review and meta-analysis** - *Jonathan Hwang* **CR17** - **Does the risk of falls depend on duration of antihypertensive drug use in the elderly? A systematic review and meta-analysis** - *Richard Leu* **CR18** - **Validity of health administrative database definitions for hypertension: A review** - *Romina Pace* **CR19** - **New hypertensives in a tertiary care hospital in Sri Lanka: The epidemiology** - *Kushalee Jayawickreme* **CR20** - **ASA, Aspirin use to prevent cardiovascular and cerebrovascular events** - *Samia Rizk* **CR21** - **Epidemiological profile of hypertension at the Great Lakes Regional Hospital** - *Jean Safari* **CR22** - **Cigarette smoking as a risk factor for masked hypertension: a systematic review** - *Benjamin Sehmer*		15:30 - 17:00 **PRIMARY CARE # 4** *Co-chairs: Dr. Debra Reid & Dr. Irene Hramiak* 15:30 **New Treatment Approaches in Your Patient with Type 2 Diabetes** *Dr. Irene Hramiak* 16:00 **Dyslipidemia Update: Target 2016** *Dr. Robert Hegele* 16:30 **Management of Atrial Fibrillation for the Primary Care Setting** *Dr. Allan Skanes*
17:00 - 19:00	**DINNER IN MISSISSAUGA A&D**		

SCIENTIFIC PROGRAM

SATURDAY, OCTOBER 24, 2015

	Biomedical Research Track Room: Mississauga B	Clinical/Outcomes/Population Research Track Room: Mississauga C	Primary Care Track Room: Vista Salon
07:00 - 08:00	**BREAKFAST OF CHAMPIONS IN MISSISSAUGA A&D**		
08:00 - 09:00	**HYPERTENSION CANADA'S ANNUAL GENERAL MEETING IN VISTA SALON**		
09:00 - 10:30	**BIOMEDICAL # 3** *Co-chairs: Dr. Dylan Burger & Dr. Julie Lavoie* 09:00 **STATE OF THE ART** **Effect of Exercise Training on Preeclampsia** *Dr. Julie Lavoie* 09:30 **Particular plasma oxylipins increase the odds of high central blood pressure and are beneficially influenced by dietary flaxseed in patients with peripheral arterial disease** *Stephanie Caligiuri* 09:45 **Estrogenic neurons in the medial amygdala prevent stress-induced hypertension** *Antentor Hinton, Jr.* 10:00 **Endothelin-1 overexpression preserves endothelial function in mice with vascular smooth muscle cell-restricted PPARγ knockout** *Sofiane Ouerd* 10:15 **Effects of acute IL-17a exposure on cerebrovascular function** *Amy Randell*	**CLINICAL # 3** *Co-chairs: Dr. Alexander Logan & Robin Walker* 09:00 **Development and piloting of a curriculum for training non-physician health workers to assess and manage hypertension in low- and middle-income areas** *Jared Paty* 09:15 **A systematic review and meta-analysis of blood pressure measurement techniques in children** *Stephanie Duncombe* 09:30 **Case series of sequential physiologic changes in macular thickness in pregnancy: Observations of normal and disease specific patterns, mechanisms of response, and clinical interpretation** *Robert J Herman* 09:45 **Reliability of physician ratings for avoidable hospitalization for patients with uncomplicated hypertension, an ambulatory care sensitive condition** *Robin Walker* 10:00 **STATE OF THE ART** **Home Blood Pressure: The Role of Telemonitoring and Other Technologies** *Dr. Alexander Logan*	
10:30 - 11:00	**BREAK**		

SCIENTIFIC PROGRAM

SATURDAY, OCTOBER 24, 2015

	Biomedical Research Track Room: Mississauga B	Clinical/Outcomes/Population Research Track Room: Mississauga C	Primary Care Track Room: Vista Salon
11:00 - 12:30	**BIOMEDICAL # 4** *Co-chairs: Dr. Noriko Daneshtalab & Dr. Robert Gros* 11:00 **A model of mono-arthritis and cerebrovascular dysfunction** *Norika Daneshtalab* 11:15 **Altered vessel hemodynamics at rest and after acute physical stress in young smokers** *Alexander Cooke* 11:30 **Dysfunctional insulin signaling compromises cardiac function in obese Zucker Fatty rats** *Shuchita Tiwari* 11:45 **Iohexol plasma clearance in rat models: the clear choice for measuring early renal dysfunction** *Mandy Turner* 12:00 **STATE OF THE ART** **The Role of the Non-neuronal Cholinergic System in Regulating Cardiac Function** *Dr. Robert Gros*	**2016 DRAFT CHEP RECOMMENDATIONS SYMPOSIUM** *Chair: Dr. Raj Padwal* 11:00 **Opening Remarks** *Dr. Raj Padwal* 11:05 **Report on the Outcomes Research Task Force Initiatives** *Dr. Raj Padwal* 11:15 **eLearning Initiatives** *Dr. Guy Tremblay & Dr. Denis Drouin* 11:25 **New Evidence and the 2016 Draft CHEP Recommendations** *Dr. Doreen Rabi* 12:25 **Q&A and Closing Comments** *Dr. Raj Padwal*	
12:30 - 14:00	**LUNCH IN MISSISSAUGA A&D**		

SCIENTIFIC PROGRAM

SATURDAY, OCTOBER 24, 2015

14:00 - 15:30	**HYPERTENSION CANADA AWARDS LECTURE & AWARD PRESENTATIONS** *Chair: Dr. Ernesto Schiffrin* 14:00 **SENIOR INVESTIGATOR AWARD LECTURE** **From Biology to Breakthroughs for the Other Kind of Hypertension-Pulmonary** *Dr. Duncan Stewart* 14:30 **VANCOUVER 2010 LECTURE** **Role of Novel PPARγ Pathways in the Control of Vascular Function and Arterial Pressure** *Dr. Curt Sigmund* 15:00 **World Hypertension League Awards** 15:10 **Hypertension Canada's Recognition Awards** 15:20 **Hypertension Canada Trainee Presentation Awards** Room: Mississauga C

NOTES

Congrès Hypertension Canada

Canadian Hypertension Congress

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AWARD RECIPIENTS

AWARD RECIPIENTS

2015 DISTINGUISHED SERVICE AWARD

Martin G Myers MD, FRCPC
Sunnybrook Health Sciences Centre
Toronto, Ontario

Dr. Myers, for his more than 35 years of continuous voluntary service and leadership to hypertension organizations at the local, regional and national levels, is the 2015 Distinguished Service Award recipient. Dr. Myers was instrumental in the development of the Toronto Hypertension Society, founded the Ontario Hypertension Society, and is credited in part with conceiving the original idea for the Canadian Hypertension Society. He served on its first Board of Directors and continued to be a Director for the next ten years. In addition to Dr. Myers' governance roles, he was dedicated to ensuring scientific content of the highest quality for the Society's meetings.

Dr. Myers continues to serve as the Chairman of Hypertension Canada's CHEP Guidelines Automated Blood Pressure Devices Sub-Committee, a role he has held since 2006. In addition, his clinical research studies involving automated measurement of blood pressure in the office setting has led to findings that have supported important changes to Hypertension Canada's CHEP Guidelines. Automated office blood pressure measurement is now recommended for assessing a patient's blood pressure in order to obtain a more accurate reading that eliminates the "white coat effect," in which blood pressure is abnormally elevated when measured in a clinical setting. Dr. Myers' research has the potential to make healthcare more effective and efficient throughout the world.

Dr. Myers' work exemplifies the essence of Hypertension Canada's Distinguished Service Award.

2015 SENIOR INVESTIGATOR AWARD

Duncan J Stewart, MD, FRCPC
Ottawa Hospital Research Institute
Sprott Centre for Stem Cell Research
Ottawa, Ontario

The 2015 Senior Investigator Award recognizes Dr. Stewart's unique, dynamic and extensive background as a pioneering Canadian cardiovascular researcher. He has made many important discoveries in blood vessel biology and is dedicated to translating these discoveries into benefits for patients and society. He is known for his leadership of the Ottawa Hospital Research Institute (OHRI), his more than 200 published peer-reviewed manuscripts and his numerous distinctions and prizes; including the Dexter Man Chair of Cardiology and Research Achievement Award of the University of Toronto, and the Research Achievement Award of the Canadian Cardiovascular Society.

Throughout Dr. Stewart's career, he has demonstrated leadership by bringing diverse groups of clinicians and scientists together, putting Canada at the forefront of global translational cardiovascular and regenerative medicine research. He is deeply committed to the collective action that is needed to reduce the growing burden of vascular disease and its impact on Canadians. These efforts are guided by his tireless desire to promote and ultimately improve patient outcomes.

Dr. Stewart is an outstanding scientist, leader and advocate and very deserving of this recognition.

AWARD RECIPIENTS

2015 CERTIFICATES OF EXCELLENCE

Awarded in recognition of outstanding efforts and contributions in Canada, to increase public awareness, prevention and control of hypertension

Peter Bolli, MD, MACP, FRCP (Glasg.), FRCP (Edin.), FAHA
Ambulatory Internal Medicine Teaching Clinic
St. Catharines, Ontario

Dr. Bolli's substantial contributions from his long-standing career as a physician, professor and researcher in the field of hypertension are recognized by the 2015 Certificate of Excellence. He has produced close to 200 publications; has been awarded several prizes for his clinic and his achievements; including co-recipient of the first prize for Internal Medicine of the Swiss Society of Internal Medicine and Laureate Award, from the American College of Physicians and is an honourary member of five foundations and societies linked to hypertension.

Dr. Bolli has been an excellent ambassador of Hypertension Canada and its CHEP Guidelines by presenting it on several occasions to primary care physicians, promoting it at numerous national and international hypertension meetings and being an outstanding mentor to new Hypertension Canada members.

Dr. Bolli's exceptional commitment to excellence in the care of people living with hypertension spans over five decades and has helped to improve health outcomes for patients across Canada.

Tara Duhaney, MHSc
Hypertension Advisory Committee
Calgary, Alberta

The 2015 Certificate of Excellence recognizes Ms. Duhaney's substantial contributions in the field of hypertension through her role as the Policy Director for the Canadian Hypertension Advisory Committee. She has researched healthy public policies, developed healthy public food policy position statements for support of national organizations and aided the knowledge translation efforts on the policies.

Ms. Duhaney's work on a policy statement restricting the marketing of unhealthy food to children received over 24 national organization endorsements. Newer policy statements on healthy food procurement and an overarching policy statement calling for the implementation of a healthy food policy agenda by Canada's federal government have also received considerable support. Ms. Duhaney also aided the uptake of policy statements by corresponding with key political decision-makers at the federal and provincial levels, assisting with the development and updating of the HypertensionTalk.com website and writing summaries of the policies and related activities for Canadian Hypertension Advisory Committee member organizations.

Ms. Duhaney has demonstrated an exceptional commitment and outstanding contributions to the field of hypertension prevention and control.

AWARD RECIPIENTS

2015 CERTIFICATES OF EXCELLENCE

Awarded in recognition of outstanding efforts and contributions in Canada, to increase public awareness, prevention and control of hypertension

Robert Gros, PhD, FAHA
Robarts Research Institute
London, Ontario

The 2015 Certificate of Excellence recognizes Dr. Gros' substantial contributions in the field of hypertension and specifically his development of the Canadian Hypertension Congress. His prolific research career includes over 80 peer-reviewed publications, a long track record of success in securing funding, and mentoring many trainees who have developed into successful junior investigators: a testament to his abilities as a teacher and mentor.

In his position as chair of the Scientific Program Committee for the Canadian Hypertension Congress, he has made significant contributions to the development of the Congress program every year, serving to guide professional practice and research in Canada and around the world.

Dr. Gros' outstanding achievements have contributed to the field of hypertension by promoting interest in the study of its causes and treatments.

Jean L Rouleau, MD, FRCPC
Montréal Heart Institute
Montréal, Québec

The 2015 Certificate of Excellence recognizes Dr. Rouleau's substantial contributions as the Scientific Director of ICRH and his establishment of the Vascular Network. Dr. Rouleau's distinguished career as a cardiologist, scientist and administrator led to his recent role as Dean of the Faculty of Medicine, Université de Montréal and now as Scientific Director of the ICRH.

The formation of the Vascular Network, a key instrument in better integrating the efforts of vascular scientists nationally, was conceived and shepherded by Dr. Rouleau. This Network is a textbook example of forging win-win partnerships, the likeness of which has characterised his term as Scientific Director.

Dr. Rouleau's exceptional commitment and trailblazing efforts have contributed greatly to the advancement of hypertension research and the strength of the hypertension community in Canada.

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ABSTRACT GUIDE

BIOMEDICAL POSTER SESSION #1: 19:00 - 20:30 MISSISSAUGA FOYER

BMR1 - Proteomic analysis of microparticles derived from human endothelial cells and podocytes

Mercedes Munkonda- University of Ottawa, Ottawa Hospital Research Institute, Maddison Turner- University of Ottawa, Ottawa Hospital Research Institute, Dylan Burger- University of Ottawa, Ottawa Hospital Research Institute

Background: Microparticles (MPs) are small (0.1-1.0 µm), membranous vesicles shed from the cell surface following stress/injury. Our laboratory and others have shown that endothelial cell (EC) MPs are increased in hypertension and diabetes and exert biological effects (i.e. oxidative stress, inflammation) on target cells through paracrine signaling. More recently, we reported that MPs are produced by human podocytes (hPOD) and their levels in urine reflect glomerular injury. **Methods:** hPOD MPs and EC MPs were isolated and lysates were subjected to polyacrylamide gel electrophoresis. Proteins were visualized by silver staining and gel lanes were cut into 10 x 4 mm bands. Proteins were digested in-gel using trypsin and analyzed by liquid chromatography-tandem mass spectrometry. The observed MS/MS spectra were matched against human sequences from SwissProt. **Results:** A total number of 392 proteins were identified with 35 proteins found to be common to both MP populations with 251 proteins unique to EC MPs and 106 proteins unique to hPOD MPs. Proteins common to both MP populations included membrane-associated proteins such as Annexin A2, Annexin A5, Na+/K+ATPase, and Rab11. 251 proteins were identified as unique to EC MPs and included markers of endothelial cell injury/activation such as platelet endothelial cell adhesion molecule, Von Willebrand factor, and plasminogen activator inhibitor 1. 106 proteins were identified as unique to hPOD MPs including markers of podocyte differentiation (CDH13), renal injury (GDF15), as well as proteins critical to podocyte foot process assembly (integrins beta-1 and alpha 3). **Conclusion:** In summary, we have identified several proteins common to MPs from multiple origins as well as proteins unique to EC MPs and hPOD MPs. Analysis of such proteins may provide novel insights into the health status of the cells from which they originate, as well as the mechanisms regulating MP production and signaling.

BIOMEDICAL POSTER SESSION #1: 19:00 - 20:30 MISSISSAUGA FOYER

BMR2 - Novel inhibitory effects of an oxazol-5-one (DI) compound on Rho-associated kinase II functions in human coronary artery smooth muscle cells

Justin A. MacDonald, University of Calgary

Background: The (4Z)-2-(4-chloro-3-nitrophenyl)-4-(pyridin-3-ylmethylidene)-1,3-oxazol-5-one (DI) compound was developed by structure-based virtual screening and in silico computational modeling [J Med Chem (2009) 52:7323–7327]. The selectivity of DI for zipper-interacting protein kinase (ZIPK) was previously reported by screening a large panel of kinases and determining the inhibition efficacy. Our assessment of the DI compound revealed another target, the Rho-associated protein kinase 2 (ROCKII). **Methods/Results:** In vitro studies showed DI to be a potent, competitive inhibitor of ROCKII, Ki ofof 132 nM. This finding was supported by in silico molecular surface docking of DI with the ROCKII ATP-binding pocket. Furthermore, time course analysis of myosin light chain (LC20) dual phosphorylation (Ser-19 and Thr-18) catalyzed by ROCKII in vitro revealed a significant decrease in the phosphorylation upon treatment with DI compound. In addition, we investigated ROCK II signaling pathway in situ in human coronary artery vascular smooth muscle cells (CA-VSMC). ROCKII down-regulation using siRNA revealed several potential substrates involved in smooth muscle contraction pathway (e.g., LC20, Par-4) and actin cytoskeleton dynamics (cofilin). The application of DI to CA-VSMC attenuated the phosphorylation of LC20 (Ser-19), Par-4 (Thr-155) and cofilin (Ser-3). Notably, cofilin and Par-4 phosphorylation were not significantly decreased with a novel ZIPK selective inhibitor (HS-38). In addition, CA-VSMCs treated with DI compound underwent cytoskeletal changes that were associated with diminution of cofilin phosphorylation. **Conclusions:** We conclude that DI is not selective for ZIPK but is also a potent inhibitor of ROCKII and this compound could be a potential drug to study ROCK-associated diseases (e.g., hypertension, pulmonary hypertension, stroke and heart failure).

ABSTRACTS
Thursday, October 22

BIOMEDICAL POSTER SESSION #1: 19:00 - 20:30 MISSISSAUGA FOYER

BMR3 - Gamma/Delta T cells mediate angiotensin II-induced hypertension and vascular injury

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Objective: Both innate antigen presenting cells and the adaptive immune system, have been shown to pathophysiology of hypertension remains unclear. There is a small subset of "innate-like" T cells expressing γ/δ T cell receptor (TCR) rather than the α/β TCR that could play a role in bridging between the innate and adaptive immune. However, it is unknown whether γ/δ T cells contribute to development of hypertension. **Method/Results:** Thirteen to 15 week-old male C57BL/6 wild-type and Tcrd-/- mice, which are devoid of γ/δ T cells, were infused or not with angiotensin (Ang) II (490 ng/kg/min, SC) for 7 or 14 days (n=4-9). Telemetric blood pressure, mesenteric artery endothelial function and vascular remodeling by pressurized myography and spleen T cell profile by flow cytometry was evaluated. Fourteen days of Ang II increased systolic blood pressure by 42 mmHg (P≤0.01) in wild-type compared to control mice. The frequency of γ/δ T cells (2.3-fold, P≤0.05) and activated (CD69+) γ/δ T cells (1.6-fold) was increased after 7 days of Ang II, and 7 days later was respectively unchanged or further increased (2.4-fold) in wild-type compared to control mice. Ang II decreased mesenteric artery relaxation responses to acetylcholine by 42% (P≤0.01) and increased media/lumen by 45% (P≤0.01) in wild-type mice compared to controls. No γ/δ T cells were detected in Tcrd-/-treated or not with Ang II. All the above Ang II effects were abrogated in Tcrd-/- mice. Conclusion: These data suggest that γ/δ T cells mediate Ang II-induced blood pressure rise and vascular injury. γ/δ T cells could be key immune cells bridging innate and adaptive immune responses during the development of hypertension.

BIOMEDICAL POSTER SESSION #1: 19:00 - 20:30 MISSISSAUGA FOYER

BMR4 - Induction of human endothelin-1 overexpression for 3 months causes blood pressure rise and small artery endothelial dysfunction and stiffening

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Background: The mechanisms of blood pressure (BP) regulation by endothelin (ET)-1 produced by endothelial cells are complex and remain unclear. Recently, we developed a transgenic mouse with tamoxifen-inducible endothelium-restricted human ET-1 overexpression (ieET-1) using Cre/loxP technology. ieET-1 mice exhibited BP rise after three weeks of induction in an ET type A receptor-dependent manner, in absence of vascular and kidney injury. It is unknown whether long-term exposure to ET-1 overexpression results in sustained BP elevation and vascular injury. **Methods/Results:** Nine to 12-week old male ieET-1 mice and control ieCre mice expressing a tamoxifen-inducible Cre recombinase (CreERT2) under the control of endothelium-specific Tie2 promoter were treated with tamoxifen (1 mg/kg/day, s.c.) for 5 days and studied 3 months later. Analysis of 24-hour urine collection from metabolic cages indicated that the excretion of urinary sodium, potassium and protein was similar in both groups. Renal artery flow assessed by ultrasonography was decreased in ieET-1 compared with control (1.9±0.2 vs 3.0±0.3 mL/min, P0.01). Telemetry experiments revealed that systolic BP was increased in ieET-1 compared with ieCre mice (144±5 vs 117±3 mmHg, P0.001). Plasma aldosterone levels measured by ELISA were increased in ieET-1 compared with ieCre mice (1.99±0.20 vs 1.29±0.12 ng/mL, P0.05). Mesenteric artery (MA) endothelial function and vascular remodeling were assessed by pressurized myography. MA endothelium-dependent relaxation responses to acetylcholine were impaired in ieET-1 compared to ieCre mice (36.3±4.7 vs 71.4±9.7%, P0.01), whereas endothelium-independent relaxation responses to sodium nitroprusside were unchanged. MA media/lumen and media cross-sectional area were similar in both groups, but stiffness was increased in ieET-1 compared to ieCre mice, as indicated by leftward displacement of the stress-strain curves (strain at 140 mmHg: 0.61±0.04 vs 0.71±0.02, P0.05). Conclusions: The results demonstrate that long-term exposure to endothelial ET-1 overexpression caused sustained BP rise and small artery endothelial dysfunction and stiffening.

BIOMEDICAL POSTER SESSION #1: 19:00 - 20:30 MISSISSAUGA FOYER

BMR5 - Divergent roles of GPER vs. ER in estrogen-mediated regulation of LDL cholesterol metabolism

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Background: The beneficial effect of estrogen in regulation of plasma lipid metabolism has been long appreciated. However the receptor mechanisms remain largely undefined. Our recent studies demonstrated that GPER activation increased LDL receptor protein expression and decreased proprotein convertase subtilisin kexin type 9 (PCSK9) expression (Hussain et al. Arteriosclero Thromb Vasc Biol. 2015 Jan; 25(1)213-11). In this follow-up studies we have assessed the roles of classical estrogen receptors (ERα and ERβ) vs GPER in estrogen-mediated regulation of LDL receptor and PCSK9 expression in human liver hepatoma, HepG2, cells. **Results:** Activation of classical ERα or ERβ by either estradiol (E2) or their specific agonists, PPT or DPN respectively, mediated concentration-dependent increases in levels of LDL receptor protein expression similar in magnitude to the effects previously seen with GPER activation by it specific agonist G1(Hussain et al. Arteriosclero Thromb Vasc Biol. 2015 Jan;25(1)213-11). Inhibition of GPER-mediated effects either using the antagonist G15 or via down-regulation of GPER expression by transduction of an adenoshGPER construct partially blocked the E2-mediated increase in LDL receptor expression. Similarly, DPT, PPN and E2 all led to comparable increases in LDL receptor mRNA content. In contrast, GPER activation by G1 has no effect on LDL receptor mRNA expression. In regards to PCSK9 regulation, DPT and PPT mediated concentration-dependent increases in PCSK9 protein content, while E2 or the GPER agonist G1 decreased PCSK9 expression. Inhibition of GPER either by G15 or by transduction of the adenoshGPER construct blocked G1- and E2- inhibition of PCSK9 protein expression, but had no impact on PPT or DPN-mediated increases of PCSK9 expression. Further, DPN and PPT increased PCSK9 mRNA expression, while G1 decreased PCSK9 mRNA expression. E2 had no effect. Finally, following downregulation of GPER by transduction of an adenoshGPER construct E2 treatment now mediated an increase in PCSK9 mRNA expression- consistent with an effect occurring via ER activation. **Conclusion:** Our findings suggest that both ERs and GPER mediated increases in LDL receptor expression. ERs mediate there effects primarily via increased LDL receptor transcription with a parallel increase in PCSK9 expression and GPER primarily via inhibition of PCSK9 expression.

BIOMEDICAL POSTER SESSION #1: 19:00 - 20:30 MISSISSAUGA FOYER

BMR6 - Sodium nitroprusside (SNP) attenuates hypertension in spontaneously hypertensive rats: role of Gi proteins and oxidative stress

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Background: Nitric oxide (NO) donors are used as promising therapeutic agents for the treatment of cardiovascular diseases such as angina pectoris, myocardial infarction and congestive heart failure, however, the molecular mechanisms underlying the therapeutic activities remains poorly understood. We previously showed that NO donor, SNAP, decreased the expression of Giα proteins and associated functions in A10 vascular smooth muscle cells (VSMC) as well as in aortic VSMC from spontaneously hypertensive rats (SHR). Since the enhanced expression of Giα proteins has been implicated in the pathogenesis of hypertension, we undertook the present study to investigate whether in vivo treatment of SHR with NO donor; sodium nitroprusside (SNP) could attenuate the development of high blood pressure (BP) and to explore the role of Gi proteins, oxidative stress and signaling mechanisms responsible for this response. **Methods:** 8 week-old SHR and Wistar-Kyoto (WKY) rats were intraperitoneally injected with SNP at a concentration of 0.5mg/kg body weight twice a week. The levels of Gi and other proteins were determined by western blotting techniques. **Results:** Intraperitoneal injection of SNP attenuated the high BP by about 50 mmHg; however, this treatment did not affect BP in WKY rats. In addition, the hyper proliferation , increased production of superoxide anion, NAD(P)H oxidase activity, overexpression of Nox1/Nox2/Nox4, p47phox, superoxide dismutase 1 (SOD1), SOD2, Giα proteins, increased phosphorylation of platelet-derived growth factor receptor (PDGF-R), epidermal growth factor receptor (EGF-R), c-Src, and ERK1/2 exhibited by aortic VSMC from SHR were also attenuated to WKY levels by SNP treatment. Furthermore, SNP treatment also restored the decreased levels of endothelial NO towards WKY levels. **Conclusions:** These results suggest that in vivo treatment of SNP attenuates the high BP in SHR through the inhibition of enhanced levels of Giα proteins, oxidative stress, c-Src and EGF-R/PDGF-R activation and MAPK signaling pathways. (Supported by grant from CIHR)

ABSTRACTS
Thursday, October 22

BIOMEDICAL POSTER SESSION #1: 19:00 - 20:30 MISSISSAUGA FOYER

BMR7 - ER stress inhibition decreases hypertensive proteinuria in a CKD mouse model

Zahraa Mohammed-Ali, [1], Chao Lu, [2], Kjetil Ask, [1,2], Jeffrey G. Dickhout, [1,2].
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Background: Endoplasmic reticulum (ER) stress is an imbalance between protein-folding capacity and demand for protein synthesis and is involved in the pathogenesis of hypertension and chronic kidney disease (CKD). ER stress contributes to inflammation and fibrosis in various disease states. Inflammation and renal fibrosis contribute to CKD progression. We hypothesized that inhibiting ER stress using 4-phenylbutyrate (4-PBA), a molecular chaperone, would impede disease progression. **Methods/Results:** CKD was induced in male C57BL/6 mice by uninephrectomy and subcutaneous implantation of an Angiotensin II osmotic infusion pump and a slow release deoxycorticosterone acetate (DOCA) pellet. Mice were placed on 1% sodium chloride in their drinking water. Mice were sacrificed on days 7, 14, 18 and 21 post-implantation. Mice undergoing the CKD model experienced an increase in systolic and diastolic blood pressure and an increase in albuminuria and protein cast formation at all-time points compared to sham controls. Nanostring analysis showed a significantly higher expression of ER stress genes ASK1, Akt, Bcl2, and phlda1 and chaperones, HSP27, and HSP47 as early as day 7 post-implantation. Changes in inflammatory and fibrotic genes followed from day 14 onwards. Particularly, we observed an increase in genes showing immune cell infiltration, MCP-1, Arginase 2, CD44 and CD68, and fibrosis genes, α-smooth muscle actin and collagen. Therefore, ER stress preceded inflammation and fibrosis in our model. To test the role of ER stress in CKD development, 4-PBA was administered in the drinking water of mice undergoing the CKD model. 4-PBA prevented the development of hypertension and significantly reduced albuminuria and protein cast formation in the kidney in response to CKD. **Conclusion:** Our findings demonstrate a decrease in CKD progression with 4-PBA treatment. Further studies would determine the effect of 4-PBA on inflammation and fibrosis. Identifying molecular targets and pathways underlying CKD will help develop therapeutics to reduce disease progression.

BIOMEDICAL POSTER SESSION #1: 19:00 - 20:30 MISSISSAUGA FOYER

BMR8 - A primitive somite-derived stem cell in the adult mouse is capable of myelopoiesis

Sarah K. Steinbach[1,2], Angela Li[7], Rickvinder Besla[6], Martha H. Carruthers[2], Eric A. Shikatani[6], Clinton S. Robbins[3,6-8] and Mansoor Husain[1,2,4-6,8]
[1]McEwen Centre for Regenerative Medicine, and Divisions of [2]Experimental Therapeutics and [3]Advanced Diagnostics, Toronto General Research Institute, 101 College Street, Toronto, Ontario, Canada, M5G 1L7. Departments of [4]Medicine, [5]Physiology, [6]Laboratory Medicine & Pathobiology, [7]Immunology and the [8]Heart and Stroke Richard Lewar Centre of Excellence, University of Toronto, 1 Kings College Circle, Toronto, Ontario, Canada, M5S 1A8.

Background: Tissue resident macrophages have been shown to be derived from the yolk sac. Given the lineage heterogeneity of the aorta, we hypothesized that macrophages may be derived from alternate sources. **Methods & Results:** Using specialized Cre mice which irreversibly mark cells of specific embryonic origin, we isolated distinct subsets of progenitors from the aorta that are derived from neural crest (Wnt1), somite (Myf5) and another (Brachyury; T) mesoderm source. In Myf5- and T-marked lineages, this tdT+ population gave rise to clonally-derived spheres that differentiated into VSMC, adipocytes, S100β glia and macrophages in vitro. Further evidence of macrophage progenitor activity was demonstrated in an in vitro CFU assay where tdT+ cells gave rise to F4/80+CD11b+ macrophages. Transplantation of tdT+ CD45 negative aortic stem cells results in lineage marked macrophage formation in the aorta, but not the bone marrow, blood or spleen, demonstrating that this stem cell is capable of homing and undergoing myelopoiesis. In vivo, leukocytes were found to be lineage-marked suggesting derivation from somitic mesoderm. To ensure that this intriguing finding was not the result of ectopic expression of Myf5 outside of the somite, we acquired a tamoxifen inducible Myf5-CreER mouse and crossed it to a floxed tdTomato reporter mouse. Unfortunately, no labeled white blood cells of any kind were found in the adult aorta, bone marrow, blood or spleen when induced with tamoxifen at E12.5. When we examined skeletal muscle however, we found that ~40% of the macrophages were lineage marked. In 1, 5 and 10 day old neonates, ~80% of the macrophages in the skeletal muscle were lineage marked. Intriguingly, we also found that ~10% of the macrophages in the aorta were tdT+ in a small number of neonatal animals. **Conclusion:** The aorta harbors a somite-derived stem cell that gives rise to macrophages, but only in the neonate in vivo. The presence of lineage marked macrophages in skeletal muscle suggests that this stem cell may be located there as well. Given the regenerative potential of the neonatal aorta and skeletal muscle, we suggest that somite-derived macrophages may be involved in regeneration.

BIOMEDICAL POSTER SESSION #1: 19:00 - 20:30 MISSISSAUGA FOYER

BMR9 - Doxycycline hyclate administration differentially affects vascular mineral accrual in CKD based on vessel anatomical location

Jason G.E. Zelt Queen's University, Kimberly Laverty Queen's University, Rachel M. Holden Queen's University, Michael A. Adams Queen's University.

Background: Substantial regional heterogeneity exists in the ability of blood vessels to accumulate calcium and phosphate. A pathological accumulation of these minerals results in vascular calcification (VC). Given that vascular extracellular matrix (ECM) structure varies based on function and anatomical location, the ECM likely has a role in a vessel's propensity for VC. This study aims to examine the role of ECM constituents, specifically the matrix metalloproteinases (MMPs), on vascular mineral accrual in a progressive model of VC, chronic kidney disease (CKD). **Methods:** Male Sprague-Dawley rats (n=17) were provided a CKD-generating diet (0.25% adenine) for 7 weeks and stratified by serum creatinine after three weeks into two treatment groups: CKD (0.25% adenine, n=8, creatinine: 472.3±81.58) and CKD with doxycycline (CKD-DX, 0.30mg/kg doxycycline twice daily with 0.25% adenine, n=9, creatinine: 382.2±63.99). An additional age-matched Control group (n=6, creatinine: 41.62±4.267) was included. **Results:** Standardized to mean control levels, central arteries (thoracic aorta and abdominal aorta) of CKD rats possessed 2.49x the amount of calcium compared to the CKD-DX group, whereas the peripheral arteries (carotid artery, iliac artery, and renal artery) of CKD rats contained 4.87x the amount of calcium compared to CKD-DX rats. Accumulation of phosphate in CKD-DX rats was significantly diminished in the peripheral arteries (carotid, iliac, renal, inferior pudendal, and femoral arteries) in relation to CKD animals (p=0.0029). Central artery phosphate accumulation was not significantly different between CKD-DX and CKD groups (p=0.2291). **Conclusions:** These findings in an experimental model of CKD support prior observed regional heterogeneity in vascular mineral accrual, and suggest that differing characteristics of the vascular ECM influence this heterogeneity. As doxycycline is a known inhibitor of MMPs, a family of vascular ECM remodelling proteins associated with VC, there is potential that peripheral vessels are more susceptible to their activity compared to central vessels.

BIOMEDICAL POSTER SESSION #1: 19:00 - 20:30 MISSISSAUGA FOYER

BMR10 - The relationship between fibroblast growth factor 23 and kidney function

Emilie Ward [1], Wilma Hopman [2], Michael Adams PhD [1], Christine White MD [3], Rachel Holden MD [3], [1] Department of Biomedical and Molecular Sciences, [2] Kingston General Hospital, [3] Department of Medicine, Queen's University.

Background: Fibroblast growth factor -23 (FGF-23), a counter-regulatory hormone involved in phosphate homeostasis, is increasingly linked to cardiovascular disease in individuals without chronic kidney disease. FGF-23 promotes phosphaturia in conjunction with its cofactor, α-klotho. When evaluated by estimated glomerular filtration rate (eGFR), FGF-23 becomes significantly increased when eGFR is < 60 ml/min/1.73m2. We determined the level of FGF-23 and measures of phosphate homeostasis in individuals with GFR measured by inulin clearance. **Methods and Results:** GFR was measured (mGFR) in 80 individuals (ages 19-88) by inulin clearance. FGF-23, calcium, phosphate, parathyroid hormone (PTH) and α-klotho were measured in serum. The mean (SD) GFR was 45.1 (30.4) ml/min. GFR was correlated with FGF-23 (r=-0.7, p<0.001), PTH (r=-0.5, p<0.001), age (r=-0.4, p=0.002), phosphate (r=-0.4, p=0.002) and α-klotho (0.2, p=0.05). Median FGF-23 was 10.3 [IQR, 6.67-21.4]. FGF-23 correlated with age (r=0.5, p <0.001), phosphate (r=0.5, p<0.001) and PTH (r=0.5, p<0.001) but not with α-klotho. Compared to the reference group (GFR > 75 ml/min), FGF-23 was significantly increased at GFR of < 30 ml/min (p<0.001) with a trend towards an increase in the 31-45 ml/min group (p=0.07). There was a significant increase in FGF-23 and decrease in α-klotho in participants > 75 yrs. compared to all other age groups while phosphate and PTH were similar. The correlation between FGF-23 and GFR remained significant in the following age groups: < 45 (r=-0.6, p=0.03), 45-60 (r=-0.5, p=0.01), 60-75 (r=-0.4, p=0.004)). The correlation between FGF-23 and GFR was not significant in individuals > 75 yrs. **Conclusion:** Although FGF-23 was strongly associated with GFR, it was only significantly different than the reference group (>75 ml/min) when GFR was < 30 ml/min. This represents a lower level of kidney function than previously reported. Advancing age (>75 yrs.) may influence levels of FGF-23 independent of phosphate and kidney function.

ABSTRACTS
Thursday, October 22

CLINICAL/OUTCOMES/POPULATION POSTER SESSION #1: 19:00 - 20:30 MISSISSAUGA FOYER

CR1 - Are home blood pressure devices accurate? A systematic review of the evidence

Jeanne Francoise Kayibanda, Ottawa Hospital research Institute; Marcel Ruzicka, The Ottawa Hospital; Ayub Akbari, Ottawa Hospital Research Institute; Swapnil Hiremath, Ottawa Hospital Research Institute.

Background: Out of office blood pressure (BP) measurements, especially home BP monitoring, are recommended for diagnosis and management of hypertension by many societies including by the Canadian Hypertension Education Programme (CHEP). Though validation protocols exist for individual monitors, little data exists on the real world accuracy of home BP monitors, in actual use. We undertook this systematic review to estimate the accuracy of home BP monitors as reported in the literature. **Methods:** We undertook a literature search of MEDLINE and EMBASE from 1946 and 1947 until April 2015 respectively. We included studies that evaluated the accuracy of home BP devices against a mercury sphygmomanometer considered as the gold standard. Two reviewers independently selected studies, extracted data and assessed quality. Disagreements between the two reviewers were resolved by a third author. Results: Our search revealed 798 non-duplicate citations. After applying selection criteria, nineteen studies, involving 4954 patient-devices (median 91, interquartile range 69, 489) were included in the systematic review. The reported inaccuracy of home BP monitors, compared to mercury sphygmomanometer, ranged from 10% to 72% for systolic BP, with each study using different thresholds for definition of inaccuracy. The absolute mean difference for systolic BP between home BP monitor and the standard ranged from 2.4 mm Hg to 10.4 mm Hg and for diastolic BP from 1 to 8.7 mm Hg. **Conclusion:** The existing literature reports a relatively high degree of inaccuracy in home BP monitors being used. Data is limited by varying definitions being used for reporting inaccuracy. Consideration should be given to standardised definitions of accuracy, and real world monitoring of accuracy as home BP monitors use for clinical decision making becomes widespread. Research into predictors of inaccuracy in home BP monitors is also necessary.

CLINICAL/OUTCOMES/POPULATION POSTER SESSION #1: 19:00 - 20:30 MISSISSAUGA FOYER

CR2 - Implementation of Canada's sodium reduction strategy: Evaluation of actions, barriers and facilitators to reducing sodium in public institutions

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**Co-First Authors*

Background: Canada's Sodium Reduction Strategy is a population health approach to reducing the effects of excess dietary sodium on hypertension and cardiovascular diseases. Recommendation 10-1 was to "develop consistent sodium guidelines and procurement policies for use by food service operations in publicly-funded institutions" (e.g., health and care institutions); however, little coordinated action by governments has occurred. The purpose of this study was to conduct an environmental scan to examine current actions, attitudes, barriers, and facilitators related to sodium reduction in hospitals and long-term care (LTC) facilities. **Method/Results:** A cross-sectional 46-question survey was administered to food service administrators working in hospitals and LTC facilities in Ontario. Twenty-seven participants representing 9,823 patient/resident beds were included (33% response rate). Overall, 63.0% of institutions had an established sodium target ranging from 1500-4000 mg/day, of which 53.9% had a target exceeding the recommended Tolerable Upper Level. Among the facilities that knew the sodium content of their regular menus (70.0%), the average reported level was 2845 ± 1025 mg/day, with most estimates (64.3%) excluding sodium from added salt. Among respondents, 63% believed it is important to reduce sodium on inpatient/resident menus. The top reported facilitators to sodium reduction were: support from group purchasing organizations to identify lower sodium foods (85.2%), increased availability of pre-packaged lower sodium products (77.8%), the government making it a priority and providing support and resources (74.1%), and improved taste of lower sodium foods (74.1%). Only 37.0% believed that patient/resident satisfaction would decrease with sodium reduction. **Conclusions:** Sodium reduction targets and practices are variable among food service operations, and in some cases inconsistent with national recommendations. This data supports the need for consistent and coordinated sodium reduction policies in public settings and for multi-sectoral support, including action from food industry, group purchasing organizations, and hospital administration.

CLINICAL/OUTCOMES/POPULATION POSTER SESSION #1: 19:00 - 20:30 MISSISSAUGA FOYER

CR3 - Is dietary intake of calcium associated with surrogate markers of cardiovascular disease in healthy postmenopausal women?

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Introduction: Postmenopausal women are at increased risk of osteoporosis and calcium intake is thus recommended; however, calcium intake has uncertain cardiovascular (CV) outcomes. We examined the association of dietary calcium intake (dCa) with arterial stiffness and hemodynamics, and carotid intima-media thickness (cIMT) in healthy postmenopausal women. **Methods:** Healthy postmenopausal women (no CV risk factors) were recruited. Arterial stiffness (carotid-to-femoral pulse wave velocity [cfPWV]), peripheral and central systolic and diastolic blood pressures (pSBP, pDBP, cSBP, cDBP), mean arterial BP (MAP), and hemodynamic parameters (pulse pressure [PP], augmentation pressure (AP), augmentation index corrected for a heart rate of 75 bpm [AIx75]) were obtained by applanation tonometry. cIMT (B-mode ultrasonography) of common carotid arteries was measured. Measurements were compared across tertiles of dCa and serum ionised calcium (s-iCa) by one-way analysis of variance. **Results:** We evaluated 43 postmenopausal women (mean age 59.5±6.3 years; BMI 25.7±3.6 kg/m2, waist-to-hip ratio 0.87±0.05): average dCa 855.13±363.87 mg per day, s-iCa level 1.25±0.04 mmol/L. Third tertile of dCa had higher cSBP (110.3 [103.4, 116.3] vs. 95.0 [90.0, 102.5] mmHg), central PP (43.0 [34.4, 50.8] vs. 33.0 [29.0,40.0] mmHg), AP (15.9±5.7 vs. 10.4±5.4 mmHg) and AIx75 (28.6±6.1 vs. 20.8±9.1 %) than second tertile (all, $p<0.05$). Both peripheral and central (p/cBP) were normal (all tertiles). Right-side cIMT were higher in the second and third tertiles (0.62 [0.53, 0.67] mm and 0.59 [0.57, 0.69] mm, respectively) than first tertile (0.54 [0.49, 0.58] mm) of s-iCa (all, $p<0.05$). Subjects in all tertiles had similar age, BMI, and waist-to-hip ratio. **Conclusion:** We observed that higher dCa was associated with higher, though within normal range, cSBP, cPP, AP and AIx75. Higher s-iCa was associated with higher right-side cIMT. Importantly, our population had optimal/normal p/cBP. A larger, more representative sample is needed to determine the relationship between dCa/s-iCa and CV surrogate markers.

CLINICAL/OUTCOMES/POPULATION POSTER SESSION #1: 19:00 - 20:30 MISSISSAUGA FOYER

CR4 - Hypertension screening, management and follow-up by community pharmacists

Tsuyuki RT, Kaczorowski J, Syron L, Berg A, Farrell J, Padwal, RS, Feldman, RD.

Background: Hypertension continues to be a significant public health burden in Canada. Community pharmacies are accessible, visited frequently and are staffed with health care professionals who can play an important role in hypertension care including detection, management, referral and follow up. **Methods:** Between February 9, 2015 – March 8, 2015, we enrolled 25,754 individuals (average age 55 SD 18, 50% female); more than 900 assessments per day. We followed CHEP-recommended procedures for BP measurement and used the validated PharmaSmart PS2000. All patients received counselling and recommendations based on their results as well as educational material endorsed by Hypertension Canada. In patients with significantly elevated BP (SBP>150 mmHg) the subject's family physician was contacted using a standardized protocol and the pharmacist performed a 60-day follow-up call with a request to come in and repeat their blood pressure measurement. In some locations, a dietitian was available to discuss low-sodium food choices. **Results:** Only 24% patients living with diabetes were at target blood pressure levels, while 68% individuals who did not have diabetes had normal blood pressure readings. More than half (55%) of subjects had measured their blood pressure within the past 2 months. The location of their last blood pressure measurement included the doctor's office (43%), pharmacy (33%) and home (8%). Twelve percent (2995) of those having an initial consultation had a systolic BP >150 mmHg and therefore qualified for a phone follow-up by the pharmacist. There were 1223 (41%) phone follow-ups completed. Of this group, 524(43%) had a follow-up doctor visit and 238 (19%) reported having a new medication or a change to their current blood pressure medication. Almost half of subjects (602, 49%) reported improved adherence. In terms of the location of their last blood pressure measurement, 26% measured it at the doctor's office, 41% used the pharmacy blood pressure kiosk and 13% were using a home blood pressure monitor. There were 390 patients who subsequently returned to the pharmacy to measure their blood pressure, of which 337 (86%) were 150mm Hg. **Conclusions:** In a >25,000-subject community-based screening program, >28% had BP levels above target. Pharmacy-based BP measurement is feasible, reaches many individuals in the community, and identifies those needing further intervention.

ABSTRACTS
Thursday, October 22

CLINICAL/OUTCOMES/POPULATION POSTER SESSION #1: 19:00 - 20:30 MISSISSAUGA FOYER

CR5 - Attenuation of arterial stiffness gradient with age and its impact on central pulse wave profile in dialysis patients

Catherine Fortier, David Turgeon, Marie-Pier Desjardins, Karine Marquis, Marcel Lebel and Mohsen Agharazii. CHU de Québec Research Center-Université Laval.

Background: Arterial stiffness has been considered as an index of vascular ageing. However, the relationship between age and stiffness of various segments of the arterial tree is heterogeneous. Moreover, the hemodynamic impact of vascular stiffness is thought to be related to the attenuation or even reversal of the physiological arterial stiffness gradient. The objectives of the present study were to evaluate the impact of age on aortic (central elastic artery) and brachial (muscular conduit artery) stiffness and to assess hemodynamic correlates of the arterial stiffness gradient. **Method/Results:** In 310 dialysis patients (67 years [25th-75th percentiles: 56-76], 185(60%) men, 134(43%) diabetes and 162(52%) cardiovascular disease), we measured aortic and brachial stiffness by pulse wave velocity of carotid-femoral (cf-PWV) and carotid-radial (cr-PWV) using direct distances (Complior). Arterial stiffness gradient was determined by cf-PWV/cr-PWV (PWV ratio). Central pulse wave parameters were obtained through generalized transfer function applied to the radial artery pulse profile (Sphygmocor). Mean cf-PWV, cr-PWV and PWV ratio were respectively of 13.52 ±4.07m/s, 8.76 ±1.68m/s and 1.59 ±0.52m/s. Cr-PWV decreased with age (β=-0.031, R=-0.274, P0.001), while cf-PWV (β=0.144, R=0.529, P0.001) and PWV ratio (β=0.021,R=0.613, P0.001) increased. In contrast with cf-PWV, PWV ratio was not associated with MBP (P=0.997). **Conclusion:** The use of PWV ratio may be a more logical choice for risk determination than aortic stiffness as it provides a better estimation of the loss of arterial stiffness gradient and is not influenced by MBP.

CLINICAL/OUTCOMES/POPULATION POSTER SESSION #1: 19:00 - 20:30 MISSISSAUGA FOYER

CR6 - Nutritional consequences and benefits associated with dietary sodium reduction in individuals

Katherine Jefferson BASc, University of Ontario Institute of Technology, Mavra Ahmed MSc, University of Toronto, Susanna Mak MD PhD, Mount Sinai Hospital, Johane Allard MD, University Health Network, Gary Newton MD, Mount Sinai Hospital, JoAnne Arcand PhD RD, University of Ontario Institute of Technology.

Background: Canada's Sodium Reduction Strategy is a population-based approach to address the adverse effects of excess sodium on blood pressure. Despite a comprehensive set of recommendations, including reduced sodium content of processed and prepared foods, minimal government action has occurred, shifting the burden of sodium reduction to the personal actions of Canadians. Since reducing sodium requires numerous dietary changes, it is unknown if unintended nutritional consequences occur when sodium intake is reduced. The objective of this study was to evaluate nutritional changes resulting from a dietary sodium reduction. **Methods/Results:** In 18 stable systolic HF patients (60±11 years, 78% male), nutritional changes were documented at baseline and after one week of a 2g/day sodium diet, as measured by 24-hour urine collections and food records prior to baseline and each day during the study. Following a 49% reduction in dietary sodium (3.6±0.2 to 1.8±0.2 g/d), we observed a significant reduction in calories (2467±187 to 1931±97 kcal/d, p0.01), carbohydrate (293±27 to 232±14 g/d, p=0.01), calcium (995±124 to 609±52 mg/d, p0.01), thiamin (2.0±0.2 to 1.5±0.2 mg/d, p=0.02), and folate (413±48 to 331±43 mcg/d, p=0.02) intakes. There was a decrease in saturated fat (32±5 to 21±2 g/d, p=0.03) and a trend o higher potassium (1262±82 to 1405±67 mg/1000kcal, p=0.06) intakes. There were decreases in body weight (93±7 to 91±7 kg, p<0.01) and systolic blood pressure (122±5 to 115±4 mmHg, p0.01). **Conclusion:** There were multiple nutritional consequences and benefits associated with sodium reduction among individual patients. When translated to a population level, personal action to reduce dietary sodium may result in a greater proportion of Canadians with inadequate intakes of nutrients of public health concern, such as calcium and folate. Overall, this data supports the strategy of reducing sodium added to processed and prepared foods, so that large changes in individual dietary patterns are not required.

CLINICAL/OUTCOMES/POPULATION POSTER SESSION #1: 19:00 - 20:30 MISSISSAUGA FOYER

CR7 - The effect of Vitamin D ergocalciferol supplements on mild and moderate high blood pressure

Samia Rizk

Objectives: To observe the effectiveness and safety of ergocalciferol intervention to mild and moderate essential hypertension with vitamin D deficiency. **Method:** 103 patients, ages 55 and above, were newly diagnosed as mild-moderate essential hypertension with vitamin D deficiency. They were divided into an observation group (53 cases) and a control group (50 cases) randomly. Patients of the observation group took ergocalciferol and nifedipine XL and the control group patients took nifedipine XL only. The two groups were both checked for: 25-hydroxyvitamin D, blood pressure, heart rate, renin-angiotensin-aldosterone level, serum calcium, serum phosphate, urea and serum creatinine before the treatment and after 4 weeks treatment, 12 weeks treatment and 24 weeks treatment. **Results:** After the treatment, the basic line of vitamin D level was elevated in the two groups, the level of systolic pressure and diastolic pressure was obviously declined ($P < 0.01$). The level of renin of the control group patients was dramatically declined compared with the basic line ($P < 0.05$). After 12 weeks of treatment compared with the control group, the level of renin, angiotensin were obviously declined ($P < 0.05$) in the patients of the observation group. After 24 weeks treatments, the systolic pressure of the observation group patients declined more than in the control group ($P < 0.05$). **Conclusions:** Blood pressure of mild to moderate essential hypertension with vitamin D deficiency can be obviously reduced after taking Vitamin D supplement, ergocalciferol, especially the systolic pressure, it is possible that the increasing of vitamin D level can depress renin-angiotensin-aldosterone system.

CLINICAL/OUTCOMES/POPULATION POSTER SESSION #1: 19:00 - 20:30 MISSISSAUGA FOYER

CR8 - The metabolic acidosis among patients with high blood pressure in the Great Lakes Region

Louise Nzigire, Antoine Flahault

Introduction: Metabolic acidosis is a factor in the decline of glomerular filtration rate, malnutrition, chronic inflammation and bone resorption. The aim of our work is to determine the time and stage of occurrence of metabolic acidosis in patients with hypertension and deduct the impact of bicarbonate supplementation on changes of glomerular filtration rate and nutritional status and bone. **Patients and Methods:** We conducted a retrospective, comparative study including 50 patients followed for severe hypertension who presented a metabolic acidosis. We compared two groups: patients who received supplementation with bicarbonates and patients without supplementation and we followed the evolution of glomerular filtration rate, rates of bicarbonatémie and albumin and the serum calcium rate, serum phosphorus and PTH levels of both groups of patients. **Results:** The average age of our patients was 55.7 ± 15.12 years; the average time to onset of metabolic acidosis compared to the first consultation was 9.5 ± 15,8mois. After a median follow-up 47.5 months, the decline in GFR was similar in both groups. The alkaline reserve was significantly higher at the last follow in the group with supplementation (24.80 ± 1,60mmol / L vs 20.5 ± 3,01mmol / L; $p < 0.0001$) with significant improvement in serum albumin (38.89 ± 4,02g / L vs 34.81 ± 4,90g / L; $p = 0.01$). We did not notice any difference between the two groups regarding changes in serum calcium, serum phosphate and PTH. **Discussion and Conclusion:** Metabolic acidosis occurs in our study in stage 3 and stage 4 and its correction has improved the nutritional status determined by the rate of albumin. We have not noticed any impact on the evolution of glomerular filtration rate nor on changes in calcium phosphate parameters.

ABSTRACTS

Thursday, October 22

CLINICAL/OUTCOMES/POPULATION POSTER SESSION #1: 19:00 - 20:30 MISSISSAUGA FOYER

CR10 - Prevalence of hypertension and associated factors among residents of Ibadan-North local government area of Oyo State, Nigeria

Dr. Ajayi, Ikeoluwapo. O., Department of Epidemiology & Medical Statistics, Faculty of Public Health, College of Medicine, University of Ibadan, Nigeria, Dr. Akpa, Onoja Matthew, Department of Epidemiology & Medical Statistics, Faculty of Public Health, College of Medicine, University of Ibadan, Nigeria.

Objectives: This study aimed to investigate the prevalence of hypertension and associated factors among the residents of Yemetu community; an urban-slum in Ibadan-North Local Government Area of Oyo State, Nigeria. **Methods:** A descriptive cross-sectional design was used. The study involved 806 respondents aged from 18-90 years from 171 households. They were selected by cluster sampling technique. It was a house-to-house survey. Behavioural risk factors were measured using World Health Organization (WHO) STEPwise approach to chronic disease risk factor surveillance (STEPS 1 & 2), while physical activities were measured using International Physical Activity Questionnaire (IPAQ). Hypertension was defined as Systolic blood pressure (SBP) ≥ 140 and/or Diastolic blood pressure (DBP) ≥ 90mm Hg or currently on antihypertensive medication. Data were analyzed using descriptive statistics, Chi-square and binary logistic regression tests at $p<0.05$. **Results:** The overall prevalence of hypertension was 33.1% (male 36.8% and female 31.1%). The proportion of self-more than a year ago, while 18.6% had never checked. The mean age of the respondents was 38.8 ±15.6 years. The body mass index of the respondents was 5.2%, 52.0%, 29.5% and 13.3% for underweight, normal, overweight and obese, respectively. Alcohol and tobacco use were found in 11.5% and 3.2%, respectively. The result of binary logistic regression analysis revealed that hypertension was significantly associated with being in age groups 30-49 years (OR 2.258, 95% CI: 1.311 - 3.884), ≥ 50 years (OR 7.145, 95% CI: 3.644 - 14.011), being overweight or obese (OR 2.281, 95% CI: 1.022 – 5.088). Hypertension was inversely associated with being underweight (OR 0.537, 95% CI: 0.395 – 0.832). **Conclusion:** This study revealed a high prevalence of hypertension among the inhabitants of Yemetu community, which puts them at risk of cardiovascular disease. The data underscores the need for urgent steps to create awareness and implement interventions for prevention and early detection of hypertension especially among those aged ≥30 years and the overweight or obese.

CLINICAL/OUTCOMES/POPULATION POSTER SESSION #1: 19:00 - 20:30 MISSISSAUGA FOYER

CR11 - Evaluation of the Ontario Stroke Network's Hypertension Management Program: A model for stroke prevention in primary care settings

Stephen Sundquist, Ontario Stroke Network

Background: Since 2012, the Ontario Stroke Network (OSN) has delivered the provincial Hypertension Management Program (HMP) to Primary care health teams. The HMP is modeled on Ontario's Chronic Disease Prevention and Management framework and supports best practice care delivery as outlined in the Canadian Hypertension Education Program (CHEP) guidelines. The HMP goal is to improve cardiovascular disease prevention through a focus on hypertension. In 2013, the OSN undertook a comprehensive evaluation of the HMP to inform stakeholders on the effectiveness, ongoing sustainability, and feasibility for program expansion. **Method:** A mixed-methods approach was used, including a quantitative HMP data repository analysis to evaluate changes from patient baseline of blood pressure (BP), relevant lab values and risk factor self-management. The qualitative evaluation involved site visits and surveys with participating health care providers (HCPs) and enrolled patients which further addressed experiences with program delivery. **Results:** Results showed BP reductions from baseline values in HMP patients. Statistically significant reductions were noted in systolic and diastolic BP for patients with elevated BP and those with diabetes at baseline (p values ranged from <0.01 to 0.0001). HCPs expressed increased confidence in hypertension (HTN) diagnosis and treatment, better understanding and compliance with CHEP guidelines, improved inter-professional communication and felt successful in providing patients with the knowledge and skills for self-management of HTN. Patients reported greater confidence in BP self-monitoring and addressing modifiable risk factors based on health information received. Areas cited for improvement were workflow efficiencies and more effective, enhanced patient- and provider-focused resources for providing health information. **Conclusions:** Evaluation outcomes of HMP delivery were positive overall, showing benefit for patients with hypertension and HCPs. Opportunities have been identified to explore program evolution including: improving point-of-care access to evidence and outcomes reporting for HCPs, as well as creating capacity for comparing HMP patient outcomes to population data.

CLINICAL/OUTCOMES/POPULATION POSTER SESSION #1: 19:00 - 20:30 MISSISSAUGA FOYER

CR12 - Effect of a workplace organizational intervention targeting adverse psychosocial work factors on blood pressure and hypertension

Chantal Brisson (CHU de Quebec), Mahée Gilbert-Ouimet (CHU de Québec), Alain Milot (Université Laval), Michel Vézina (Université Laval), Benoit Masse (Université de Montréal).

Background: The relationship between adverse psychosocial factors at work and cardiovascular disease has been documented in a number of large scale studies. Few studies have examined the impact of reducing these adverse exposures on cardiovascular health. This study examined the effects of a workplace organizational intervention targeting adverse psychosocial work factors on ambulatory blood pressure (ABP) and hypertension. **Methods:** The study population was composed of white-collar workers employed in large public organizations. The study design was a quasi-experimental before-after study with an intervention (N=1,093) and a control group (N=1,074). The intervention was defined as all organizational changes that aimed to reduce targeted adverse psychosocial work factors. These changes were made by the managers and monitored by the research team. ABP was measured at baseline, and at 6 and at 36 month after the intervention. The evolution of ABP means and hypertension prevalence in the intervention and the control group was used to estimate the effect of the intervention. **Results:** ABP means and hypertension prevalence significantly decreased in the intervention group while no such reductions were observed in the control group. The intervention effect was -2.14 mmHg (95% CI: -3.11, -1.17) for systolic BP and -1.09 (-1.80, -0.38) for diastolic BP. The prevalence ratio comparing change in hypertension prevalence in the intervention and the control group was 0.84 (95% CI: 0.73-0.96). p for group x time interactions were < 0.01 in all cases. **Conclusion:** Our findings suggest that adverse psychosocial work factors are relevant targets for the primary prevention of hypertension. The implementation of organizational interventions in workplaces could lead to meaningful improvements in cardiovascular health.

ABSTRACTS
Friday, October 23

BIOMEDICAL ORAL SESSION #1: 08:00 - 08:15 MISSISSAUGA B

Modeling type II diabetes-associated vasculopathies with skin-derived precursors (SKPs)

Sarah K. Steinbach[1,2], Terrance M. Yau[1,3], Maral Ouzounian[1,3], Mark Chandy[3], Husam Abdel-Qadir[3], Thomas K. Waddell[1,2,4], Mansoor Husain[1-3,5,6]
[1]Toronto General Research Institute, University Health Network, Toronto, Canada. [2]McEwen Centre for Regenerative Medicine, University Health Network, Toronto, Canada. [3]Peter Munk Cardiac Centre, University Health Network, Toronto, Canada. [4]Division of Thoracic Surgery, University Health Network, Toronto, Canada. [5]Heart and Stroke Richard Lewar Centre of Excellence, University of Toronto, Toronto, Canada. [6]Department of Medicine, University of Toronto, Toronto, Canada.

Background: Vascular complications of diabetes are a major cause of morbidity and mortality. In the current study we aimed to determine if adult patient-specific vascular smooth muscle cells (VSMC) can be derived from SKP found in chest or leg skin samples of patients undergoing cardiothoracic surgery, and to ascertain if they remain 'functional'. **Methods & Results:** 130 patients were recruited for this study. Multivariate linear regression analysis revealed a significant effect of coronary artery disease, lipids and age on the number of cells in skin. SKP derived from aged patients with or without type II diabetes (T2D) undergoing cardiothoracic surgery were differentiated into VSMC at similar frequencies to foreskin-derived SKP from children (>80% yield). However, adult patient-specific SKP failed to passage, suggesting accelerated senescence and/or disease. Although SKP from patients with T2D were also able to differentiate into VSMC, SKP from diabetics were isolated at significantly lower efficiencies compared to non-diabetics (2.49x105±0.06 vs. 5.96x105 ±0.09 cells/g skin). VSMC from adults with T2D exhibited increased responsiveness to PE and NE compared to non-diabetics suggesting a diabetes-specific phenotype. In addition, VSMC from patients with T2D had altered calcium handling characteristics and showed impaired wound healing responses compared to patients without T2D. **Conclusion:** This demonstrates that SKP-derived VSMC from patients with T2D carry metabolic memory and represent a promising platform for studying the epigenetic alterations associated with T2D-associated vascular dysfunction. Moreover, few methods enable molecular and cellular studies of aging and T2D in the vasculature. We report a novel minimally-invasive method of studying human VSMCs differentiated from progenitors obtained from small skin biopsies.

BIOMEDICAL ORAL SESSION #1: 08:15 - 08:30 MISSISSAUGA B

Role of potassium-chloride cotransporter type 3 in the cardiometabolic physiology in mice

Garneau, AP (CRCHUM & CRHDQ); Noel, M (CRHDQ); Drolet, MC (IUCPQ); Couet, J (IUCPQ); Lavoie, JL (CRCHUM); Isenring, P (CRHDQ).

Background: Potassium-chloride cotransporter type 3 (KCC3) is a member of the cation-chloride cotransporters which mediates electroneutral export of its substrates in various cell types such as adipocytes, cardiomyocytes, vascular smooth muscle cells and kidney epitheliocytes. Neurologic disorders have been reported in mice models with systemic disruption of Kcc3, which also display arterial hypertension. However, little is known regarding their vascular and metabolic features. **Method/Results:** In order to understand the role of KCC3 in the Cardiometabolic physiology and pathophysiology of hypertension, we characterized the phenotype of a mouse line systemically knocked-out for Kcc3 by gene trap. We measured cardiovascular parameters using sphygmomanometry, and in vitro aortic reactivity. Different molecular markers and vasoactive mediators were quantified by quantitative PCR and ELISA. These analyses were completed with biochemical dosings, and neurological tests. We found that Kcc3 inactivation is accompanied by neuromuscular weakness, a decrease in pulse pressure, heart rate, aortic reactivity to adrenergic stimulation and aortic wall thickness, along with an increase in diastolic pressure, cardiac mass and aldosteronemia. Interestingly, our mouse model also displays a marked reduction in adiposity without a decrease in caloric intake. Moreover, fatty acid synthase and adipose triglyceride lipase mRNAs tended to be upregulated in visceral fat from knockout mice. Preliminary data also indicate a significant reduction in circulating leptin and an elevation in adiponectin, ICAM-1 and pentraxin 3. **Conclusion:** The phenotypic abnormalities observed suggest that these cardiovascular disorders are caused at least in part by the absence of KCC3 in cardiovascular tissue and they are concordant with changes in vascular function. The leanness of Kcc3 knockout mice will be analyzed through more detailed metabolic and mechanistic studies. Finally, KCC3 appears as an interesting target in the treatment of hypertension and obesity: elucidation of its regulation could lead to promising tissue-targeted interventions.

BIOMEDICAL ORAL SESSION #1: 08:30 - 08:45 MISSISSAUGA B

A conserved microRNA cluster as a potential master regulator in angiotensin II-induced vascular damage

Tlili Barhoumi[1], Júlio C. Fraulob-Aquino[1], Chantal Richer[3], Mathieu Lajoie[3], Daniel Sinnett[3,4], Pierre Paradis[1], Ernesto L. Schiffrin[1,2] [1]Vascular and Hypertension Research Unit, Lady Davis Institute for Medical Research, [2]Department of Medicine, Sir Mortimer B. Davis-Jewish General Hospital, McGill University, [3]Division of Hematology-Oncology, Research Center, CHU Ste-Justine, [4]Department of Pediatrics, Faculty of Medicine, Université de Montréal, Montréal, Canada.

Introduction: Non-coding RNAs (ncRNAs), including long ncRNAs (lncRNAs) and microRNAs (miRNAs), account for ~98% of the transcribed RNAs. They have been shown to play a role in cardiovascular disease. Vascular damage is an early manifestation and a cause of end-organ damage in hypertension. However, it is unknown whether ncRNAs are involved in the development of vascular injury in hypertension. We hypothesize that ncRNA regulation plays a role in vascular remodeling and in the pathophysiology of hypertension. **Method/Results:** Ten week-old male C57BL/6 mice underwent sham surgery or angiotensin (Ang) II infusion for 7 or 14 days. Blood pressure (BP) was measured by telemetry. Total RNA was extracted from mesenteric arteries and used to construct libraries for total RNA and small RNA deep sequencing using Illumina HiSeq-2500. Differential expression analysis and heat maps were done in R. Targetscan was used to predict interactions between differentially expressed miRNAs (DEmiRs) and genes (DEGs). MEME Suite was used to predict differentially expressed transcription factor targets in the DEGs. Cytoscape was used to perform functional enrichment analysis and construct molecular networks integrating the above interactions and the gene expression profile. Differentially expressed mRNAs, lncRNAs, miRNAs and other small ncRNAs were identified in the Ang II-treated groups. Functional enrichment analysis showed enrichment of extracellular matrix in both 7-day and 14-day Ang II-induced DEGs, but developmental process in only the 14-day Ang II-induced DEGs. We identified 10 DEmiRs whose expression levels were correlated with BP, 9 of which are located in a single miRNA cluster that is highly conserved in humans. **Conclusions:** We have identified a conserved miRNA cluster that may play a pivotal role in the regulation of vascular damage. A sub-network of genes presenting the interaction between the miRNA cluster and other BP-correlated ncRNAs has been selected for future investigation to identify therapeutic targets.

BIOMEDICAL ORAL SESSION #1: 08:45 - 09:00 MISSISSAUGA B

Pudendal artery structure and function as early markers for elevated pulse wave velocity

Paul Jeronimo1, Jason Zelt[1], Kristin McCabe[1], Kim Laverty[1], Mandy Turner[1], Rachel Holden[2], Michael Adams[1], [1] Department of Biomedical and Molecular Sciences, Queen's University, Kingston, ON, [2] School of Medicine, Queen's University, Kingston, ON.

Background: Vascular calcification (VC), a common manifestation of cardiovascular disease (CVD) in patients with chronic kidney disease (CKD), increases arterial stiffness and negatively affects cardiovascular hemodynamics. Recent research indicates that peripheral vasculature, such as the pudendal artery, has a distinct susceptibility to vascular calcification. The objective of this study was to evaluate whether calcification of the pudendal artery in CKD is a sentinel for alterations in pulse wave velocity (PWV), an indicator of arterial stiffness. **Methods/Results:** Male Sprague Dawley rats (n=78) were placed on an adenine diet (0.25%, 7 weeks) to induce CKD. The adenine protocol generated a range of CKD severity (serum creatinine 8 to 513 µM, mean 209 µM). At 7 weeks, the carotid to iliac bifurcation PWV was measured under general anesthetic (isoflurane 2%). Calcium and phosphate content was analyzed in the pudendal artery, and vessel contractility was assessed via potassium-induced maximum contraction. Accumulating tissue phosphate and calcium (~ 6 to 795 nmol of phosphate/mg tissue; ~ 3 to 1380 nmol of calcium/mg tissue) negatively correlated with vessel contractility (r2=0.15, p0.0001) Increased mineral levels and decreased contractility of the pudendals were significantly correlated with an increase in PWV ($p<0.0001$). **Conclusion:** These findings show that the progressive accumulation of phosphate and calcium in the vascular microenvironment is particularly severe in the pudendal artery (éVC and êcontractile function), and occurs prior to systemic changes. The pudendal artery calcification appears to be an early sentinel for alterations in central haemodynamic parameters, including PWV. This research is funded by CIHR.

ABSTRACTS
Friday, October 23

CLINICAL/OUTCOMES/POPULATION ORAL SESSION #1: 08:30 - 08:45 MISSISSAUGA C

Long term exercise decreases von Willebrand Factor antigen levels in abdominally obese adults

Cynthia Pruss[1], Robert Ross[2], Paula James[3], Rachel Holden[3], Michael Adams[1] [1]Department of Biomedical and Molecular Sciences, [2]School of Kinesiology and Health Studies, [3]Department of Medicine, Queen's University, Kingston Ontario.*

Background: In obese individuals, the amount and intensity of exercise required to impact health is unknown. The circulating protein Von Willebrand Factor (VWF) binds platelets in primary hemostasis and is a chaperone for factor VIII. VWF is a biomarker for both endothelial dysfunction and hemostatic imbalance. Elevated VWF levels associate with cardiovascular risk factors (e.g. age, diabetes, and hypertension) and lower survival. However, the effect of chronic exercise on the levels of VWF in obesity is unknown. **Methods/Results:** Sedentary 35-65 year old males and females with elevated waist circumference (>102 cm (male) and >88 cm (female), 55% positive for metabolic syndrome) provided informed consent. The study was approved by the Queen's University Health Sciences Research Ethics Board. Participants were randomly assigned to 4 groups: low amount low intensity (LALI), high amount low intensity (HALI), high amount high intensity (HAHI), and control. BMI, waist circumference, two hour OGTT, VO2 Peak, C reactive protein, ABO blood group, total VWF (VWF:Ag), VWF propeptide, and VWF collagen binding activity were measured. Improvements were observed in VO2peak, waist circumference, and BMI for all exercise groups. Only HAHI improved in 2HOGTT glucose and Matsuda Index. Triglycerides, HDL cholesterol, blood pressure, C reactive protein and fasting glucose were not significantly different between groups at week 24. At week 16, HAHI and HALI VWF:Ag significantly decreased from week 0 (HAHI: -8.4 ±25U/dL ±SD, P=0.004, student's t-test; HALI: -9 ±22 U/dL, P=0.003), compared to control. VWF:Ag further decreased at 24 weeks for HAHI (-12.8 ±20.8, P=0.003) and HALI (-12.7 ±18.8, P=0.001) compared to control. LALI did not change significantly. **Conclusion:** This study demonstrates that higher amounts of exercise are able to significantly lower VWF levels in obesity, which is a group at risk for cardiovascular events previously linked to higher VWF levels.

CLINICAL/OUTCOMES/POPULATION ORAL SESSION #1: 08:45 - 09:00 MISSISSAUGA C

Impact on blood pressure from an interactive mobile based self-management system in patients with advanced chronic kidney disease

Jassal SV, Miller JA, Porter EC, Min, K, Cafazzo J, Seto E, Uddin A, Logan AG. Department of Medicine, University Health Network and Mount Sinai Hospital, Department of Pharmacy and Centre for Global eHealth Innovation, University Health Network, Toronto, Ontario.

Background: To improve chronic disease management, we developed an interactive mobile-based system to support self-care activities for patients with advanced chronic kidney disease (CKD) and help them become active partners with their clinical team in managing their condition. The system focused on four behavioural elements: monitoring blood pressure (BP), managing medications, symptom assessment and tracking laboratory results. We conducted a 6-month prospective study to determine the system's acceptability and clinical effectiveness. **Methods:** The system was tested in ten multidisciplinary renal clinics at a single academic renal centre in Toronto. Patients and clinicians were instructed at a regular clinic visit on its use. **Results:** We enrolled 47 patients whose mean age was 59 years with 33% aged 65 years or more. Only 38% were smartphone owners and 11% have never used or owned a mobile phone. All had >1 chronic condition and took 10-15 medications daily. User adherence was high (>80% performed 80% of recommended assessments). By the end of the study systolic BP fell by 6.8±20.4 mmHg ($p<0.05$) and diastolic BP, by 0.9±9.1 mmHg. For those with uncontrolled hypertension at baseline, systolic BP fell by 13.5 ± 21.5 mmHg ($p<0.05$). From home BP readings, 27% were newly identified as having 'masked' hypertension. A total of 127 medication reviews led to interventions to address medication errors in 74 (58%) instances. Medication possession ratio also improved. On study exit interviews, patients and clinicians consistently felt the system helped engage and empower patients in their CKD care. **Conclusion:** The mobile-based system was acceptable and improved several important clinical parameters including BP. The results strongly support evaluating the system in a larger randomized controlled trial of longer duration.

CLINICAL/OUTCOMES/POPULATION ORAL SESSION #1: 09:00 - 09:15 MISSISSAUGA C

Next-generation cloud-based blood pressure devices in chronic disease management: A direct intra-arterial pressure calibration of an oscillometric wrist cuff device for clinically reliable and accurate blood pressure measurements

Sarah Melville[1,2], B.Sc., CRA, Sohrab Lutchmedial[1], M.D., FRCP(C), & Keith R. Brunt1[-3], Ph.D. [1]Department of Cardiology, New Brunswick Heart Centre, Saint John Regional Hospital [2]Department of Pharmacology, Dalhousie Medicine New Brunswick, Dalhousie University [3]Office of the Vice President of Research, University of New Brunswick.

Background: Home blood pressure (BP) monitoring is an emerging clinical tool. Rigorous assessments of medical devices are necessary to establish these tools for clinical use. Here, we simultaneously compared indirect BP using a cloud-based digital oscillometric wrist cuff to that of direct arterial BP measurements taken by pressure catheter during routine cardiac catheterization procedures. **Methods:** Patients scheduled for cardiac catheterization were pre-screened for bilateral BP equality. During a catheterization procedure, simultaneous pressure measurements were made with the wrist cuff device and intra-arterial catheter at both the right radial artery (RRA) and at the ascending aorta (AA). Correlate and paired statistical analyses were performed and the cloud-based algorithms were subsequently adjusted based on the absolute BP measures. **Results:** Indirect BP readings of the wrist cuff device were highly correlated to direct BP readings at the RRA and the AA. Yet, paired measures revealed that diastolic pressures were significantly different at the RRA and the AA (N=10; P<0.0001). However, systolic BP readings were comparatively different at the AA (P<0.05), but not at the RRA. The absolute pressure measures were used to calibrate the cloud-based algorithm mathematically using a combination of train-setting and jack-knife processing. Following the adjustment of the device against the gold-standard of BP measurement, no significant differences were determined in either systolic or diastolic pressures at the RRA or AA comparatively. **Conclusions:** The emergence of secure cloud-based home vital sign monitoring provides new tools for physicians to manage chronic diseases and make clinically informed decisions. Most mobile health applications and home-based devices are currently for information and entertainment purposes only. Migrating the most utilitarian functions to become a clinically reliably tool is possible. Thus, digital cloud-based applications and vital sign devices could soon support the diagnosis of hypertension and patient management, particularly for distance care and chronic diseases.

CLINICAL/OUTCOMES/POPULATION ORAL SESSION #1: 09:15 - 09:30 MISSISSAUGA C

Cardiovascular risk and prevention strategies at the Great Lakes Regional Hospital

Jean Safari, Antoine Flahault, Louise Nzigire

Background: Cardiovascular risk has not been assessed in the Great Lakes Region. However, poor diet, physical inactivity, smoking and alcohol are the main risk factors. **Patients and Method:** Multicentre prospective cohort of 1232 patient's incidents in Internal Medicine at M0 in October 2012 and followed to M24 in October 2014. Blood pressure was measured by a Rossmax machine. Cardiovascular factors between M12 and M24 were assessed by Cox adjusted analyzes, the factors of variability between M0 and M12 by multivariate logistic regression. **Results:** 842 (68, 3%) were hypertensive. Increased Blood Pressure to a status of metabolic syndrome was independently and strongly associated with Cardiovascular mortality between M12 and M24 (RR 2.01, CI 1.2 to 3.37; p = 0.009). Among patients with high BP, factors M6 to the decrease were hypoalbuminemia (OR: 0.96; 95% CI from 0.93 to 0.99; p = 0.01), decreased weight (BMI: 27 kg / m2) (OR: 2.02; OC: 1.37 to 2.99; p <0.0001) and to lesser extent the use of native vitamin D (OR: 1.02, 1, 01 to 1.03, p = 0.014). Factors to M12 associated with CV mortality between M12 and M24 were age, CRP ≥ 10 mg / L and physical inactivity (OR: 5.43; 95% CI 2.17 to 13.62; p 0, 0001). **Discussion:** Cardiovascular diseases account for 82% of deaths in low income countries. It is possible to prevent most cardiovascular disease through early detection and support including counseling and medication. Conclusion: EPIRAGL advocates Control, increasing physical activity of the population by building footpaths and cycle paths; reducing harmful use of alcohol.

ABSTRACTS
Friday, October 23

BIOMEDICAL ORAL SESSION #2: 10:00 - 10:15 MISSISSAUGA B

Oxidative stress contributes to the enhanced expression of Gqα and PLCβ1 proteins and hypertrophy of VSMC from SHR: role of growth factor receptor transactivation

Atef Me and Anand Srivastava MB, Department of Molecular and Integrative Physiology, Faculty of Medicine, University of Montreal, Montreal, Quebec, Canada.

Background: We showed previously that vascular smooth muscle cells (VSMC) from spontaneously hypertensive rats (SHR) exhibit enhanced expression of Gqα and PLCβ1 proteins which contribute to increased protein synthesis (hypertrophy) through the activation of MAP kinase signalling. Since oxidative stress has been shown to be increased in hypertension, we undertook the present study to examine the role of oxidative stress in enhanced expression of Gqα and PLCβ1 proteins and VSMC hypertrophy and further explore the underlying mechanisms responsible for this response. **Method and Results:** Protein expression and phosphorylation were determined by Western blotting and protein synthesis was determined by [3H]-leucine incorporation. The increased expression of Gqα and PLCβ1 proteins as well as increased protein synthesis exhibited by VSMC from SHR were significantly attenuated by antioxidants: N-acetylcysteine (NAC), a scavenger of superoxide anion, DPI, an inhibitor of NAD(P)H oxidase, PP2 (c-Src inhibitor), AG1024 (IGFR inhibitor), AG1478 (EGFR inhibitor) and AG1295 (PDGFR inhibitor). In addition, the levels of IGF-1R and EGFR proteins and not of PDGFR were also enhanced in VSMC from SHR which were attenuated significantly by NAC, DPI and PP2. Furthermore NAC, DPI and PP2 also attenuated the enhanced phosphorylation of IGF-1R, PDGFR, EGFR, c-Src and EKR1/2 in VSMC from SHR. **Conclusion:** These data suggest that enhanced oxidative stress in VSMC from SHR activates c-Src which through the transactivation of growth factor receptors and MAPK signaling contributes to enhanced expression of Gqα and PLCβ1 proteins and resultant enhanced protein synthesis.'

Key Words: Oxidative stress, VSMC, hypertrophy, SHR, Gqα, PLCβ1

BIOMEDICAL ORAL SESSION #2: 10:15 - 10:30 MISSISSAUGA B

Effect of 4-phenylbutyric acid treatment on hypertension development and pre-hypertensive tachycardia in the SHR

Daniel Chung, Chao Lu, and Jeffrey G. Dickhout. Department of Medicine, Division of Nephrology, McMaster University and St. Joseph's Healthcare Hamilton, Hamilton, Ontario, Canada.

Background: Previous work from our research group has shown that the SHR develops elevated heart rate before the development of high blood pressure. Further, this pre-hypertensive tachycardia co-segregates with inbreeding lines in the SHR-WKY population predicting the highest level of blood pressure. We have also determined that ER stress inhibition prevents the development of high blood pressure in the SHR. Therefore, we hypothesized that ER stress inhibition would prevent the pre-hypertensive tachycardia found within the SHR. We also hypothesized that this would occur due to the lengthening of the QT interval. **Methods:** High blood pressure begins to develop in the young SHR at 4-weeks of age. Radio-telemetry transmitters (HD-X11 transmitter, Data Sciences International) were implanted to monitor blood pressure development, heart rate and electrocardiogram activity in both SHR and WKY. SHR and WKY were both randomized into 4-PBA (1 g/kg/day) or vehicle groups at 5-weeks of age to determine if ER stress inhibition would prevent the development of hypertension. ECG parameters including RR interval, QT interval, and Q-T Bazett corrected interval (QTcb) were analyzed to assess cardiac activity. **Results:** Beginning at 5 weeks of age, the 4-PBA treated SHR group exhibited significantly reduced HR, and elevated RR, QT, and QTcb intervals. Between the WKY groups, the differences in HR, RR, QT, and QTcb intervals were not significant, but did exhibit a similar trend. 4-PBA seemed to lower heart rate, with greater efficacy in the SHR strain. Additionally, systolic BP recordings are significantly lower in the 4-PBA treated young SHR group than in the non-treated SHR. **Conclusions:** 4-PBA treatment abolished pre-hypertensive tachycardia in the SHR preventing the development of hypertension. 4-PBA may be a promising therapeutic approach against the development of essential hypertension.

BIOMEDICAL ORAL SESSION #2: 10:30 - 10:45 MISSISSAUGA B

Handle region peptide induces adipogenesis in subcutaneous adipose tissue to promote healthy fat distribution

Paul Tan[1,2,5], Catherine Michel[1], Thi M.-D. Nguyen[6], Peter W. Schiller[6], Jolanta Gutkowska[1,3] and Julie L. Lavoie[1,4,5] [1]Centre de Recherche du Centre Hospitalier de l'Université de Montréal (CRCHUM) [2]Department of Biochemistry and Molecular Medicine, [3]Medicine, and [4]Kinesiology of the Université de Montréal, [5]Montréal Diabetes Research Center, [6]Institut de Recherches Cliniques de Montréal, Montréal, Québec, Canada.

Background: We have previously reported that the (pro) renin receptor [(P) RR] is increased in adipose tissue of obese mice. They gained less body weight and had reduced visceral adipose tissue when treated with the handle region peptide (HRP), a (P) RR blocker. Improved insulin sensitivity was also observed although it was unclear through which mechanisms this occurred. Thus, the aim of the present study is to elucidate mechanisms implicated in these beneficial effects. **Methods/Results:** Mice were on a normal or a high fat/high carbohydrate diet for 10 weeks and simultaneously infused with saline or the HRP. Perigonadal fat (PGF), peri-renal fat (PRF), abdominal subcutaneous fat (SCF) and blood were collected in mice at the end of the treatment. Peroxisome proliferator-activated receptor gamma (PPARγ), the master regulator of adipogenesis, and adiponectin, an insulin sensitizer, were evaluated by Western Blot and Real Time PCR. Circulating high molecular weight (HMW) adiponectin and free fatty acids (FFA) were quantified. PPARγ2 was decreased in PRF of control obese mice which was normalized when treated with the HRP. Independently of the diet, PPARγ2 was increased in SCF of mice treated with the HRP compared to control animals. No effect of diet and treatment was observed in PGF. Adiponectin gene expression was decreased only in PGF of obese mice and the HRP increased its expression only in SCF of obese animals. Circulating HMW adiponectin was decreased in obese mice and was normalized with the HRP. Although circulating FFA tended to increase in control obese mice, the HRP normalized FFA level only in obese animals. **Conclusion:** Our data suggest that the HRP favors adipogenesis de novo in SCF to buffer excess FFA from the circulation thus decreasing ectopic deposition of fat in obese mice. Improved insulin sensitivity with the HRP seemed to result from higher circulating HMW adiponectin.

BIOMEDICAL ORAL SESSION #2: 10:45 - 11:00 MISSISSAUGA B

Preferential disposition of radiolabeled phosphate to the vasculature modifies calcium incorporation in experimental chronic kidney disease

Jason G.E. Zelt[1], Kieran Quinn, Bruno Svajger[1], Kim Laverty[1], Rachel M. Holden[3], Michael A. Adams[1]. [1]Department of Biomedical and Molecular Sciences, Queen's University, Kingston, ON, [2]Department of Medicine, University of Toronto, [3]School of Medicine, Queen's University, Kingston, ON.

Background: Pathogenic vascular accumulation of calcium and phosphate (vascular calcification; VC) is a marker of advancing cardiovascular disease (CVD) in chronic kidney disease (CKD). The study sought to determine whether the distribution pattern to cardiovascular tissue for acute, parenteral administration of radiolabelled phosphate and calcium in rats is differentially altered and whether the disposition of the two minerals are linked in experimentally induced CKD (0.25% adenine). **Methods and Results:** Phosphate ($^{33}PO_4$) and calcium (^{45}Ca) disposition, assessed in blood and tissues (36 tissues) 20 minutes after an intravenous infusion of 1) phosphate (i) 100 mM phosphate + $^{33}PO_4$ or (ii) 100 mM phosphate + ^{45}Ca and 2) saline (saline + ^{45}Ca). Immediately post-infusion, $^{33}PO^4$ activity levels in blood were significantly higher (2.3x) in CKD animals and remained significantly elevated (3.5x) above non-CKD 20 minutes post infusion ($p<0.05$). In contrast, there was no difference in ^{45}Ca clearance between CKD and non-CKD animals following the phosphate infusion. The tissue distribution pattern of $^{33}PO_4$ and ^{45}Ca was markedly different between groups following the phosphate challenge. Compared to non-CKD animals, CKD animals had increased i) $^{33}PO_4$ incorporation in the vasculature (4.0x), skeletal muscle (2.7x), and heart (1.8x) and ii) ^{45}Ca incorporation in vasculature (2.2x) and kidney (1.5x). There was no difference in ^{45}Ca clearance between the two CKD groups (saline vs phosphate). Despite this, the phosphate infusion increased (1.7x) the ^{45}Ca incorporation into the vasculature ($p<0.0001$). **Conclusions:** The research reveals that the disposition of circulating phosphate and calcium is dramatically altered in CKD; resulting in preferential deposition of phosphate and calcium into vascular tissue following a parenteral phosphate infusion. A key finding is that the increased vascular ^{45}Ca deposition in CKD is secondary to alterations in phosphate handling. Together, these studies provide compelling evidence that it is phosphate dysregulation at the tissue level that mechanistically underlies the development of calcification. This research is funded by CIHR.

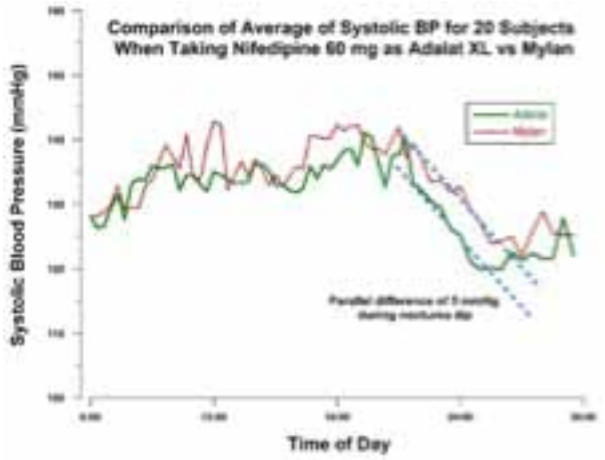
ABSTRACTS
Friday, October 23

CLINICAL/OUTCOMES/POPULATION ORAL SESSION #2: 10:30 - 10:45 MISSISSAUGA C

Differences in 24-h ambulatory blood pressure (ABPM) responses between differing nifedipine osmotic delivery formulations coincide with differences in dissolution profiles

Navdeep Dehar, Robert J. Herman, Kelly B. Zarnke, Ross D. Feldman. University of Calgary, Calgary, AB, & Western University, London, ON.

Background: Pharmacy switches between generic medications happen frequently. Neither clinical response, nor timing of drug release from modified release formulations are directly evaluated in bioequivalence studies. Differences in clinical response to two differing nifedipine modified-release technologies were assessed with ABPM. **Methods:** A 20-patient randomized crossover study of Mylan-nifedipine ER (MyN) vs. Adalat XL (AdN) 60 mg was done. After each 2-week acclimatization period (daily morning dosing), 24-hr ABPM was recorded. Systolic (SBP), both 24 h and terminal 8 h (22:00-06:00 h), and averaged SBP curves over 24 h were compared. A difference in average BP > 2mmHg, which changes stroke risk by 8%, was considered clinically meaningful. **Results:** Mean ± SE 24-h SBP was 132.7 ± 2.4 mmHg with AdN, and 135.0 ± 2.3 mmHg with MyN (p=0.030). ANOVA of terminal 8 h of the dosing interval showed SBP 124.9 ± 2.8 mmHg for AdN and 128.8 ± 2.3 mmHg for MyN (p=0.04). Inter-formulation differences in SBP ranged from -11 to +5. AdN was > 2 mmHg lower than MyN in 10 patients. **Conclusion:** The mean 24-h and terminal 8-h SBP were statistically significantly lower in subjects taking AdN, than when taking MyN. Averaged SBP curves (Figure) when taking MyN were higher in the 2 h after dosing; similar to AdN during daytime; and higher during the terminal phase of the dosing interval. This is consistent with the longer lag time and reduced terminal release observed during in-vitro dissolution of MyN (first-order release design), but not seen with AdN (zero-order design). Because nifedipine has only a 2-h half-life, differences in extent and time distribution of drug delivery, can allow important concentration fluctuations within the 24-h dosing interval. These results suggest that regulatory bodies should not consider formulations that use differing delivery technologies as being comparable or eligible for bioequivalence testing.



CLINICAL/OUTCOMES/POPULATION ORAL SESSION #2: 10:45 - 11:00 MISSISSAUGA C

Effect of the double exposure to adverse psychosocial work factors and high family responsibilities on blood pressure among white-collar working women: A 5-year prospective study

Chantal Brisson, PhD, Axe santé des populations et pratiques optimales en santé, Centre de recherche FRQS du CHU de Quebec City, Quebec, Canada; Alain Milot, MD, MSc, FRCPC, Medicine department, Laval University, Quebec City, Quebec, Canada; Michel Vézina, MD, MPH, FRCPC, - Social and preventive medicine department, Laval University, Quebec City, Quebec, Canada.

Background: Psychosocial word factors of the demand-control (DC) and effort-reward imbalance (ERI) models may contribute to increase BP. Women are more likely to be exposed to these psychosocial factors than men. In addition, women spent approximately twice as much time per week to family responsibilities than men (30.1 compared to 17.5). Multiple roles, such as being a mother and an employee may induce a physiological and a psychological stress leading to cardiovascular health problems. This study aims to evaluate the effect of the double exposure to adverse psychosocial work factors and high family responsibilities on women BP over a 5-year follow-up. **Method/Results:** The study was composed of over 1,000 working women assessed at baseline and 3-year and 5-year follow-ups. Ambulatory BP measures were taken every 15 minutes during a working day. Psychosocial work factors of the DC and ERI models were self-reported by questionnaire using validated scales. Family responsibilities were also self-reported, using items related to "the number of children and their age" and "housework and children care". BP means at baseline and follow-ups were respectively modeled with analyses of covariance. Women having a double exposure to effort-reward imbalance at work and high family responsibilities had higher BP mean level than women not exposed to these factors. Indeed, statistically significant associations have been observed between the double exposure assessed at baseline and fully adjusted BP mean at baseline (diastolic: +2.75 mm Hg), at 3-year follow-up (systolic: +2.22 mm Hg and diastolic: +2.55 mm Hg), and at 5-year follow-up (systolic: +2.94 mm Hg and diastolic: + 3.10 mm Hg). **Conclusion:** A double exposure to effort-reward imbalance at work and high family responsibilities might contribute to elevate women's BP. Also, BP elevations related to this double exposure might persist over several years.

CLINICAL/OUTCOMES/POPULATION ORAL SESSION #2: 11:00 - 11:15 MISSISSAUGA C

Antihypertensive drug initiation versus chronic use and its impact on fracture risk in the elderly: A systematic review and meta-analysis

Debra A. Butt[1,2], Raghad Alharthy[3], Jeevitha Srighanthan[3], George Tomlinson[4], Angela M. Cheung[3,4], [1]The Scarborough Hospital, Department of Family and Community Medicine, [2]University of Toronto, Department of Family and Community Medicine Research Program, [3]University Health Network Osteoporosis Program, 4University of Toronto Department of Medicine, Toronto, Canada.

Background: Previous meta-analyses examining the association of antihypertensive drugs on fracture risk have reported little on drug duration. The purpose of this meta-analysis was to determine how duration of antihypertensive medications impact fracture risk in the elderly. **Method:** We searched Medline, EMBASE, Cochrane, CINAHL and Web of Science until March 11, 2015 for studies evaluating the effect of antihypertensive drugs on fracture risk and reviewed bibliographies of relevant articles. Two independent reviewers extracted data and assessed study quality. Using a random-effects model weighted by inverse variance of the effect size, we determined the pooled adjusted and unadjusted risk ratios (RR) and 95% CIs. Statistical heterogeneity was tested using the I^2statistic. **Results:** Of the 909 citations identified, 13 studies met inclusion criteria and 23 additional studies were retrieved from bibliographies of articles. There were 36 observational studies involving 2,695,266 participants (51-100% female, average age range 65-85 years). Thiazide diuretic initiation (≤ 45 days) was associated with an increased hip fracture risk (RR 1.68, 95% CI 1.27-2.22, 3 studies, I^2=32%) while use ≥ 1 year was associated with a decreased risk (RR 0.85, 95% CI 0.74-0.97, 8 studies, I^2=46%). Angiotensin-converting enzyme inhibitor initiation (≤ 90 days) was associated with a non-significant increased fracture risk (RR 1.18, 95% CI 0.87-1.59, 3 studies, I^2=72%) however, there was no association for ≥ 1 year use (RR 1.10, 95% CI 0.71-1.71, 3 studies, I^2=98%) which was consistent for angiotensin receptor blockers (RR 0.91, 95% CI 0.79-1.05, 3 studies, I^2=71%) and calcium channel blockers (RR 1.01, 95% CI 0.81-1.25, 3 studies, I^2=98%). Chronic beta-adrenergic blocker use was associated with a decreased fracture risk (RR 0.89, 95% CI 0.81-0.98, 7 studies, I^2=77%). **Conclusion:** This meta-analysis suggests that hypertensive seniors have a higher fracture risk during antihypertensive drug initiation and this risk changes with chronic antihypertensive drug use.

ABSTRACTS
Friday, October 23

CLINICAL/OUTCOMES/POPULATION ORAL SESSION #2: 11:15 - 11:30 MISSISSAUGA C

Socioeconomic status and longitudinal change in aortic stiffness: the Whitehall II study

Eric J. Brunner (University College London), Martin J. Shipley (University College London)

Background: The inverse association between socioeconomic status (SES) and cardiovascular disease (CVD) is well documented. Aortic stiffness expressed as aortic pulse wave velocity (PWV) is a strong predictor of cardiovascular disease events. No previous study has longitudinally examined the effect of SES on arterial stiffening over time. **Objective:** This study aimed to examine the association between SES and aortic PWV change over 5 years. **Methods:** The Whitehall II study is a longitudinal cohort study of British civil servants. In the present study, the baseline sample was composed of 3836 men and 1406 women who attended the Phase 9 (2007-09) clinical examination (mean age = 65.5 years). Aortic PWV was measured at Phase 9 and at Phase 11 (2012-13) by applanation tonometry. The mean difference in the 5-year change in PWV was examined according to household income, education, grade level and father's social class, using linear mixed models. **Results:** PWV increase (m/s) over 5 years was higher among participants with lower household income (0.64, 95% confidence interval: 0.38 - 0.90), education (0.28 95% confidence interval: 0.00, 0.56) and grade level (0.44, 95% confidence interval: 0.18 - 0.71), after adjusting for socio-demographic variables, body mass index, alcohol consumption, smoking and other cardiovascular indicators including systolic BP, mean arterial BP and heart rate variability. **Conclusion:** The present study supports the presence of socioeconomic disparities in arterial stiffening progression. Our findings suggest that arterial aging could be an important pathophysiological pathway explaining the impact of SES on CVD.

BIOMEDICAL POSTER SESSION #2: 15:00 - 17:00 MISSISSAUGA FOYER

BMR11 - 4-phenylbutyric acid inhibited salt sensitive hypertension in the model of Dalh salt sensitive rat

Victoria Yum, Rachel Carlisle, Jeffrey Dickhout. Department of Medicine, Division of Nephrology, McMaster University and St. Joseph's Healthcare Hamilton, Hamilton, Ontario, Canada.

Background: Hypertension is defined as the sustained elevation of blood pressure above 140/90 mmHg and is the leading risk factor for death worldwide. Although there are a variety of factors that increase the risk of hypertension, high sodium intake has been identified as a critical factor. Endoplasmic reticulum (ER) stress occurs due to a lack of proteostasis in the ER. Recent work in our laboratory has shown that treatments to restore proteostasis are able to lower blood pressure in hypertension. We hypothesized that the suppression of ER stress with a low molecular chemical chaperone, 4-phenylbutyrate (4-PBA), would reduce salt sensitive hypertension. **Methods:** We utilized the Dahl salt sensitive hypertensive (Dahl S) rat as an established model of salt sensitive hypertension. To induce hypertension, 12 week old male Dahl S rats were placed on a high salt (HS, 8% NaCl) diet for 4-weeks. Another group of animals were pretreated with 4-PBA (1 g/kg/day) one week prior to HS feeding. Dahl S rats fed with a normal salt (NS, 0.4% NaCl) diet were used as control. These animals were implanted with radio telemetry devices (PA-C40 transmitter, Data Sciences International) to acquire blood pressure, heart rate, core body temperature and activity. Ratio telemetry data was analyzed with Ponemah software (Data Sciences International). **Results:** Telemetry data obtained through 24-hour blood pressure collection demonstrated after 4 weeks of HS diet, BP was significantly increased in Dahl S rats, where 4-PBA treatment (1g/kg/day) significantly lowered systolic, diastolic and mean arterial pressures in the HS fed Dahl S rats. Dahl S rats fed with NS diet did not have increase of BP. **Conclusions:** 4-PBA treatment prevented the development of salt sensitive hypertension in the Dahl S rats. This suggests that ER stress plays an important role in the salt sensitive hypertension development.

BIOMEDICAL POSTER SESSION #2: 15:00 - 17:00 MISSISSAUGA FOYER

BMR12 - Targeted deletion of matrix metalloproteinase 2 prevents angiotensin II-induced vascular injury, mediated in part by inhibition of epidermal growth factor receptor phosphorylation in vascular smooth muscle cells

Tlili Barhoumi, Muhammad Oneeb Rehman Mian, Julio C. Fraulob-Aquino, Asia Rehman, Noureddine Idris-Khodja, Pierre Paradis and Ernesto L. Schiffrin, McGill University

Background: Matrix metalloproteinase 2 (MMP2) is involved in cardiovascular disease. Whether MMP2 plays a role in hypertension and vascular damage is unknown. We hypothesized that *Mmp2* knockout will prevent angiotensin (Ang) II-induced blood pressure (BP) rise and vascular injury. **Method/Results:** Ten to 12-week-old male *Mmp2* knockout ($Mmp2^{-/-}$) and wild-type (WT) mice were infused with Ang II (1000 ng/kg/min, sc) for 14 days. Systolic BP was measured by telemetry and mesenteric arteries (MA) endothelial function and vascular remodeling by pressurized myography. Reactive oxygen species (ROS) generation using dihydroethidium staining, vascular cell adhesion protein 1 (VCAM-1) and monocyte chemotactic protein-1 (MCP-1) expression and monocyte/macrophage infiltration by immunofluorescence were determined in aortic wall or perivascular fat (PVAT). Spleen T cells and monocyte profile were assessed by flow cytometry. Vascular smooth muscle cells (VSMCs) were isolated from MA of WT and *Mmp2* knockout mice, stimulated 5 min with 100 nM Ang II and epidermal growth factor receptor (EGFR) phosphorylation measured by Western-Blotting. Ang II increased Systolic BP by 50 mmHg (P0.01), decreased vasodilatory responses to acetylcholine by 70 % (P0.01), induced a ≥1.4-fold increase in media-to-lumen ratio and media cross-sectional area (P0.05), and enhanced MA stiffness (P0.01), as shown by a leftward shift of the stress/strain relationship, in WT mice. Ang II increased aortic ROS generation 25-fold in WT mice (P0.01). Ang II increased aortic VCAM-1 and MCP-1 expression 3- and 6-fold, respectively, and PVAT monocyte/macrophage infiltration 8-fold in WT (P0.05). Ang II increased ≥1.8-fold spleen activated $CD4^{+}CD69^{+}$ and $CD8^{+}CD69^{+}$ T cells and pro-inflammatory $Ly\text{-}6C^{hi}$ monocytes (P0.001) in WT mice. Ang II increased EGFR phosphorylation 2-fold in VSMCs. *Mmp2* knockout prevented or reduced all of the above except SBP elevation (P0.05). **Conclusion:** MMP2 plays an important role in Ang II-induced vascular injury, which could be mediated at least in part through VSMC EFGR activation.

BIOMEDICAL POSTER SESSION #2: 15:00 - 17:00 MISSISSAUGA FOYER

BMR13 - Plasma oxylipins as potential diagnostic markers and therapeutic targets for cardiovascular and cerebrovascular events in patients with peripheral arterial disease

Stephanie P.B. Caligiuri,[1-3] Harold Aukema,[1,5] Amir Ravandi,[2,3,4] Randy Guzman,[4] and Grant N. Pierce[1-3] [1]Canadian Centre for Agri-food Research in Health and Medicine (CCARM) and [2]the Institute of Cardiovascular Sciences, St. Boniface Hospital Research Centre, and [3]the Departments of Physiology, [4]Internal Medicine, and [5]Human Nutritional Sciences, University of Manitoba, Winnipeg, Canada.

Background: Patients with peripheral arterial disease (PAD) are at an elevated risk for cardiovascular and cerebrovascular events. Diagnostic markers and novel therapeutic targets would be of great benefit in their treatment and quality of life. Oxylipins, bioactive molecules that include the eicosanoids but also the novel octadecanoids and docosanoids, may be a target as they regulate inflammation and vascular tone. However, their relationship to cardiovascular/cerebrovascular events in patients with PAD has yet to be determined. **Methods:** Plasma oxylipins, the presence of past/current cardiovascular and cerebrovascular events, and statistics were analyzed by HPLC-MS/MS technology, patient record assessment, and logistic regression, respectively (n=100). **Results:** The prevalence of transient ischemic attacks (TIA), cerebrovascular accidents (CVA), angina, and myocardial infarctions (MI) was 16%, 10%, 16%, and 24%, respectively. Forty-three plasma oxylipins were quantified, of which 8 were significantly associated with events. For example, 6-keto prostaglandin$F_{1\alpha}$ was protective against TIAs with an odds ratio of 0.066 (0.061, 0.072). Every 1 unit increase in plasma 16-hydroxyeicosatetraenoic acid (HETE) increased the odds of angina and CVA prevalence by 9.1 (8.5, 9.7) and 55.1 (50.9, 59.5) fold, respectively. Plasma oxylipin profiles of patients with incident MIs and CVAs were also analyzed and compared to the PAD population naive of these events. The patient with an incident CVA had ≥ 3-fold higher concentrations of 14-hydroxydocosahexanoic acid, prostaglandin $F_{2\alpha}$, and 16-hydroxyeicosatetranoic acid (HETE) 5 weeks prior to his CVA versus the naive population. Those with an incident MI (n=3) tended to have lower concentrations of many plasma oxylipins prior to the MI except for prostaglandin $F_{2\alpha}$ which was 1.5-fold higher versus the naive population. **Conclusion:** Specific oxylipins are strongly associated with the prevalence of cardiovascular and cerebrovascular events in patients with PAD. Therefore, oxylipins may be useful diagnostic/risk markers or therapeutic targets for cardiovascular and cerebrovascular events.

ABSTRACTS
Friday, October 23

BIOMEDICAL POSTER SESSION #2: 15:00 - 17:00 MISSISSAUGA FOYER

BMR14 - The extent of vascular remodeling following injury is dependent on the balance between ERα and GPER

Qingming Ding[1], Yasin Hussain[2], Robert Gros[2], Jozef Chorazyczewski[2] J. Geoffrey Pickering JG[2] and Ross D Feldman[1]. [1]Department of Medicine, Memorial University of Newfoundland, St. John's, NL [2]Departments of Medicine, Physiology and Pharmacology, Robarts Research Institute, London, ON.

Background: Estrogens are important physiological and pathophysiological regulators of cardiovascular (CV) function. The traditional view of the mechanism for these CV effects has focused on the activation of "classical" steroid receptors (i.e., estrogen receptors-ER). However, recent studies have elucidated the mechanism of estrogen's CV effects mediated via GPER (aka GPR30). We are beginning to appreciate that, to understand the effect of estradiol in CV regulation, one must understand the balance between GPER- and ER- mediated effects. In vascular smooth muscle cells, estrogen-mediated regulation of apoptotic cell death and proliferation is divergently regulated by activation of ER vs. GPER. However, the significance of this divergence in the in vivo regulation of vascular growth processes was unknown. To determine the importance of the balance between GPER vs. ER expression in regulation of vascular growth we compared the effects of increasing GPER expression vs. decreasing ERα expression in modulating the response to vascular injury in a rat carotid ligation model. **Results:** Under baseline conditions, 1 week following carotid ligation and endothelial disruption with distilled water installation there was an 51±7% increase in medial thickness (n=3), paralleling neointimal proliferation. Notably, vascular injury also resulted in downregulation of vascular smooth muscle GPER mRNA content without downregulation of ERα content. Recovery of vascular GPER expression using an adenoGPER construct resulted in a significant attenuation of the medial hypertrophic response (Control (adenoGFP): 53±4% increase [n=11], GPER: adenoGPER: 33±3% [n=5], $p<0.05$). **Conclusions:** in these studies we demonstrate that, in vivo, the balance between GPER and ERα is a significant regulator of vascular remodeling following injury. Receptor-specific modulation of estrogen's growth regulatory effects may be an important new approach in modifying the extent of vascular remodeling in both acute settings like vascular injury and perhaps in longer term models of regulation like hypertension.

BIOMEDICAL POSTER SESSION #2: 15:00 - 17:00 MISSISSAUGA FOYER

BMR15 - Therapeutically targeting the cystic fibrosis transmembrane regulator (CFTR) in cerebrovascular dysfunction associated with subarachnoid hemorrhage (SAH)

J. Fares[1,2], K. Yagi[3], M. Sumiyoshi[3,4], J. Kroetsch[1,2], D. Dinh[1,2], D. Lidington[1,2], L. R. Macdonald[4], St-S. Bolz[1,2] Department of Physiology[1], University of Toronto, Ontario, Canada; Toronto Centre for Microvascular Medicine[2], Li Ka Shing Knowledge Institute, St. Michael's Hospital, Ontario, Canada; Department of Neurosurgery[3], University of Tokushima Graduate School, Tokushima, Japan; Department of Neurosurgery[4], St Michael's Hospital, Ontario, Canada.

Background: Subarachnoid hemorrhage (SAH) is a devastating type of stroke, in which an intracranial bleed initiates delayed cerebrovascular constriction and subsequent ischemia. Current therapeutic strategies attempt to alleviate the cerebrovascular constriction; however, their lack of specificity disrupts cerebral autoregulation and consequently, limits their overall efficacy. We have identified an inflammatory mechanism that pathologically augments myogenic vasoconstriction (an intrinsic mechanism of resistance arteries that matches flow resistance to the prevalent transmural pressure): at its core, tumor necrosis factor α (TNFα) down-regulates the cystic fibrosis transmembrane conductance regulator (CFTR) and thereby enhances pro-constrictive sphingosine-1-phosphate (S1P) signaling. **Methods:** Induction of SAH via a stereotactic surgical procedure is used to mimic the hemorrhagic stroke. Pressure myography is employed in the assessment of myogenic autoregulation, MRI (FAIR) for cerebral blood flow, Modified Garcia Neurological Test for behavioural assessment, and histology for brain tissue apoptosis. **Results:** We demonstrate that SAH down-regulates CFTR protein expression in cerebral arteries by a TNFα-dependent mechanism; CFTR mRNA expression is unaltered, indicating the involvement of a post-translational effect. As our model predicts, reducing CFTR activity in control cerebral olfactory arteries (100nM CFTR (inh)-172 in vitro) enhances myogenic tone and thus, mimics the SAH phenotype. Therapeutically increasing microvascular CFTR expression in vivo (3mg/kg/day C-18) normalizes the elevated myogenic tone in SAH; olfactory arteries from sham-operated mice are not affected. Since CFTR is more highly expressed in cerebral arteries relative to skeletal muscle arteries, C-18's effects may be localized to the cerebral microcirculation. We conclude that C-18 specifically remedies the enhanced vasoconstriction in SAH. CFTR **Conclusions:** We open the doors to a new therapeutic target that can be used to normalize the enhanced constriction by leaving the adaptive auto regulatory mechanism intact, therefore preventing ischemic injury in SAH.

BIOMEDICAL POSTER SESSION #2: 15:00 - 17:00 MISSISSAUGA FOYER

BMR16 - Endothelin-1 overexpression exaggerates type 1 diabetes-induced endothelial dysfunction by altering oxidative stress balance

Noureddine Idris-Khodja[1], Sofiane Ouerd[1], Muhammad Oneeb Rehman Mian[1], Jordan Gornitsky[1], Tlili Barhoumi[1], Pierre Paradis[1], Ernesto L. Schiffrin[1,2] [1]Vascular and Hypertension Research Unit, Lady Davis Institute for Medical Research and [2]Department of Medicine, SMBD-Jewish General Hospital, McGill University, Montréal, QC, Canada.

Objective: Increased endothelin (ET)-1 expression has been shown to cause endothelial dysfunction and oxidative stress. Plasma ET-1 is increased in patients with diabetes mellitus. Since endothelial dysfunction often precedes vascular complications in diabetes, we sought to determine whether ET-1 contributes to diabetes-induced endothelial dysfunction. We hypothesized that overexpression of ET-1 in the endothelium will exaggerate diabetes-induced endothelial dysfunction. **Method/Results:** Diabetes was induced by streptozotocin treatment (STZ,55 mg/kg/day, ip) for 5 days in 6 week old male wild-type (WT) mice and in mice overexpressing human ET-1 restricted to the endothelium (eET-1). Mice were studied 14 weeks later. Blood-glucose tests confirmed STZ-induced diabetes as indicated by an increase in glycemia in both groups (P0.05). Plasma ET-1 measured by ELISA was increased in vehicle- (15.9±4.6 vs 0.6±0.04 pg/mL, P0.05) and STZ-treated eET-1 (4.9±0.6 vs 0.8±0.1 pg/mL, P0.05) compared to respective WT controls. Mesenteric artery (MA) endothelium-dependent vasodilatory responses to acetylcholine assessed by pressurized myography were reduced 27% by diabetes in WT (P0.05), and further decreased by 20% in eET-1 (P0.05). Mitochondrial superoxide production quantified by fluorescence imaging of MA sections incubated with MitoSOX™ Red reagent was increased 1.8-fold by diabetes in WT (P0.05) and further augmented by 31% in eET-1 (P0.05). RNA was extracted from MA and reverse transcription-quantitative PCR performed to examine gene expression. Nitric oxide synthase 3 (Nos3) expression was increased by 43% in vehicle-treated eET-1 compared to WT (P0.05). Diabetes reduced Nos3 expression in eET-1 by 31% (P0.05) but not in WT. Diabetes caused an increase in superoxide dismutase 1 (Sod1 52%) and Sod2 (32%) expression in WT (P0.05) but not in eET-1. **Conclusions:** Increased levels of ET-1 exaggerate diabetes-induced endothelial dysfunction. This may be caused by a decrease in Nos3expression, an increase in mitochondrial oxidative stress and a decrease in antioxidant capacity.

BIOMEDICAL POSTER SESSION #2: 15:00 - 17:00 MISSISSAUGA FOYER

BMR17 - Heme oxygenase and pancreatic regeneration

Joseph Fomusi Ndisang; University of Saskatchewan College of Medicine, Departments of Physiology.

Background: The destruction of pancreatic beta cells and ensuing insulin deficiency in type-1 diabetes remains a great health challenge. Although we had previously shown that treatment with the heme oxygenase (HO) inducer, hemin, improved pancreatic lesion in streptozotocin-induced diabetes, the mechanism are still unclear. Here, we report the effects of hemin on proteins of regeneration during pancreatic repair in streptozotocin-induced diabetes. **Method/Results:** Histology/morphology, spectrophotometry, enzyme-immunoassay (EIA), enzyme-linked immunosorbent assay (ELISA) and Western-immunoblotting were used. Streptozotocin was used to induce diabetes, while heme oxygenase was upregulated with hemin or inhibited with chromium mesoporphyrin. Treatment with the hemin suppressed pancreatic histopathological lesions such as vacuolization, interstitial edema, mononuclear cell infiltration and fibrosis and correspondingly enhanced several proteins of pancreatic regeneration including Oct3/4, Pax-2, beta-catenin and ISL-1 as well as markers of stem cell markers such as cKit and Sca-1, suggesting the involvement of stem cells and proteins of regeneration during pancreatic repair/regeneration in streptozotocin-induced diabetic animals. Interestingly, the restoration of pancreatic morphology was accompanied by increased production of insulin, and the potentiation of proteins implicated in insulin signal transduction cascade such as insulin-receptor substrate (IRS-1), IRS-2, phosphatidylinositol-3-kinase (PI3K) and GLUT4 in skeletal muscles and the liver from streptozotocin-induced diabetic animals. Furthermore, hemin attenuated oxidative stress and inflammation in the pancreas, skeletal muscles and liver. These effects were nullified by the HO-inhibitor, chromium mesoporphyrin. **Conclusion:** Collectively, our data suggest that upregulating the HO-system with hemin is a useful strategy to potentiate Oct3/4, Pax-2, beta-catenin and ISL-I during the restoration of pancreatic morphology after insult in streptozotocin-induced diabetic animals. Importantly, the restored pancreas produced adequate levels of insulin and improved glucose metabolism.

ABSTRACTS
Friday, October 23

BIOMEDICAL POSTER SESSION #2: 15:00 - 17:00 MISSISSAUGA FOYER

BMR18 - Stromal interaction molecule-1 mediates Angiotensin-II-induced expression of early growth response protein-1 in vascular smooth muscle cells

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Background: The early growth response protein 1 (Egr-1) is a zinc finger transcription factor that has been suggested to regulate the expression of genes linked with inflammation and cell cycle regulation. An upregulation of Egr-1 expression has been reported in models of atherosclerosis and intimal hyperplasia. Various vasoactive peptides and growth promoting stimuli have been shown to induce the expression of Egr-1 in VSMC. Angiotensin-II (Ang-II) is a key vasoactive peptide that has been implicated in the pathogenesis of vascular diseases. Ang-II elevates the intracellular level of calcium through activation of store operated calcium entry involving inositol-3-phosphate receptor (IP3R)-coupled depletion of endoplasmic reticular calcium and stromal interaction molecule 1 (STIM-1). However, an involvement of IP3R/STIM-1- induced alteration in calcium pathway in Ang-II-induced Egr-1 expression in VSMC remains unexplored. Therefore in the present studies we have examined the role of Ang-II-induced calcium release in Egr-1 expression in VSMC and investigated the contribution of STIM-1 in this process. **Methods and results:** Calcium chelation with BAPTA-AM as well as pharmacological blockade of IP3R with 2-aminoethoxydiphenyl borate (2-APB) decreased Ang-II-induced calcium release measured in cells loaded with Fura-2. Consistent with this, both BAPTA-AM and 2-APB attenuated Ang-II-induced enhanced expression of Egr-1 protein and mRNA levels. Furthermore, RNA interference targeting STIM-1 significantly abrogated STIM-1 protein and mRNA expression and resulted in an attenuation of Ang-II-induced Egr-1 expression. **Conclusion:** In summary, our data establish that Ang-II-induced Egr-1 expression is mediated by STIM-1 and calcium release in A-10 VSMC. (Supported by a grant from CIHR)

BIOMEDICAL POSTER SESSION #2: 15:00 - 17:00 MISSISSAUGA FOYER

BMR19 - Origins of adventitial Sca1+ progenitor cells

Sarah K. Steinbach[1,2], Angela Li[7], Rickvinder Besla[6], Martha H. Carruthers[2], Eric A. Shikatani[6], Adam P. Johnston[9], Freda D. Miller[9], Clinton S. Robbins[3,6-8,10] and Mansoor Husain[1,2,4-6,8,10] [1]McEwen Centre for Regenerative Medicine, Divisions of [2]Experimental Therapeutics and [3]Advanced Diagnostics, Toronto General Research Institute, 101 College St., Toronto, Ontario, Canada, M5G-1L7. Departments of [4]Medicine, [5]Physiology, [6]Laboratory Medicine & Pathobiology, [7]Immunology and the [8]Heart and Stroke Richard Lewar Centre of Excellence, University of Toronto, 1 Kings College Circle, Toronto, Ontario, Canada, M5S-1A8. [9]Hospital for Sick Children, 686 Bay St., Toronto, Ontario, Canada, M5G-0A4. [10]Peter Munk Cardiac Centre, University Health Network, 200 Elizabeth St., Toronto, Ontario, Canada M5G-2C4.

Background: Adventitial Sca1$^+$ progenitor cells are abundant vascular smooth muscle progenitor cells that play a role in atherosclerosis and in neointima formation during vascular injury. Currently, the origins of adventitial Sca1$^+$ progenitor cells are unknown and shown not to be neural crest or bone marrow derived. The purpose of this study was to identify a novel stem cell present in the adult murine aorta that gives rise to vascular smooth muscle cells (VSMCs) and to identify the origins of adventitial Sca1$^+$ progenitor cells. **Methods/Results:** Using a Myf5-*Cre* animal crossed to a floxed tdTomato reporter, which labels the somitic mesoderm, we were able to culture stem cells derived from the 8-12 week old mouse aorta in defined serum-free stem cell medium. These stem cells displayed multilineage potential and could differentiate into Calponin+ VSMCs and S100β+ glial cells *in vitro*. In accord with their multilineage potential, these stem cells were positive for the stem cell marker Sox2 by indirect immunofluorescence. Intriguingly, a significant proportion of Sca1$^+$ progenitor cells were lineage marked *in vivo* suggesting a somitic origin for Sca1$^+$ progenitor cells in the aorta. In order to determine at which stage Sca1$^+$ progenitor cells were specified *in vivo*, we acquired a tamoxifen inducible Myf5-*Cre*^{ER} mouse. We demonstrate that adventitial Sca1$^+$ progenitors are specified during early somitogenesis starting at E8.5. Since the stem cells cultured *in vitro* also expressed Sox2, we wanted to determine if adult Sox2 stem cells in the adult aorta could also give rise to Sca1$^+$ adventitial progenitor cells. Using a Sox2-Cre^{ERT2} mouse crossed to a floxed tdTomato reporter, we demonstrated that approximately 34±5% of adventitial Sca1 progenitor cells were also derived from Sox2 stem cells in the aortas of adult animals. **Conclusion:** Adventitial Sca1 progenitor cells are derived from the somitic mesoderm. They are also replenished *de novo* through Sox2$^+$ stem cells in the adult mouse aorta. Further studies will be performed to determine the physiological significance of Sox2 stem cells in disease.

BIOMEDICAL POSTER SESSION #2: 15:00 - 17:00 MISSISSAUGA FOYER

BMR20 - The contribution of organ fibrosis to hypertension through the unfolded protein response pathway

Jeffrey Dickhout, Department of Medicine, Division of Nephrology, McMaster University and St. Joseph's Healthcare Hamilton, Hamilton, Canada.

Background: Hypertension leads to end-organ damage characterized by the fibrosis of the damaged tissue. Fibrosis is an aberrant wound-healing response that leads to the excessive deposition of scar tissue. The blood vessels and kidneys are particularly susceptible to the effects of hypertension, and this may in turn drive the disease progression. TGF-β is a profibrotic cytokine that can induce vascular smooth muscle cells (VSMCs) and fibroblasts to acquire an extracellular matrix (ECM)-secreting phenotype, and is present in tissues undergoing fibrotic remodeling. The unfolded protein response (UPR) is a conserved cellular pathway that allows cells to adapt to an accumulation of unfolded proteins within the endoplasmic reticulum. We hypothesized that the inhibition of this pathway can disrupt the secretion of fibrotic proteins from VSMCs and fibroblasts in vitro. **Method/Results:** Sprague-Dawley renal fibroblasts and aortic smooth muscle cells were treated with TGF-β1, which led to the induction of UPR markers GRP78 and GRP94. TGF-β induced the differentiation of renal fibroblasts into myofibroblasts, as shown by the elevation of α-smooth muscle actin. Supplementation of the growth medium with 200 μM L-ascorbic acid resulted in the secretion of Type I collagen from the renal fibroblasts, which was attenuated by the administration of the IRE1α inhibitor STF-083010. The chemical chaperone 4-phenylbutyrate also reduced the expression of Type I collagen from these cells. **Conclusions:** Our results suggest that UPR activation is required for VSMCs and fibroblasts to acquire a synthetic and secretory phenotype. The UPR presents a plausible target for the prevention of tissue fibrosis.

CLINICAL/OUTCOMES/POPULATION POSTER SESSION #2: 15:00 - 17:00 MISSISSAUGA FOYER

CR13 - The expanding role of Microbiota: from gastrointestinal disorders to blood pressure control

Omar Albassam, KAUH, Jeddah, KSA

Introduction: The role of microbiota in gastrointestinal disease is probably accepted now based on evidence. An overwhelming increase in the publications related to the topic is noticed in the last years. However, the role of microbiota in modifying blood pressure is just emerging. **Methods:** We used PubMed search engine looking up for the terms of "gut microbiota", "hypertension"," blood pressure" (BP). There were a total of 48 publications. We excluded those not related to hypertension. We selected and reviewed 6 publications that were most relevant to the title of our paper. **Results:** Normally the gut microbiota lives in a healthy gut environment (symbiosis). When this relationship is altered, the balance will be shifted toward unhealthy state (dysbiosis). A change in microbiota population will arise where Fermicutes genera increases and Bacteriodes genera decreases. Local and systemic inflammatory response will be triggered. Agents such as SCFA (Small Chain Fatty Acids) will be released into systemic circulation. These SCFA will act on certain receptors called olfactory receptor (Olfr78) and Gr41. Stimulating Olfr78 receptors in the kidneys will stimulate renin secretion and raise BP. Stimulating Gr41 in the vessels will lower BP. The resultant effect will be either an increase or a decrease in blood pressure (BP). Treatment with probiotics or antibiotics (minocycline) may restore the normal gut microbiota balance. This may suppress the inflammatory response and normalize the blood pressure. **Conclusions:** The relationship between microbiota and hypertension is still in its infancy. However, the emerging new evidence mostly in animal studies is promising. The gut microbiota may be a new player in hypertension control. The Fermicutes/ Bacteriodes ratio may be utilized as a new marker for hypertension. A novel therapy for the treatment of hypertension may arise in the form of probiotics and antibiotics (minocycline). However, we still need long term human trials to confirm these emerging preliminary findings.

ABSTRACTS
Friday, October 23

CLINICAL/OUTCOMES/POPULATION POSTER SESSION #2: 15:00 - 17:00 MISSISSAUGA FOYER

CR15 - The effect of short - versus long - acting antihypertensives on blood pressure variability

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Introduction: Hypertension (HTN) affects 19.7% of Canadian adults. HTN is a common modifiable risk factor for cardiovascular and cerebrovascular disease (CVD). Intra-individual blood pressure (BP) variability is increasingly viewed as a better predictor for cardiovascular and cerebrovascular disease than average BP. Despite the numerous and effective treatment options available, HTN remains a public health concern worldwide because of inadequate BP control rates. **Methods:** Data from medical charts of patients from clinics of a tertiary health care centre were collected. Using 24h ambulatory BP measurements, parameters of diurnal and nocturnal BP as well as BP variability (BPV) were calculated and analyzed in terms of the duration of action (determined by the half-life and trough-to-peak ratio) of used antihypertensives (antiHTN), as well as their relative dosage. **Results:** 321 patients were identified. The cohort was mainly male (53%) and had a mean age of 61.47 ± 15.32 years. Despite a high treatment rate (60% using 1-3 antiHTN), day, night, and 24-hour systolic BP (SBP) was controlled in only 40% of the cohort. The variability of the observed effects across primary antiHTN classes do not show a better BP or BPV control with longer acting drugs ($p>0.05$). Nocturnal SBP dipping was 70% uncontrolled although other parameters of BP were controlled, and a combination of antiHTN was given. **Conclusion:** Certain long-acting or high dose antiHTN may improve control of ambulatory BP parameters, but not nocturnal BP or BPV.

CLINICAL/OUTCOMES/POPULATION POSTER SESSION #2: 15:00 - 17:00 MISSISSAUGA FOYER

CR16 - Effect of antihypertensive medications on bone mineral density: A systematic review and meta-analysis

Jonathan Hwang[1], Jeevitha Srighanthan[2], Richard Leu[1], George Tomlinson[3], Debra A. Butt[4,5]; [1]University of Western Ontario, [2]University Health Network Osteoporosis Program, [3]University of Toronto Department of Medicine, [4]The Scarborough Hospital, Department of Family and Community Medicine, [5]University of Toronto, [5]Department of Family and Community Medicine Research Program; Toronto, Canada.

Background: While there have been narrative reviews on the relationship between antihypertensive medications and bone mineral density (BMD), a systematic meta-analysis has yet to be performed. The purpose of this systematic review and meta-analysis was to determine the effects of antihypertensive medications on BMD. **Methods:** A computerized search of the published literature was conducted using PubMed, EMBASE, Web of Science, Cochrane, and CINAHL from inception to April 30, 2015 to identify studies on antihypertensive medications (thiazides, beta-adrenergic blockers (BBs), calcium channel blockers (CCBs), angiotensin-converting enzyme inhibitors (ACEIs) and angiotensin receptor blockers (ARBs)) and BMD. Studies were excluded if they did not measure BMD by dual-energy X-ray absorptiometry. Summary estimates were determined using the random effects model of DerSimonian and Laird with [95% CI]s; heterogeneity was assessed using the I^2 statistic. **Results:** Of the 2390 citations identified, 12 studies met the inclusion criteria and 9 additional articles were identified from the bibliographies of qualifying articles. There were 5 randomized controlled trials (RCTs) with 754 participants (mean age 58-68 years, 73% female) and 16 observational studies with 44,887 participants (mean age 61-77 years, 63% female). In RCTs, taking thiazides for 2-3 years resulted in a non-significant increase of 0.58% [0.12-1.05] in lumbar spine (LS) BMD compared to controls. In observational studies, taking thiazides was associated with a mean difference of 0.03 g/cm^2 [-0.00-0.05] in LS BMD, 0.02 g/cm^2[-0.00-0.03] in total hip (TH) BMD and 0.02 g/cm^2 [0.01-0.04] in femoral neck (FN) BMD. Taking BBs was associated with a mean difference of 0.05 g/cm^2 [0.03-0.07] in LS BMD, 0.02 g/cm^2 [0.01-0.03] in TH BMD and 0.03 g/cm^2 [0.02-0.04] in FN BMD. Taking ACEIs was associated with a mean difference of 0.03 g/cm^2 [0.02-0.04] in TH BMD and 0.03 g/cm^2 [0.02-0.04] in FN BMD. **Conclusions:** This meta-analysis suggests that antihypertensive medications such as thiazides, BBs and ACEIs may have beneficial effects on BMD.

CLINICAL/OUTCOMES/POPULATION POSTER SESSION #2: 15:00 - 17:00 MISSISSAUGA FOYER

CR17 - Does the risk of falls depend on duration of antihypertensive drug use in the elderly? A systematic review and meta-analysis

Richard Leu[1], Jonathan Hwang[1], Jeevitha Srighanthan[2], George Tomlinson[3], Angela M. Cheung[2,3], Debra A. Butt[4,5]. [1]University of Western Ontario, [2]University Health Network Osteoporosis Program, [3]University of Toronto Department of Medicine, [4]The Scarborough Hospital, Department of Family and Community Medicine, [5]University of Toronto, Department of Family and Community Medicine Research Program; Toronto, Canada.

Background: Previous meta-analyses examining antihypertensive drugs and falls risk have scant information on drug duration. The purpose of this meta-analysis was to examine the effect of antihypertensive drug duration on falls risk in the elderly. **Methods:** We searched Medline, EMBASE, Cochrane, CINAHL and Web of Science until April 29, 2015 for studies assessing the effect of antihypertensive drug therapy on risk of falls and reviewed bibliographies of qualifying articles. Using a random-effects model weighted by inverse variance of the effect size, we determined the pooled adjusted risk ratios with 95% CIs. Statistical heterogeneity was assessed using the I^2 statistic. **Results:** Of the 4709 citations identified, 10 studies met inclusion criteria and 9 articles were retrieved from bibliographies of articles. There were 19 observational studies involving 584,391 participants (31-100% female, average age range 69-84 years). The risk of falls was increased during antihypertensive drug initiation (≤ 45 days) in users of thiazide diuretics (RR 2.21, 95% CI 1.57-3.13, 3 studies, I^2=53%), angiotensin-converting enzyme inhibitors (ACEIs) (RR 1.48, 95% CI 1.13-1.94, 3 studies, I^2=54%), beta-adrenergic blockers (BBs) (RR 1.67, 95% CI 1.44-1.94, 3 studies, I^2=0%) and angiotensin receptor blockers (ARBs) (RR 1.21, 95% CI 0.82-1.78, 4 studies, I^2=71%), although not significant. Calcium channel blocker (CCB) initiation was not associated with an increased falls risk (RR 0.77, 95% CI 0.30-1.99, 3 studies, I^2=85%). However, when antihypertensive drugs were used for ≥ 1 year, there was no association with risk of falls in users of ACEIs (RR 0.97, 95% CI 0.87-1.09, 5 studies, I^2=30%), ARBs (RR 0.98, 95% CI 0.90-1.07, 3 studies, I^2=0%), CCBs (RR 0.99, 95% CI 0.86-1.14, 5 studies, I^2=58%) and BBs (RR 1.04, 95% CI 0.95-1.14, 6 studies, I^2=7%). **Conclusion:** Hypertensive elderly patients may have a higher falls risk during antihypertensive drug initiation that diminishes with chronic use.

CLINICAL/OUTCOMES/POPULATION POSTER SESSION #2: 15:00 - 17:00 MISSISSAUGA FOYER

CR18 - Validity of health administrative database definitions for hypertension: A review

Romina Pace[1], Elham Rahme[1,2,3], Kaberi Dasgupta [1,2,3]. [1] Department of Medicine, McGill University, [2] Research Institute of the McGill University Health Centre, [3] Division of Clinical Epidemiology, McGill University.

Background: Health administrative data are frequently used for hypertension surveillance. We aimed to determine the accuracy of various claim based algorithms for hypertension reported in the literature. **Methods:** We conducted a preliminary literature review in Medline using MeSH terms that included: hypertension, administrative databases, and validity. The studies retained were required to report the specificity and sensitivity of the algorithms investigated. We excluded definitions that required access to prescription information given that such data are not always available. **Results:** 5 studies were deemed relevant and reviewed. 1 study utilized survey data, 3 studies utilized physician records and one study used both as a "gold standard" comparison to claim based definitions. The prevalence of hypertension based on the "gold standard" in these studies ranged from 15.3 %-50.1%. Algorithms included: (1) 1 physician billing claim in 3 years,(2) 2 physician billing claims in 3 years, (3) 2 physician billing claims for hypertension within 2 years or 1 hospitalization with a hospital discharge diagnosis of hypertension, and (4) 2 physician billing claims or 1 hospital discharge in 3 years. We compiled the data, when available, for these definitions across the studies retained. The sensitivities of these 4 algorithms ranged from 0.64- 0.85 and specificities were between 0.75-0.95. NPV and PPV were between 0.75-0.82 and 0.86-0.95, respectively. The kappa values in all studies showed moderate to substantial agreement (k=0.45-0.72) between claim based definitions and the "gold standard". The use of only one billing claim was less specific than the algorithms using 2 billing claims which demonstrated similar sensitivity and specificity. **Conclusion:** All algorithms requiring 2 physician billing claims are sufficiently sensitive and specific for research purposes. The addition of hospital discharge diagnosis to physician outpatient billing information does not increase sensitivity or specificity.

ABSTRACTS
Friday, October 23

CLINICAL/OUTCOMES/POPULATION POSTER SESSION #2: 15:00 - 17:00 MISSISSAUGA FOYER

CR19 - New hypertensives in a tertiary care hospital in Sri Lanka: The epidemiology

Kushalee P Jayawickreme[1], Madhushanka Ekanayake[1], Samath D Dharmaratne [2],Department of Medicine[1], Department of Community Medicine[2], Faculty of Medicine, University of Peradeniya,Sri Lanka.

Background: Hypertension is a frequently seen non-communicable disease which currently affects 1 in 4 American adults and 90% of individuals during their lifetime. Epidemiological studies of hypertension have not been carried out in Sri Lanka in the recent years. Therefore, we conducted this study to describe the epidemiology of newly diagnosed hypertensives attending the clinic in the Teaching Hospital, Peradeniya, Sri Lanka. **Methods/Results:** This cross sectional descriptive study was conducted at the Teaching Hospital, Peradeniya, Sri Lanka in 2014. All people attending the hypertensive clinic through a referral and diagnosed as a hypertensive for the first time according to the JNC 7 report categorization were included in the study. Data was collected through an interviewer administered structured questionnaire. Ethical clearance was obtained from the Institutional Ethical Review Committee of the Faculty of Medicine, University of Peradeniya. Data was entered into an Excel data sheet and was analyzed by SPSS statistical software. 280 people were diagnosed as new hypertensives and with a mean age of 57.8 years (SD=12.3 years). The majority was females (65.8%) and married (89.4%). 62.9% were diagnosed as having stage II disease with the measured BP above 159/99. They presented with a mean systolic BP of 161.9 mm HG (SD=20.6 mm HG) and a mean diastolic BP of 96.7 mm HG (SD=11.4 mm HG). The mean SBP and the mean DBP at the first visit were not statistically significantly different by sex (p=0.588 and p=0.856). **Conclusions:** As the majority had higher BP on the first diagnosis, measures to increase the probability of detecting hypertension earlier would be useful to reduce morbidity and mortality associated with hypertension.

CLINICAL/OUTCOMES/POPULATION POSTER SESSION #2: 15:00 - 17:00 MISSISSAUGA FOYER

CR20 - ASA, Aspirin use to prevent cardiovascular and cerebrovascular events

Samia Rizk

Objective: To investigate the use of aspirin in patients with essential hypertension complicated with different risk factors and clinical conditions to prevent cerebrovascular and cardiovascular events. **Method:** 300 people were chosen, face to face questionnaire; patients with essential hypertension were assessed for aspirin consumption. **Results:** Total of 300 patients with hypertension were enrolled; 153 (51%) were male and other 147 (49%) were female. The mean age was 62.21 years (men aged 50 and above or women aged 60 and above), mean blood pressure was 142.8/86 mm Hg, and mean hypertension course was 10.46 years. A total of 105 (35%) patients were taking aspirin, of which 88 % of patients were using aspirin dose 75–150 mg/day, 8 % less than 75 mg daily and 1.5 % more than 150 mg daily. Only 1.08% who had used aspirin withdrew. Common causes of the withdrawal were as follows: 43.2% for fear of potential long-term side effects, 22.5% for discomfort after taking 17.2% for economic reasons 15.4% for no doctors' re-prescription 1.8% for inconvenience in prescription.
Out of the 105 candidates, 55 of the cases had diabetes, of whom 22 (40 %) were taking aspirin. 10 of patients had dyslipidemia, of whom 3 (30%) were taking aspirin. 10 patients had coronary heart diseases, only 8 patients (80 %) were taking aspirin. 30 cases complicated with ischemic stroke, only 18 cases (60%) were taking aspirin. **Conclusion:** Most of the patients are taking an appropriate dose of aspirin. There are still a considerable number of patients with indications for aspirin who did not take it. We should strengthen the education of patients and physicians to increase the use of aspirin to prevent cardiovascular and cerebrovascular events.

CLINICAL/OUTCOMES/POPULATION POSTER SESSION #2: 15:00 - 17:00 MISSISSAUGA FOYER

CR21 - Epidemiological profile of hypertension at the Great Lakes Regional Hospital

Louise Nzigire

Background & Objective: High blood pressure is the second morbid factor decreasing the number of years of life. Our aim is to determine the prevalence of hypertension and propose intervention strategies. **Patients & Methods:** The study was conducted from September 2014 to March 2015 at the Great Lakes Regional Hospital where blood pressure of 3214 patients was taken by Rossmax machine. Data processing used SPSS software 18.0®. The confidence interval was 95%. **Results:** Hypertension was defined as the presence of antihypertensive treatment or BP≥ 140/90 mm Hg. The adjusted prevalence was 50.7% (n = 1632). Among hypertensive 18.9% (n = 608) had uncontrolled Blood Pressure (BP> 150/100 mmHg). The causes of systo-diastolic hypertension were essential hypertension (61.3%, n = 1971), atherosclerotic 8.8%, n = 283; Cushing syndrome: 2.9%, n = 95), primary aldosteronism (2.9%, n = 39); arterial stenosis 0.6%, n = 19 and pheochromocytoma (0.1%, n = 3). The causes of systolic blood pressure were hyperthyroidism (16.6%; n = 532), hydronephrosis (3.5%; n = 102) and aortic coarctation (0.9%; n = 29). **Conclusion:** In the prevention of high blood pressure, health workforce will be to determine the cause and to classify hypertension according to the systolo-diastolic or systolic blood pressure.

CLINICAL/OUTCOMES/POPULATION POSTER SESSION #2: 15:00 - 17:00 MISSISSAUGA FOYER

CR22 - Cigarette smoking as a risk factor for masked hypertension: a systematic review

Benjamin Sehmer MB BCh BAO, Andrea Blotsky MD, Benedicte Nauche MLIS, Ally Prebtani MD, Elham Rahme PhD, Kaberi Dasgupta MD.

Background: Masked hypertension is associated with an increased risk of cardiovascular disease and mortality; however the risk factors for masked hypertension are not well defined. The identification of these risk factors may enhance appropriate patient selection for screening with ambulatory blood pressure monitoring. We evaluated the association of smoking with masked hypertension by performing a systematic review and meta-analysis. **Method/Results:** We included cross-sectional studies that evaluated the prevalence of masked hypertension using ambulatory blood pressure monitoring (ABPM) or home blood pressure (HBP) measurements and also reported on subjects' current cigarette smoking status. Masked hypertension was defined as clinic BP<140/90 mmHg and elevated ABPM or HBP >135/85 mmHg. A search was conducted in December, 2014 for articles and conference abstracts in Medline, Embase, Web of Science, Biosis Previews via Ovid, PubMed, Scopus, and the Cochrane Library. Risk of bias was assessed regarding selection methods, clinic BP measurement, home and ambulatory BP measurement, and measurement of confounding variables including age and sex. Of the 2339 articles screened, we included 15 involving 24,586 patients with clinic BP 140/90 mmHg. Of these, 7295 (30%) had masked hypertension, and 17291 (70%) had sustained normotension. Eighty percent of masked hypertensives were smokers compared with 15% of sustained normotensives, yielding a crude odds ratio of 1.22 (95%CI 1.13-1.31). Only 3 studies reported adjusted odds ratios of 1.39 (95%CI 1.29-1.53), 1.93 (95%CI 1.06-3.52) and 2.57 (95% CI 1.50-4.42), respectively. **Conclusion:** In a preliminary analysis, smoking is a risk factor for masked hypertension. A potential limitation is that studies with null or inconclusive associations may not have published their findings.

ABSTRACTS
Saturday, October 24

BIOMEDICAL ORAL SESSION #3: 09:30 - 09:45 MISSISSAUGA B

Particular plasma oxylipins increase the odds of high central blood pressure and are beneficially influenced by dietary flaxseed in patients with peripheral arterial disease

Stephanie P.B. Caligiuri[1-3], Delfin Rodriguez-Leyva[1,2] Harold Aukema[1,4], , Amir Ravandi[2,3,5] Randy Guzman[6], & Grant N. Pierce[1-3] [1]Canadian Centre for Agri-food Research in Health & Medicine (CCARM) & [2]the Institute of Cardiovascular Sciences, St. Boniface Hospital Research Centre, & [3]the Departments of Physiology & Pathophysiology, [4]Human Nutritional Sciences, [5]Internal Medicine & 6Surgery, University of Manitoba, Winnipeg, Canada.

Background: A novel therapeutic target for hypertension is a class of highly bioactive molecules called oxylipins. Oxylipins include the well-known eicosanoids but also the more novel octadecanoids and docosanoids. Oxylipins are endogenously produced from polyunsaturated fatty acids and regulate vascular tone and inflammation. Dietary flaxseed may be a novel strategy to target oxylipins as flaxseed contains an abundant level of polyunsaturated fatty acids which can alter the oxylipin substrate profile. **Methods:** A randomized, double-blinded, controlled clinical trial, the Flax-PAD trial, was used to assess 1) the impact of dietary flaxseed on central aortic BP and 2) the relationship of plasma oxylipins to central aortic blood pressure (n=81). Central BP and plasma oxylipins were assessed by pulse wave analysis and HPLC-MS/MS technology, respectively. **Results:** At baseline, the central BP (systolic/diastolic) in the placebo and flaxseed group were, 131/73 ± 2.5/1.4 mmHg and 128/71 ± 2.6/1.4 mmHg, respectively. At 6 months the central BP in the placebo and flaxseed groups were, 132/74 ± 2.9/1.8 mmHg and 124/70 ± 2.6/1.6 mmHg ($p<0.05$). Significant associations were observed between 17 of the 43 detected oxylipins (primarily produced from arachidonic acid) and central blood pressure. After logistic regression analysis, every 1 nM increase in 16-hydroxyeicosatetraenoic acid (HETE) increased the odds of having high central systolic BP by 15-fold, of having high central diastolic BP by 6-fold and of having high central mean arterial pressure by 15-fold. In addition, every 1 nM increase in 5,6-dihydroxyeicosatrienoic acid (DiHETrE) and 11,12-DiHETrE increased the odds of having high central mean arterial pressure by 45- and 18-fold, respectively. Flaxseed induced a significant decrease in these as well as 4 other vasoconstrictive oxylipins ($p<0.05$). **Conclusion:** Oxylipins are strongly associated with central aortic blood pressure, are beneficially affected by dietary flaxseed, and may be effective therapeutic targets for hypertension in PAD.

BIOMEDICAL ORAL SESSION #3: 09:45 - 10:00 MISSISSAUGA B

Estrogenic neurons in the medial amygdala prevent stress-induced hypertension

Xia Yan, Physiology, Baylor College of Medicine; Pingwen Xu, Pediatrics, Baylor College of Medicine; Yanlin He, Pediatrics, Baylor College of Medicine; Alex Henderson, Pediatrics, Baylor College of Medicine; Corey L. Reynolds, Pediatrics, Baylor College of Medicine; Yong Xu, Molecular and Cell Biology, Pediatrics, Baylor College of Medicine, Houston, TX, 77030.

Background: Psychological stress contributes to the development of hypertension in humans, and estrogens prevent stress-induced hypertension with unknown mechanisms. Stress-induced hypertension is associated with increased neural activity in the medial amygdala (MeA), a brain region expressing abundant estrogen receptor-α (ERα). Thus, we hypothesize that estrogens prevent stress-induced hypertension partly through actions on ERα expressed by MeA neurons. **Methods/Results:** We used DREADD technology to selectively activate SIM1 neurons in the MeA in conscious mice and tested effects of MeA SIM1 neural activity on blood pressure (BP). In addition, Cre-LoxP system was used to genetically remove ERα from SIM1 neurons in female mice; in parallel, we stereotaxically injected AAV-Cre into the MeA of $ER\alpha^{lox/lox}$ female mice to delete ERα from all MeA neurons. We tested effects of estrogen replacement (vs. estrogen depletion) on stress-induced hypertension in these mutant mice and their wild type littermates. Finally, we used both c-fos immunoreactivity (in vivo) and slice electrophysiology (ex vivo) to examine cellular activities of MeA neurons in wild type mice or in mutant mice lacking ERα from SIM1 neurons, with estrogen depletion or supplement, at basal or stressed condition. We showed that selective activation of MeA SIM1 neurons increased basal BP and potentiated hypertensive responses provoked by psychological stress (restraint) in conscious mice. While estrogen replacement prevented stress-induced hypertension in wild type female mice, deletion of ERα from SIM1 neurons, or deletion of ERα from the MeA attenuated these anti-hypertensive effects of estrogens during stress. We also demonstrated that estrogens protect against increased neural activity under stress, and that MeA neurons lacking ERα showed increased excitability compared to wild type MeA neurons. **Conclusion:** Our results indicate that the antihypertensive effects of estrogens are partly mediated through ERα in the MeA neurons.

BIOMEDICAL ORAL SESSION #3: 10:00 - 10:15 MISSISSAUGA B

Endothelin-1 overexpression preserves endothelial function in mice with vascular smooth muscle cell-restricted Pparγ knockout

Noureddine Idris-Khodja[1], Sofiane Ouerd[1], Michelle Trindade[1], Jordan Gornitsky[1], Asia Rehman[1], Tlili Barhoumi[1], Stefan Offermanns[3], Frank J. Gonzalez[4], Pierre Paradis[1], Ernesto L. Schiffrin[1,2] [1]Vascular and Hypertension Research Unit, Lady Davis Institute for Medical Research, [2]Department of Medicine, SMBD-Jewish General Hospital, McGill University, Montréal, QC, Canada; [3]Department of Pharmacology, Max-Planck-Institute for Heart and Lung Research, Bad Nauheim, Germany; [4]Laboratory of Metabolism, Division of Basic Sciences, National Cancer Institute, NIH, Bethesda, MD, USA.

Objective: Peroxisome proliferator-activated receptor γ (PPARγ) agonists reduce blood pressure (BP) and vascular injury in hypertensive rodents and humans. Vascular smooth muscle cell (VSMC) Pparγ inactivation using a tamoxifen inducible Cre-Lox system enhanced angiotensin II-induced vascular injury. Transgenic mice with endothelial endothelin (ET)-1 overexpression (eET-1) exhibit endothelial dysfunction, increased oxidative stress and inflammation. We hypothesized that VSMC Pparg inactivation (smPpary$^{-/-}$) will exaggerate ET-1-induced vascular damage. **Method/ Results:** Eleven week-old male control, eET-1, smPpary$^{-/-}$ andeET-1/smPpary$^{-/-}$ mice were treated with tamoxifen (1 mg/kg/day, s.c.) for 5 days and sacrificed 4 weeks later. Systolic BP was 14 mmHg higher ineET-1 compared to control (P0.05) and unaffected by Pparγ inactivation. SmPpary$^{-/-}$ mesenteric artery (MA) vasodilatory responses to acetylcholine were reduced by 37% compared to control (P0.05). SmPpary$^{-/-}$ presented an increased contractile sensitivity to ET-1 compared to control (EC_{50}: $6.4x10^{-10}$ vs $2.4x10^{-10}$ M, P0.05), which was abrogated by a nitric oxide synthase (NOS) inhibitor. MA ET type A and B receptor (Ednra and Ednrb) mRNA expression was increased by ~60% in smPpary$^{-/-}$ and eET-1 compared to control (P0.05), whereas Ednra expression was decreased and Ednrb increased respectively by 18% and 17% in eET-1/smPpary$^{-/-}$ compared to smPpary$^{-/-}$ (P0.05). Nos2 and Nos3 expression was increased by 270% and 21% in eET-1, respectively, and Nos2 expression was increased 2-fold in smPpary$^{-/-}$ compared to WT (P0.05). Reactive oxygen species production was increased in eET-1 (70%), smPpary$^{-/-}$ (120%) andeET-1/smPpary$^{-/-}$ (180%) compared to control (P0.05). MA monocyte chemoattractant protein-1 expression was ≥70% higher in smPpary$^{-/-}$ andeET-1/smPpary$^{-/-}$ compared to control (P0.05). Perivascular fat monocyte/macrophage infiltration was ~2-fold higher in eET-1 and smPpary$^{-/-}$ compared to control (P0.05), and further increased by 68% ineET-1/smPpary$^{-/-}$ (P0.05). **Conclusion:** Increased ET-1 paradoxically preserves endothelial function in mice with smPpary inactivation, despite enhanced oxidative stress and inflammation.

BIOMEDICAL ORAL SESSION #3: 10:15 - 10:30 MISSISSAUGA B

Effects of acute IL-17a exposure on cerebrovascular function

Amy Randell, BSc (Pharm), Simon Rousseau, Noriko Daneshtalab, PhD, Memorial University of Newfoundland and *McGill University.*

Background: Patients with autoimmune disease, such as rheumatoid arthritis (RA), have a higher incidence of hypertension and stroke than the normal population. Recent studies have shown that IL-17 plays a critical role in the propagation of the inflammatory process during chronic inflammatory autoimmune disorders like RA and has recently become a target under investigation for RA treatment. We believe that IL-17a may also play a role in the cerebrovascular dysfunction observed in the middle cerebral artery (MCA) of an animal model for hypertension and RA, potentially mitigating the increased propensity for hemorrhagic stroke (HS) development. **Objective:** To investigate the effects of acute IL-17a exposure on MCA function in a hypertensive animal model. **Methods:** We used a Spontaneously Hypertensive Rat (SHR) model, weaned at 5 weeks of age and fed a Purina diet. They were sacrificed at 6-8 months of age and their MCA's were isolated and placed on a pressure myograph. Control MCA's were pressurized at 100 mmHg and left to equilibrate for 45-60 mins in HEPES buffer bath while the experimental group were pressurized and incubated with IL-17a (100 ng/ml) during equilibration. The ability of the MCAs to undergo pressure dependent constriction (PDC) and react to vasoactive peptides was evaluated. **Results and Discussion:** MCA's incubated with IL-17a showed a significant decrease in the ability to perform PDC as well as a diminished response to bradykinin (an indicator of endothelial function) while vascular smooth muscle function remained unchanged. **Conclusion:** IL-17a may be involved in affecting aspects of MCA's ability to function normally, possibly involving endothelial function and also causing deficits in PDC response. This may, in turn, increase the chances of HS development in presence of inflammatory stimulus.

ABSTRACTS
Saturday, October 24

CLINICAL/OUTCOMES/POPULATION ORAL SESSION #3: 09:00 - 09:15 MISSISSAUGA C

Development and piloting of a curriculum for training non-physician health workers to assess and manage hypertension in low- and middle-income areas

Maheer Khan (Population Health Research Institute), Hadi Musa (Population Health Research Institute), Pablo Lamelas (Population Health Research Institute), J.D. Schwalm (McMaster University and Population Health Research Institute).

Introduction: Cardiovascular disease (CVD) is the major cause of mortality globally and is increasing in low- and middle-income countries (LMIC), driven particularly by high rates of untreated hypertension. A growing trend of task-shifting to non-physician healthcare workers (NPHW) has proven to be a cost-effective method of delivering culturally relevant counselling and prevention strategies to at-risk populations. The training of NPHW, however, is often undisclosed and unclear. Our aim was to develop and evaluate a standardized, evidence-based curriculum to train NPHW in identifying hypertension and providing counselling in the community. **Methods/Results:** The core curriculum was developed with an asset based community development (ABCD) framework, placing significant importance on the empowerment of local NPHW. In the development phase, the interdisciplinary team consulted both local and international resources to design an interactive NPHW training curriculum for the assessment and management of hypertension in LMIC, culminating in a nine-module, weeklong training package. Subsequently, training of 24 NPHW has been completed in Colombia (n=6), Malaysia (n=14), and Canada (n=4). All participants that have completed the course have achieved the minimum 85% grade on an observed standardized clinical exam (OSCE). Satisfaction with the curriculum has been high with 92% of participants agreeing that the curriculum is culturally adaptable. Three trainers have led the curriculum and agree that the program was easy to run, and content was clear and concise. **Conclusion:** An approach to lowering the global burden of CVD using NPHW shows tremendous potential as task-shifting can reduce costs and provide health care access to marginalized populations in LMIC. We have successfully developed and piloted an evidence-based, culturally sensitive curriculum to train NPHW in identifying hypertension, and providing counselling for the reduction of CVD risk factors. This has been used to successfully train NPHW and has been met with a positive response.

CLINICAL/OUTCOMES/POPULATION ORAL SESSION #3: 09:15 - 09:30 MISSISSAUGA C

A systematic review and meta-analysis of blood pressure measurement techniques in children

Christine Voss, PhD, Division of Cardiology, Department of Pediatrics, University of British Columbia. Kevin C. Harris, MD MHSc FRCPC, Division of Cardiology, Department of Pediatrics, University of British Columbia.

Introduction: Mercury sphygmomanometers (HgS) have been considered the gold standard to measure blood pressure (BP). Aneroid sphygmomanometers (AnS) and automated oscillometric devices (OD) are mercury-free alternatives, the latter of which are now recommended for use in adults given their good inter-rater reliability. However, there is equipoise regarding the optimal mercury-free BP measurement technique in children. We conducted a systematic review and meta-analysis to determine the validity of OD and AnS compared to HgS in children. **Methods/Results:** We searched electronic databases (Medline, Embase, CINAHL, Web of Science) using relevant medical subject headings and keywords, and the following inclusion criteria: 1) age 3-18 years; 2) BP measured by HgS and at least one other method (AnS or OD); 3) BP measurement on the arm. Duplicates were removed and titles/abstracts were reviewed by one author. Two authors then independently reviewed the remaining articles to determine inclusion. In case of disagreement, authors discussed articles to reach consensus. We extracted relevant data (n, mean and SD or mean difference) from articles and performed a meta-analysis (RevMan v.5.3) to determine the weighted mean difference [95%CI] between BP measurements obtained by HgS compared to AnS or OD. Of the initial 1415 articles, 92 articles underwent full text review. We included 29 studies of OD (18,250 children). Meta-analysis showed that SBP and DBP in the OD was higher (SBP: 3.05mmHg [2.85, 3.24], DBP: 2.43mmHg [2.24, 2.61]) than the HgS measurements. We only found 3 articles that compared AnS with HgS and therefore did not conduct a meta-analysis. **Conclusion:** OD overestimates SBP in children by 3.1 mmHg and in DBP by 2.4 mmHg. The clinical importance of this difference in measurement warrants evaluation.

CLINICAL/OUTCOMES/POPULATION ORAL SESSION #3: 09:30 - 09:45 MISSISSAUGA C

Case series of sequential physiologic changes in macular thickness in pregnancy: Observations of normal and disease specific patterns, mechanisms of response, and clinical interpretation

T. Lee-Ann Hawkins, MSc, MD; R. Geoff Williams, BSc, MD; Kara Nerenberg MSc, MD; Anshula Ambasta, BSc.

Background: The eyes are a forward extension of the brain. The macula of the eye is a pure capillary bed with a microcirculation homologous to that of neural tissues. Unlike the small arterioles of most organs, the eye is known to auto regulate (AR) its capillary transudation such that it remains within tight physiologic limits despite large variations in pressure and flow within the systemic circulation. Pregnancy is accompanied by a 40-60% increase in blood volume and cardiac output in addition to increased capillary permeability, which further test the integrity of the AR reflex. **Methods:** Over 80 pregnant women had sequential SD-OCT measurements of macular thickness (a surrogate of tissue hydration). Women with normal pregnancies, pre-existing hypertension now pregnant, and hypertensive disorders of pregnancy (gestational hypertension, preeclampsia and eclampsia) were included. **Results:** We report on the data collected to date. The patterns of change in macular thickness that characterize a normal and abnormal AR response are presented (See Table). Analyses show SD-OCT has a sensitivity of 0.92, specificity 0.85, positive predictive value (PPV) 0.80, and negative predictive value 0.92 for discriminating preeclampsia from normal pregnancy. Confidence intervals around these estimates are large, but the lower limit of the 95% CI of the OR for PPV is 4.8 suggesting they are likely clinically meaningful. ROCs are pending. **Conclusions:** Preliminary data suggest SD-OCT is able to profile the AR microvascular response of neural tissues to the hemodynamic changes accompanying pregnancy. The AR curve is left-shifted in normal pregnancy. This differs from that reported for other tissues like the kidney. Patients with severe pre-existing hypertension have an exaggerated AR reflex as BP rises. AR fails in patients with severe hypertensive even before BP starts to rise. Further studies are needed to determine the role of SD-OCT in discriminating normal from hypertensive pregnancies.

Sequential Change in Macular Thickness on ETDRS Grid in 3 Patients at Differing Gestational Dates (See chart below)

Pt#	Gestational Age	Rsf	Ris	Rin	Rii	Rit	Ros	Ron	Roi	Rot	Lsf	Lis	Lit	Lii	Lin	Los	Lot	Loi	Lon
S1	BLD non preg	3	3	3	2	1	-1	2	2	1	0	0	-3	1	3	0	2	1	-1
S1	17.1w G2P1 preg	-4	-7	-9	-6	-6	-3	-7	-1	-2	-6	-7	-7	-7	-7	-1	-4	-5	-5
S1	28.2w	-1	-12	-7	-5	-12	-4	-10	-2	1	-5	-10	-7	-10	-15	-2	-6	-5	-5
S1	32.1w	-2	-8	-10	-7	-7	-5	-7	-1	-2	-4	-10	-5	-8	-12	-1	-3	-5	-5
S1	37.1wDEL24hb	-3	-10	-11	-8	-8	-6	-9	-1	-2	-1	-11	-4	-5	-13	0	-3	-5	-2
S1	5wp	1	0	2	0	-1	0	-1	2	2	1	-3	2	0	-6	2	-2	-6	1
S1b	16.6w G3P2 preg	-2	-11	-11	-6	-6	-6	-10	-7	-3	0	-5	-8	-7	-4	-3	-2	-2	-6
S1b	32.4w	-1	-10	-12	-7	-5	-5	-8	-6	-4	-1	-6	-7	-9	-6	-3	-2	-4	-8
S1b	40.4wDEL12hp	-3	-10	-13	-11	-9	-7	-12	-6	-6	-5	-8	-8	-9	-8	-1	-2	-2	-8
S1b	3.5wp	-1	-7	-8	-3	-3	-3	-7	-5	-2	-3	-4	-6	-5	-3	-1	0	-1	-8
S3	BLD 12.2w preg	1	0	2	3	1	0	2	2	0	0	-2	0	-2	-1	-2	-1	-1	-1
S3	18.4w	2	2	2	2	1	4	4	3	2	2	0	1	0	1	1	1	1	2
S3	23.4w	1	0	2	3	1	1	2	1	1	2	1	2	0	1	-1	0	-1	1
S3	29.4w	4	2	4	4	3	3	3	2	3	4	3	2	2	4	2	1	2	3
S3	32.3w	1	1	3	6	2	3	4	4	3	1	1	2	2	2	1	2	3	2
S3	35.3wDEL3wb	5	4	5	3	4	4	4	4	3	4	3	4	1	4	2	3	3	4
S3	2.4wp	5	5	5	3	3	5	4	4	6	4	4	3	2	8	4	6	3	7
S3	10.3wp	5	5	6	3	4	5	3	2	3	6	7	7	3	7	5	5	2	3
S50	BLD 16.5w preg	-2	-1	-1	0	1	1	1	-2	1	1	0	0	1	1	1	1	1	1
S50	21.3w	-3	-6	-8	-3	2	-3	-2	-4	0	1	0	-1	0	1	0	1	1	1
S50	26.4w	-9	-9	-12	-8	-6	-7	-7	-9	-6	-1	-2	-3	-3	-1	-5	-3	-3	-3
S50	27.2w	-9	-8	-10	-8	-7	-5	-6	-10	-6	-3	-1	-4	-2	-1	-3	-1	-2	-2
S50	28.5wDEL44hp	-10	-10	-16	-11	-9	-6	-3	-8	-8	-9	-6	-8	-6	-5	-8	-3	-5	-5
S50	15dp	-11	-10	-13	-10	-7	-9	-9	-11	-9	-7	-3	-6	-6	-3	-7	-3	-4	-4
S50	5.1wp	-7	-5	-7	-4	-2	-5	-6	-11	-6	-4	-1	-2	-2	0	-3	-1	-2	-2

(Text continued on next page)

The eyes are a forward extension of the brain. The macula of the eye is a pure capillary bed with a microcirculation homologous to that of neural tissues. Unlike the small arterioles of most organs, the eye is known to auto regulate (AR) its capillary transudation such that it remains within tight physiologic limits despite large variations in pressure and flow within the systemic circulation. Pregnancy is accompanied by a 40-60% increase in blood volume and cardiac output in addition to increased capillary permeability, which further test the integrity of the AR reflex. Methods: Over 80 pregnant women had sequential SD-OCT measurements of macular thickness (a surrogate of tissue hydration). Women with normal pregnancies, pre-existing hypertension now pregnant, and hypertensive disorders of pregnancy (gestational hypertension, preeclampsia and eclampsia) were included. Results: We report on the data collected to date. The patterns of change in macular thickness that characterize a normal and abnormal AR response are presented (See Table). Analyses show SD-OCT has a sensitivity of 0.92, specificity 0.85, positive predictive value (PPV) 0.80, and negative predictive value 0.92 for discriminating preeclampsia from normal pregnancy. Confidence intervals around these estimates are large, but the lower limit of the 95% CI of the OR for PPV is 4.8 suggesting they are likely clinically meaningful. ROCs are pending. Conclusions: Preliminary data suggest SD-OCT is able to profile the AR microvascular response of neural tissues to the hemodynamic changes accompanying pregnancy. The AR curve is left-shifted in normal pregnancy. This differs from that reported for other tissues like the kidney. Patients with severe pre-existing hypertension have an exaggerated AR reflex as BP rises. AR fails in patients with severe hypertensive even before BP starts to rise. Further studies are needed to determine the role of SD-OCT in discriminating normal from hypertensive pregnancies.

CLINICAL/OUTCOMES/POPULATION ORAL SESSION #3: 09:45 - 10:00 MISSISSAUGA C

Reliability of physician ratings for avoidable hospitalization for patients with uncomplicated hypertension, an ambulatory care sensitive condition

Norm Campbell, William Ghali, Doreen Rabi, Hude Quan University of Calgary.

Purpose: With high-quality, community-based primary care, hospitalizations for ambulatory care sensitive conditions (ACSC) is considered avoidable. The purpose of this study was to test the inter-physician reliability of judgments of avoidable hospitalizations for one ACSC, uncomplicated hypertension, derived from medical chart review. **Methods:** We applied the Canadian Institute for Health Information's case definition to obtain a random sample of patients who had an ACSC hospitalization for uncomplicated hypertension in Calgary, Alberta. Medical chart review was conducted by 3 experienced physicians. Implicit methods were used to judge avoidability of hospitalization using a validated 5-point scale. **Results:** There was poor agreement among three physicians raters when judging the avoidability of 82 ACSC hospitalizations for uncomplicated hypertension (κ=0.092). The κ remained the same low level when assessing agreement between raters 1 and 3 (κ=0.092), but the κ was lower (less than chance agreement) for raters 1 and 2 (κ=-0.119) and raters 2 and 3 (κ=-0.008). When the 5-point scale was dichotomized, there was fair agreement among three raters (κ=0.217). The proportion of ACSC hospitalizations for uncomplicated hypertension that were rated as avoidable was 32.9%, 6.1% and 26.8% for raters 1, 2, and 3, respectively. **Conclusion:** This study found a low proportion of ACSC hospitalization rated as avoidable, and poor to fair agreement of judgment between physician raters. This suggests the validity and utility of this health indicator is questionable. It points to a need to abandon the use of ACSC entirely; or alternatively a need to work on the development of explicit criteria for judging avoidability.

BIOMEDICAL ORAL SESSION #4: 11:00 - 11:15 MISSISSAUGA B

A model of mono-arthritis and cerebrovascular dysfunction

Noriko Daneshtalab, Memorial University, St. John's Newfoundland.

Background: The incidence of hypertension and stroke are greater in arthritic patients than in the normal population, with a poorer prognosis following stroke, particularly hemorrhagic stroke (HS). To address the gap in knowledge of the mechanistic link between arthritis and HS incidence, we developed a hypertensive mono-arthritic rat model that develops HS and studied how systemic inflammation may lead to HS. **Methods:** Stroke resistant spontaneously hypertensive rat models (SHRsr) were used. They were either given a high salt (4% NaCl), or regular Purina chow (0.9% NaCl) diet and further divided into adjuvant mono-arthritis or control groups by injection of either Complete Freund's Adjuvant or Saline intradermally in the left paw at 5-7 months of age. The animals were followed for 21 days, and monitored for inflammation and blood pressure changes. Plasma and organs were isolated after 21 days for histological studies, and signs of systemic and central inflammation. Pressure myograph studies were performed on the middle cerebral artery (MCA). **Results:** A statistically higher levels of TNF alpha was observed in the mono-arthritis (vs. control) animals. The mono-arthritis group also had severe inflammatory infiltrates, bone, and kidney damage vs. control group. The brains of mono-arthritic group showed increased inflammatory infiltrates, as well as astrocyte and microglia activation. Evidence of HS is determined with Evans Blue extravasation, which occurred in the mono-arthritic SHRsr groups, and was akin to that seen in the post-stroke stroke-prone spontaneously hypertensive rat (SHRsp)s. Pressure myograph studies indicated damage to aspects of the MCAs function, decreasing its ability to respond to increasing pressure and vasoactive peptides. **Conclusion:** Mono-arthritis leads to increase in systemic inflammation, and diminished MCA responsiveness, likely predisposing the animals to develop HS in presence of hypertension. Further studies are required to determine mechanisms of MCA damage for targeted treatment.

BIOMEDICAL ORAL SESSION #4: 11:15 - 11:30 MISSISSAUGA B

Altered vessel hemodynamics at rest and after acute physical stress in young smokers

Andrew F. Mutter, Department of Medicine, McGill University; Yessica-Haydee Gomez, Department of Medicine, Research Institute of the McGill University Health Centre; Robert J. Doonan, Department of Medicine, McGill University; Simon L. Bacon, Department of Exercise Science, Concordia University; Stella S. Daskalopoulou, Department of Medicine, Research Institute of the McGill University Health Centre.

Background: Long-standing smokers have stiffer arteries at rest; however the extent of the underlying vascular dysfunction in young healthy smokers has not been fully established. We aimed to examine the acute and chronic effect of smoking and nicotine exposure, on arterial stiffness at rest and in response to acute physical stress in young healthy individuals. **Methods:** Young healthy smokers (n=43) and non-smokers (n=80) underwent the '*arterial stress test*': blood pressure and arterial stiffness before and after (2, 5, 10, 15, 20 minutes) an exercise test to exhaustion on a treadmill. Several indices were assessed: central and peripheral systolic blood pressure (SBP) and pulse pressure (PP), PP amplification (PPA), augmentation index corrected for a heart rate (HR) of 75 beats/min (AIx75), sub endocardial viability ratio (SEVR), as well as carotid-femoral and carotid-radial pulse wave velocity (cfPWV and crPWV). Smokers were assessed under 3 conditions: a) after 12h smoking abstinence (chronic smoking), b) immediately after smoking one cigarette (acute smoking), and c) immediately after chewing nicotine gum. **Results:** Smokers at rest had elevated AIx75 ($p<0.001$), and decreased PPA ($p<0.001$) compared to non-smokers. Smoking a single cigarette increased central SBP, HR, and PPA, and lowered SEVR (all $p<0.001$. In response to acute maximal exercise, smokers failed to achieve comparable exercise time ($p=0.015$) and maximal HR ($p<0.001$) as non-smokers. Furthermore, smokers on all 3 conditions demonstrated lower exercise-induced changes in AIx75 all $p<0.001$) and SEVR (chronic $p=0.003$, acute and nicotine $p<0.001$) compared to non-smokers. After acute smoking, the exercise-induced increase in cfPWV was lower when compared to the chronic condition ($p=0.010$). **Conclusions:** These findings demonstrate that acute and chronic smoking lead to an altered vessel hemodynamic response even in young healthy smokers. Therefore, the '*arterial stress test*' may serve as a useful tool to identify vascular impairment in young smokers at an early subclinical stage.

ABSTRACTS
Saturday, October 24

BIOMEDICAL ORAL SESSION #4: 11:30 - 11:45 MISSISSAUGA B

Dysfunctional insulin signaling compromises cardiac function in obese Zucker Fatty rats

Joseph Fomusi Ndisang, University of Saskatchewan College of Medicine.

Background: Dysfunctional insulin signaling is associated with progressive alteration in cardiac structure and function. Since the mechanisms that drive insulin-resistance cardiomyopathy are not completely understood, we investigated the effects of heme oxygenase (HO) on cardiac dysfunction and impaired insulin signaling in obese Zucker rats. Moreover, the effects of HO on altered electrocardiography in conditions of impaired insulin signalling are largely unclear. **Method/Results:** Non-invasive echocardiography and invasive left-ventricular (LV) cannulation, imunohistochemical assay, spectrophotometry, enzyme-linked immunosorbent assay (ELISA) and Western-immunoblotting were used. HO was enhanced with hemin or blocked with stannous mesoporphyrin. Treatment with hemin improved insulin sensitivity, attenuated glucose intolerance, suppressed insulin resistance (HOMA-index), but potentiated important proteins of insulin-signaling including IRS-1, PI3K and GLUT4. These were associated with the potentiation of the HO-system, adiponectin and atrial-natriuretic peptide (ANP), whereas reduced inflammation/oxidative stress as well as extracellular matrix/pro-fibrotic proteins including collagen-IV, fibronectin, TGF-β1 were observed. Correspondingly, hemin treatment reduced cardiomyocyte longitudinal muscle-fiber thickness, a pathophysiological feature of cardio myocyte hypertrophy and ameliorated LV hypertrophy by reducing both diastolic and systolic LV wall thickness. These were accompanied by improved electrocardiographic and echocardiographic parameters including PR-interval, QRS-duration, ST-segment and QT-interval, mean-arterial pressure, arterial-systolic pressure, +dP/dt, and arterial-diastolic pressure, suggesting improved performance of the cardiac conduction system. In contrast, treatment with HO-blocker, stannous mesoporphyrin abolished the hemin effects. **Conclusion:** Our data suggest that upregulating the HO-system with hemin significantly improved the altered cardiac structure and function. The mechanisms for the improvement of cardiac morphology and electrophysiological properties include the attenuation of inflammatory/oxidative insults, the suppression of extracellular-matrix/profibrotic proteins, cardiac hypertrophy and fibrosis together with the potentiation of insulin signaling, adiponectin, ANP and improved glucose metabolism. Collectively, these data suggest that HO-inducers could be explored for the treatment of cardiometabolic complications comorbid with diabetes and obesity.

BIOMEDICAL ORAL SESSION #4: 11:45 - 12:00 MISSISSAUGA B

Iohexol plasma clearance in rat models: the clear choice for measuring early renal dysfunction

Mandy Turner[1], Kim Laverty[1], Martin Kaufmann[1], Glenville Jones[1], Christine White[2], Rachel Holden[2], Michael Adams[1], [1]Department of Biomedical and Molecular Sciences, Queen's University, Kingston, ON, [2]School of Medicine, Queen's University, Kingston, ON.

Background: Kidney failure and cardiovascular health are intrinsically intertwined: chronic kidney disease is not only a by-product of hypertension, but also a prominent cause. In animal models where kidney dysfunction is relevant, there is no accepted method for routine measurement of healthy and early declining renal function. This study evaluates plasma clearance of iohexol as a novel method for longitudinal glomerular filtration rate estimation as compared to previously validated inulin plasma clearance and plasma creatinine concentration. **Methods/Results:** Progressive kidney dysfunction was induced with a 0.25% adenine diet in male Sprague Dawley rats (N=8) 11-12 weeks old. Following serial tail-vein injections of iohexol (51.92 mg/kg) and FITC-inulin (2.5 µL/kg, 5% solution) under light anesthesia, 12 saphenous blood samples were taken from conscious rats over 5 hours, weekly (baseline, 5 weeks of treatment). Two reference methods for plasma clearance were used: a 2-compartment model and trapezoidal approximation of area under the curve. Both inulin and iohexol clearance reflected kidney impairment within the first week of adenine treatment ($p=0.003$ and $p=0.005$, respectively), whereas creatinine plasma concentration was not significantly elevated until the third week ($p=0.02$). Plasma iohexol clearance and plasma inulin clearance strongly correlate using all reference models ($R2=0.86$-0.96). Importantly, clearance calculated with a 1-compartment model using 2-samples was employed: samples at 30 min and 90 min post injection yielded high agreement ($R2=0.93$) and no significant bias, whereas inulin simplified to a 1-compartment method yields a mean underestimation of 22±13.2%. **Conclusion:** Iohexol plasma clearance is a more accurate and sensitive substitute for creatinine and maintains more accuracy using simplified modelling techniques than inulin. This research suggests iohexol plasma clearance is an expedient method of routine renal function assessment in rodent models. This approach will enable detection of early kidney dysfunction and facilitate concise interpretation of results derived from pre-clinical studies. (Supported by CIHR)

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Evolving Role of Single-pill Combination in HYPERTENSION

Friday, October 23rd, 2015
12:00 pm–1:30 pm (a lunch buffet will be available)
Mississauga A&D Room - Hilton Toronto Airport Hotel & Suites
Toronto, Ontario

Faculty

Co-Chairs:

Philip McFarlane, MD, FRCPC
Ernesto Schiffrin, MD, PhD, FRSC, FRCPC, FACP, FAHA

Presenters:

Ross D. Feldman, MD, FRCPC
George K. Dresser, MD, FRCPC
Stella Daskalopoulou, MD
Luc Trudeau, MD, CSPQ, FRCPC

Learning Objectives

After attending this symposium you will be able to:

1. Critique current hypertension management in Canada and identify reasons for existing care gaps.
2. Cite evidence for the benefits of single-pill antihypertensive combinations vs. stepped care or multiple-pill combinations.
3. Identify particular antihypertensive combinations that could provide the best results for your patients.
4. Debate the possible role for combination therapy in first-line antihypertensive treatment.

Agenda

12:00-12:10 pm	**Welcome and Introductions**	Dr. Ernesto Schiffrin Dr. Philip McFarlane
12:10-12:25 pm	**Current Management of Hypertension and Care Gaps**	Dr. Stella Daskalopoulou
12:25-12:40 pm	**Single-pill Therapy in First-line vs Stepped-care Approach**	Dr. Ross D. Feldman
12:40-12:55 pm	**Novel Single-pill Agents Designed for First-line**	Dr. Luc Trudeau
12:55-1:10 pm	**How Should the Guidelines Evolve for Single-pill Combination?**	Dr. George K. Dresser
1:10-1:25 pm	**Q&A**	All
1:25-1:30 pm	**Concluding Remarks**	Dr. Ernesto Schiffrin Dr. Philip McFarlane

Presented at Hypertension Canada's Canadian Hypertension Congress.

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Heather Bresch, CEO, Mylan Inc.

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