# Hypertension and Beyond

OCTOBER
16-18
2014

Congrès Hypertension Canada

Canadian Hypertension Congress

## 2014 CANADIAN HYPERTENSION CONGRESS

# PROGRAM

**DoubleTree by Hilton** **Gatineau, QC**

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Congrès Hypertension Canada

Canadian Hypertension Congress

# GENERAL INFORMATION

# CONGRESS CHAIRS' WELCOME
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Welcome to the fourth annual **Canadian Hypertension Congress**!

Our Congress is Canada's only national conference focused solely on hypertension and cardio-metabolic disease, and by its nature, represents our hypertension community's pursuit of excellence. From its preceding Canadian Hypertension Education Program (CHEP) consensus conference to its closing awards, the Canadian Hypertension Congress highlights the best and latest research in a forum that spurs continual learning and improvement for the better prevention, diagnosis, treatment, and control of hypertension and its complications as well the management of the other risk factors that contribute to the development of atherosclerosis.

As scientists and clinicians, our best work might be for naught without the development and maintenance of supportive environments where our discoveries can be translated and put into practice by health care professionals and patients. For this reason, we are opening this year's Congress with a forum on how to accelerate system changes to support the prevention and control of hypertension. We hope you'll add your valuable perspective to this important conversation.

The following two days are filled with presentations of the latest research and thinking in hypertension, and are provided in three programming streams to meet your educational needs. This year, we're pleased to offer a newly-defined stream in Primary Care – and offer a special welcome to family physicians attending from the Région de la capitale nationale.

Most important to our Congress is your participation, and your perspective. In our pursuit of excellence as conference organizers, we need to know what you want to see more of, less of, and what will make the experience more enriching for you in your learning and work. We hope you'll complete the survey evaluations in-session and post-event, or send us a note and let us know your thoughts and ideas.

We're very pleased you've joined us. Enjoy!

Bienvenue au quatrième congrès annuel, le **Congrès Hypertension Canada**!

Le Congrès Hypertension Canada est le seul du genre au pays à porter uniquement sur l'hypertension artérielle et le syndrome métabolique et, par le fait même, à représenter toute la collectivité en quête d'excellence dans le domaine de l'hypertension. Depuis la conférence de consensus du Programme éducatif canadien sur l'hypertension tenue précédemment jusqu'à la séance de clôture de remise des prix, le Congrès Hypertension Canada met en évidence les recherches les plus récentes et les meilleures qui soient dans le cadre d'un forum qui stimule l'apprentissage et le progrès continus en vue d'une amélioration de la prévention, du diagnostic, du traitement et de la maîtrise de l'hypertension artérielle et de ses complications, ainsi que de la prise en charge des autres facteurs de risque athérosclérogènes.

En tant que scientifiques ou cliniciens, nous sommes curieux, et, sans la mise en place et le maintien d'un environnement favorable dans lequel les découvertes peuvent se concrétiser et être mises en pratique par les professionnels de la santé et les patients, les meilleurs travaux pourraient passer inaperçus. Aussi le congrès de cette année s'ouvre-t-il par une table ronde sur la façon d'accélérer les changements au sein du système, qui soient propices à la prévention et à la maîtrise de l'hypertension. Nous espérons que vous ferez valoir votre point de vue, toujours important, et que vous participerez à cet important échange d'idées.

Réflexion et présentations sur les derniers travaux de recherche sur l'hypertension occuperont les deux prochaines journées, et le programme est structuré en trois parcours de manière à répondre à vos besoins de formation. De plus, cette année, nous avons le plaisir d'offrir un tout nouveau parcours en soins primaires; par ailleurs, nous saluons tout particulièrement les médecins de famille de la région de la Capitale nationale qui participent au congrès.

Enfin, l'ingrédient le plus important du congrès est votre participation, votre point de vue. En tant qu'organisateurs du congrès, toujours en quête d'excellence, nous voulons savoir ce que aimeriez avoir davantage, ou moins, et ce qu'il faudrait faire pour rendre votre expérience encore plus enrichissante qu'avant sur les plans de l'apprentissage et du travail. Nous espérons que vous remplirez les évaluations à la fin des séances ou après le congrès, ou encore vous pouvez nous faire part de vos observations dans un message envoyé ultérieurement.

Nous sommes très contents de vous compter parmi nous. Bon congrès!

*Rob Gros (left) and Ross Feldman*

**Ross Feldman, MD and Robert Gros, PhD**
Course Director and Congress Chairs
2014 Canadian Hypertension Congress
Hypertension Canada

# PRESIDENT'S WELCOME
# MOT DE BIENVENUE DU PRÉSIDENT

It is my great pleasure to wish all 2014 Canadian Hypertension Congress attendees – members, volunteers, scientists and clinicians from across disciplines, sponsors and others – a sincere thank you for your support of this Congress and of Hypertension Canada.

Canada's hypertension community is a collaborative community. Hundreds of healthcare professionals across the country, above and beyond our daily work, donate more than 15,000 hours to Hypertension Canada each year. This makes possible our research strategies, our CHEP Recommendations, our high-quality education programs and materials, this Congress, and more. The collective efforts, skills, and expertise power our mission to advance health through the prevention and control of high blood pressure and its complications.

Our collaboration extends from the health care system to corporate Canada. Complementing expert efforts is the support from corporations – our sponsors of this Congress, and of new and year-round initiatives – that help us build solutions from ideas, and bring them to people living with and at risk for hypertension, and to those professionals who treat them.

Together, our members, volunteers, sponsors and other supporters make Hypertension Canada's impact felt at home – and beyond our borders.

Earlier this year, I attended the conferences of several other nations' associations, and was amazed at the number of speakers who mentioned the CHEP recommendations either alone or in comparison to others. Our international impact – the CHEP recommendations' rigor and the evidence-based approach – resonates around the world.

During this Congress, I hope you'll reflect on your role in Hypertension Canada's far-reaching impact, and take a moment to celebrate our collective accomplishments – with our thanks.

J'ai le grand plaisir de remercier sincèrement tous les participants au Congrès Hypertension Canada de 2014 : membres, bénévoles, scientifiques et cliniciens de toutes disciplines, parrains et autres intervenants, de leur appui au congrès et à Hypertension Canada.

La collectivité qui s'intéresse à l'hypertension artérielle au Canada travaille sous le signe de la collaboration. Des centaines de professionnels de la santé, partout au pays, donnent plus de 15 000 heures de travail par année à Hypertension Canada, et ce, en sus de leur travail quotidien. Voilà comment sont rendues possibles l'élaboration des stratégies de recherche, des recommandations du Programme éducatif canadien sur l'hypertension (PECH), des programmes de formation et du matériel didactique de qualité; l'organisation du congrès et de bien d'autres initiatives. La réunion de tous ces efforts et de toutes ces compétences permet de donner corps à notre mission, qui est de faire la promotion de la santé par la prévention et la maîtrise de l'hypertension artérielle et de ses complications.

La collaboration couvre un large éventail d'activité, depuis le système de soins de santé jusqu'aux entreprises canadiennes. Complémentaire aux efforts des spécialistes est le soutien des entreprises, par exemple les parrains du congrès de 2014, ceux de nouveaux projets ou d'initiatives qui ont cours tout au long de l'année, qui aide l'organisation à mettre en œuvre des solutions créées en pensée, puis à les faire connaître aux personnes qui vivent aux prises avec l'hypertension ou qui sont prédisposées à la maladie, et aux professionnels de la santé qui les traitent.

C'est grâce à la contribution de tous : membres, bénévoles, parrains et autres intervenants, qu'Hypertension Canada laisse sa marque ici, au pays – et au-delà des frontières.

Plus tôt au cours de l'année, j'ai assisté aux congrès de plusieurs autres associations nationales et j'ai été étonné du nombre de conférenciers qui faisaient mention des recommandations du PECH soit de manière isolée, soit par comparaison avec d'autres lignes de conduite. Par la rigueur d'élaboration des recommandations du PECH et par son approche fondée sur les données probantes, l'organisation jouit d'une renommée internationale, qui trouve écho partout dans le monde.

J'espère que, durant le congrès, vous réfléchirez à votre rôle dans cette réputation d'envergure que s'est taillée Hypertension Canada et que vous prendrez le temps de souligner dans la joie et la bonne humeur les réalisations collectives, le tout agrémenté de nos remerciements.

**Ernesto L. Schiffrin, C.M., MD, PhD**
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## WHO WE ARE

### Mission:

Hypertension Canada is the only national health charity dedicated solely to **advancing health through the prevention and control of high blood pressure and its complications**.

Powered by an extensive network of inter-disciplinary expert volunteers, Hypertension Canada strives to bring about positive benefits for the millions of people in Canada who, on a daily basis, deal with the dangers of hypertension.

## 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 CIHR, foster career mentoring and succession planning, and seek opportunities to secure the future for hypertension research.

### EDUCATE

Through extensive and multi-pronged dissemination of evidence-based recommendations targeting healthcare professionals and people with hypertension, Hypertension Canada strives to improve hypertension diagnosis and management.

Hypertension Canada provides health care providers with our evidence-based CHEP recommendations to diagnose, treat and control hypertension. Developed by a multi-disciplinary committee, the CHEP recommendations are annually updated following a systematic literature review, intense discussion, and critical appraisals of all new clinical research.

CHEP


The CHEP recommendations are widely disseminated – published in lay and health care professional journals, in printed booklet form distributed nationally, and via a free mobile application – to reach health care providers in both clinical and community settings.

The CHEP recommendations are adapted to a suite of Continuing Medical Education / Continuing Professional Development programs.

These include in-person Train-the-Trainer workshops as well as new, on-line modules that allow the registrant to progress at one's own pace. Educational materials for patients help the health care provider put the new information into practice, and provide the patient with valuable resources to remember and reinforce medical direction.

Hypertension CANADA
Professional Education Program

### 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 CIHR/HSF Chair in Hypertension, 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 takes seriously its unique position and responsibility as Canada's only health organization focused on the globe's number one risk of disease and death. We serve our extensive network of professional volunteers, dedicated members, and collaborative partnerships as the most credible, reliable resource on hypertension. We share information, through our eINFO newsletter, our website, social media, and our Canadian Hypertension Congress. We strive to engage diverse perspectives, professionals, and partners to chart the most effective, collaborative and impactful approaches to our work. And together, we advance health through the prevention and control of hypertension and its complications.

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Learn more, and join us as a member, at **www.hypertension.ca**.

## QUI SOMMES-NOUS?

### Notre mission :

Hypertension Canada la seule organisation nationale de santé, sans but lucratif, **vouée uniquement à la promotion de la santé par la prévention et la maîtrise de l'hypertension artérielle et de ses complications**.

Forte d'un vaste réseau interdisciplinaire de spécialistes bénévoles, Hypertension Canada met tout en œuvre pour faire profiter des fruits de son travail les millions de personnes au Canada qui, tous les jours, sont aux prises avec l'hypertension.

## QUE FAISONS-NOUS?

### INNOVATION

Hypertension Canada vise à accroître la quantité et la qualité de la recherche sur l'hypertension artérielle et en biologie vasculaire en attirant de nouveaux chercheurs dans le domaine et en leur apportant soutien et mentorat. Nous offrons notre appui aux chercheurs, présents et futurs, par l'établissement de partenariats d'investissement avec les Instituts de recherche en santé du Canada; nous favorisons le mentorat professionnel et la planification de la relève et nous sommes à l'affût de nouvelles possibilités afin d'assurer l'avenir de la recherche sur l'hypertension.

### FORMATION

Par une large diffusion diversifiée des recommandations fondées sur des données probantes, qui cible les professionnels de la santé et les personnes hypertendues, Hypertension Canada mobilise toutes ses ressources afin d'améliorer le diagnostic et la prise en charge de l'hypertension.

PECH


Hypertension Canada communique aux fournisseurs de soins de santé les recommandations du PECH, fondées sur des données probantes afin de les aider à diagnostiquer, à traiter et à maîtriser l'hypertension artérielle. Élaborées par un comité pluridisciplinaire, les recommandations du PECH sont mises à jour tous les ans à la suite d'un examen méthodique de la documentation, de vives discussions et d'une évaluation critique de toutes les nouvelles recherches cliniques.

Les recommandations du PECH font l'objet d'une large diffusion, que ce soit dans des revues destinées au public ou à des professionnels de la santé; sous forme de livrets imprimés, distribués partout au pays; ou à l'aide d'une application mobile gratuite, de manière à joindre les fournisseurs de soins de santé tant en milieu clinique que dans la collectivité.

La présentation des recommandations du PECH varie aussi selon les besoins, de manière à convenir à divers programmes de formation médicale continue ou de formation professionnelle continue, par exemple les ateliers de Formation des formateurs, donnés en mode présentiel et les nouveaux modules offerts en ligne, qui permettent aux apprenants d'avancer à leur propre rythme.

Hypertension CANADA
Programme d'éducation professionnelle

Quant au matériel didactique conçu à l'intention des patients, il aide les fournisseurs de soins de santé à mettre en pratique les nouvelles informations, en plus d'offrir aux patients des ressources précieuses, bonnes à se rappeler, et de renforcer l'orientation médicale.

### INFLUENCE

Hypertension Canada s'efforce d'influer sur l'environnement externe pour mettre en œuvre des politiques et des stratégies qui visent à améliorer la prévention et le traitement de l'hypertension et de ses complications, et à sensibiliser les gens à la maladie, et ce, par la mobilisation de différents intervenants et des gouvernements. En collaboration avec la Chaire FMCC/IRSC en prévention et contrôle de l'hypertension artérielle et en tant qu'organisation citoyenne, soucieuse de la collectivité en soins de santé, Hypertension Canada se tient au courant des problèmes qui retiennent son attention et des différentes possibilités afin de produire des changements favorables qui la rapprochent encore davantage de sa mission.

# MOBILISATION

Hypertension Canada prend au sérieux la place de premier plan qu'elle occupe et le rôle unique qu'elle joue au Canada en tant que seule organisation de santé vouée au principal risque de morbidité et de mortalité dans le monde. Elle sert son vaste réseau de professionnels bénévoles, ses membres dévoués et les partenaires avec qui elle travaille en collaboration en se considérant comme la ressource la plus fiable et la plus sérieuse sur l'hypertension. La transmission de l'information se fait par l'intermédiaire du bulletin eINFO, du site Web, des médias sociaux et du Congrès Hypertension Canada. L'organisation cherche à recueillir divers points de vue et à attirer des professionnels et des partenaires de différents horizons afin de cerner les moyens les plus efficaces et les fructueux qui soient afin de lui permettre de réaliser son travail, et ce, dans un esprit de collaboration. C'est ainsi qu'Hypertension Canada, animée de toutes ses forces vives, réussit à faire la promotion de la santé par la prévention et la maîtrise de l'hypertension artérielle et de ses complications.

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Pour en savoir plus sur l'organisation et devenir membre, 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
- 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
- Foster discussion and debates that encourage innovation in cardiovascular health and research

## ACCREDITATION

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, and Western University (16.25 hours).

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

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Canadian Hypertension Congress

# SPEAKERS

# SPEAKERS

## THANK YOU TO OUR 2014 SPEAKERS

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# SPEAKERS

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Chief, Addictions, Centre for Addictions and Mental Health (CAMH)
Associate Professor, Departments of Family and Community
Medicine and Psychiatry and Dalla Lana School of Public Health,
University of Toronto
Toronto, Ontario

**Allan Skanes, MD, FRCPC**
Professor of Medicine, Western University
Director of Electrophysiology Lab
University Hospital
London, Ontario

**Duncan J. Stewart, MD**
Professor, Faculty of Medicine
University of Ottawa
CEO & Scientific Director
Ottawa Hospital Research Institute
Vice President, Research
The Ottawa Hospital
Ottawa, Ontario

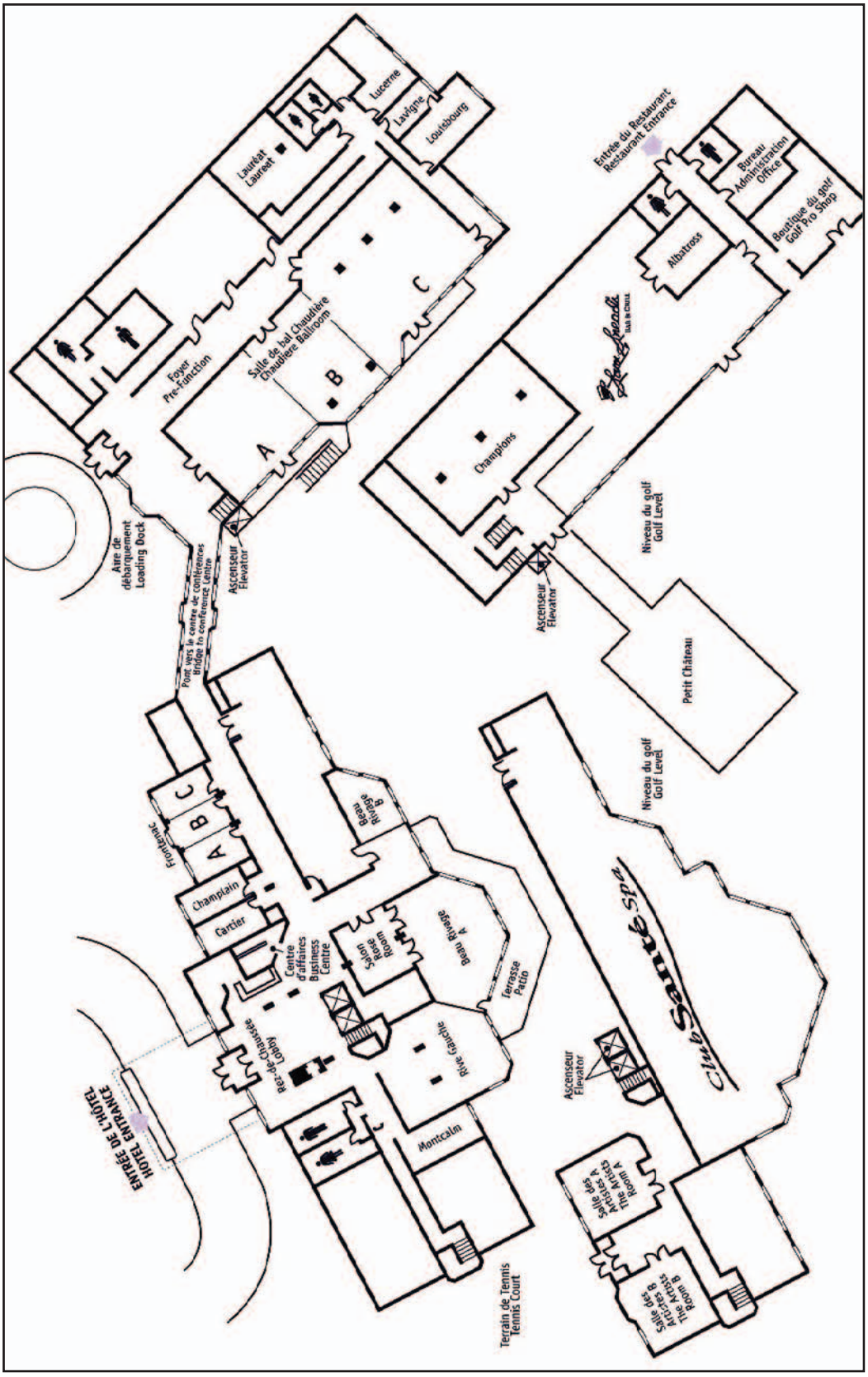
**James A. Stone, MD, PhD, FRCPC, FACC**
Clinical Professor of Medicine
University of Calgary
Calgary, Alberta

**Sheldon Tobe, MD, MScCH (HPTE), FRCPC, FACP, FASH**
Hypertension and Nephrology
HSF/NOSM Chair in Aboriginal and Rural Health Research
Professor in Medicine, University of Toronto and Northern Ontario
School of Medicine
Sunnybrook Health Sciences Centre-Div. of Nephrology
Toronto, Ontario

**Raymond Townsend**
Professor of Medicine
The Hospital of the University of Pennsylvania
Philadelphia, Pennsylvania

**Stephanie Watts**
Professor, Pharmacology and Toxicology
Assistant Dean, The Graduate School
Michigan State University
East Lansing, Michigan

# FLOOR PLAN


Lauréat
Laureat
Lucerne
Lavigne
Louisbourg
Foyer
Pre-Function
Salle de bal Chaudière
Chaudière Ballroom
A
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Champions
Albatross
Entrée du Restaurant
Restaurant Entrance
Bureau
Administration
Office
Boutique du golf
Golf Pro Shop
Niveau du golf
Golf Level
Aire de
débarquement
Loading Dock
Ascenseur
Elevator
Pont vers le centre de conférences
Bridge to conference centre
Petit Château
Champlain
Cartier
Centre
d'affaires
Business
Centre
Salon
Rose
Room
Beau Rivage
Terrasse
Patio
Rez-de-Chaussée
Lobby
Rive Cachée
Montcalm
Club Santé spa
Salle des
Artistes A
The Artists
Room
Salle des
Artistes B
The Artists
Room
Terrain de Tennis
Tennis Court
ENTRÉE DE L'HÔTEL
HOTEL ENTRANCE

Congrès Hypertension Canada

Canadian Hypertension Congress

# SCIENTIFIC PROGRAM

# SCIENTIFIC PROGRAM

## THURSDAY, OCTOBER 16, 2014

| | |
|---|---|
| 16:00 - 16:10 | **WELCOME AND OPENING REMARKS**<br>*Dr. Ross Feldman, CHC Chair*<br>Room: Chaudière AB |
| 16:10 - 17:00 | **PANEL DISCUSSION WITH AUDIENCE QUESTION AND ANSWER**<br>**System Changes for the Prevention and Control of Hypertension – Are we doing enough, are we doing too much?**<br>*Panelists: Dr. Marie-Dominique Beaulieu, Past President, College of Family Physicians of Canada,*<br>*Carlo Berardi, Past Chair of the Board, Ontario Pharmacists Association,*<br>*Rodney Ghali, Director General, Public Health Agency of Canada,*<br>*Jeff Leger, SVP, Loblaw Pharmacy*<br>*Moderator: Glen Doucet, Non-Profit and Government Relations Expert*<br>Room: Chaudière AB |
| 17:00 - 18:30 | **POSTER SESSION # 1**<br>Hallway & Foyer<br>**Biomedical Research Track**<br>**P1 - Effects of podocyte microparticles on cultured human proximal tubule cells** - *Shareef Akbari*<br>**P2 - Cardio protective role of the cholinergic system** - *Mouhamed Dakroub*<br>**P3 - Natriuretic peptide receptor-C agonist attenuates the expression of cell cycle proteins and proliferation of vascular smooth muscle cells from spontaneously hypertensive rats: role of Gi proteins and MAPkinase/ PI3kinase signaling** - *Yuan Li*<br>**P4 - Role of vascular smooth muscle cell PPARγ in aldosterone-induced vascular injury** - *Michelle Trindade*<br>**P5 - SKP-derived vascular smooth muscle cells exhibit type II diabetes associated dysfunction**- *Sarah K. Steinbach*<br>**P6 - MicroRNA and regulation of angiotensin II-induced vascular injury**- *Kugeng Huo*<br>**P7 - Differences in renal pathological responses found between salt sensitive and salt insensitive hypertension** - *Victoria Yum*<br>**Clinical/Outcomes/Population Research Track**<br>**P8 - Are Home Blood Pressure Monitors Accurate Compared to Validated Devices?** - *Eva Bruketa*<br>**P9 - Indexing Left Ventricular Mass** - *Sharmila Udupa*<br>**P10 - Immune sensitization and mortality on the waiting list for kidney transplantation** - *Ruth Sapir-Pichhadze*<br>**P12 - Formation and management of an interdisciplinary research network in vascular health** - *Amanda van Beinum*<br>**P13 - Practice Audit of Renal Denervation Procedure for Drug-resistant Hypertension** - *Praveena Sivapalan*<br>**P14 - Hospitalized Diabetes with Hypertension and Other Co-morbid Conditions in Canada** - *Andreas Wielgosz* |
| 17:00 - 20:00 | **WELCOME RECEPTION IN HALLWAY & FOYER & CHAUDIÈRE AB** |
| 17:00 - 21:00 | **SMOKING CESSATION WORKSHOP**<br>*Co-chairs: Dr. Peter Selby & Dr. Bob Reid*<br>Room: Frontenac ABC<br>**Beyond the Basics: Treating Hard Core Smokers in Clinical Practice**<br>17:00 - 17:30 **Current Evidence on Tobacco Addiction and its Treatment**<br>17:30 - 17:45 **Q & A**<br>17:45 - 18:15 **Case 1**<br>18:15 - 18:45 **Debrief of Case 1**<br>18:45 - 19:00 Break<br>19:00 - 19:30 **Case 2**<br>19:30 - 20:00 **Debrief of Case 2**<br>20:00 - 20:15 Break<br>20:15 - 20:35 **Case 3**<br>12:35 - 20:50 **Debrief of Case 3**<br>20:50 - 21:00 **Wrap Up & Next Steps** |

# SCIENTIFIC PROGRAM

## FRIDAY, OCTOBER 17, 2014

| | BIOMEDICAL RESEARCH TRACK ROOM: CHAUDIÈRE A | CLINICAL/OUTCOMES/ POPULATION RESEARCH TRACK ROOM: CHAUDIÈRE B | PRIMARY CARE TRACK ROOM: CHAUDIÈRE C |
|---|---|---|---|
| | *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 - 8:00 | **BREAKFAST IN THE PETIT CHÂTEAU** | | |
| 08:00 - 09:30 | **BIOMEDICAL # 1**<br>*Co-chairs: Dr. Dylan Burger & Dr. Duncan Stewart*<br><br>08:00<br>**Implication of renal angiotensin-(1-7) axis in the development of preeclampsia in previously hypertensive mice**<br>*Aida Kasaei Roodsari*<br><br>08:15<br>**High glucose increases endothelial microparticle formation, pro-oxidative activity and pro-coagulant activity**<br>*Dylan Burger*<br><br>08:30<br>**New Insights on Mechanisms of Foamy Macrophage Induction and Persistence**<br>*Marian Laderoute*<br><br>08:45<br>**Angiotensin II-induced vascular injury is counteracted by FOXP3+ T regulatory lymphocytes**<br>*Muhammad Oneeb Rehman Mian*<br><br>09:00 - 09:30<br>**STATE OF THE ART**<br>**Novel mechanisms of pulmonary vascular disease: how does endothelial cell apoptosis lead to runaway cell growth?**<br>*Dr. Duncan Stewart* | **CLINICAL # 1**<br>*Co-chairs: Dr. Ross Tsuyuki & Dr. Alexander Logan*<br><br>08:00 - 08:30<br>**STATE OF THE ART**<br>**Erectile Dysfunction and CV Disease**<br>*Dr. Gerald Brock*<br><br>08:30<br>**Relationship Between Extracellular Fluid Movement During Sleep and Urine Sodium Excretion in Normotensive and Hypertensive Subjects**<br>*Alexander Logan*<br><br>08:45<br>**The Control of Hypertension in Pregnancy Study (CHIPS) randomized controlled trial**<br>*Laura A. Magee*<br><br>09:00<br>**Comparison of auscultatory and oscilometric normative values for blood pressure evaluation in children**<br>*Terezie Šuláková*<br><br>09:15<br>**A randomized trial of the effect of pharmacist prescribing on improving blood pressure in the community: The Alberta clinical trial in optimizing hypertension (RxACTION)**<br>*Ross Tsuyuki* | **PRIMARY CARE # 1**<br>*Co-chairs: Dr. Raj Padwal & Dr. Mark Gelfer*<br><br>**Core Hypertension Curriculum Topics**<br><br>08:00 - 08:30<br>**Choosing BP thresholds and targets - what, for whom and why**<br>*Dr. Raj Padwal*<br><br>08:30 - 09:00<br>**Pitfalls to avoid in BP monitoring**<br>*Dr. Raymond Townsend*<br><br>09:00 - 09:30<br>**Firstline recommendations - which and why**<br>*Dr. Doreen Rabi* |
| 09:30 - 10:00 | **BREAK** | | |

# SCIENTIFIC PROGRAM

## FRIDAY, OCTOBER 17, 2014

| | BIOMEDICAL RESEARCH TRACK ROOM: CHAUDIÈRE A | CLINICAL/OUTCOMES/ POPULATION RESEARCH TRACK ROOM: CHAUDIÈRE B | PRIMARY CARE TRACK ROOM: CHAUDIÈRE C |
|---|---|---|---|
| 10:00 - 11:30 | **BIOMEDICAL # 2**<br>*Co-chairs: Dr. Michael Adams & Dr. Noriko Daneshtalab*<br><br>10:00<br>**Endothelin-1 Overexpression in Endothelial Cells Increases Blood Pressure In an Endothelin Type A Receptor-Dependent Manner**<br>*Suellen Coelho*<br><br>10:15<br>**von Willebrand Factor Synthesis and Circulating Half Life Are Progressively Elevated, but Relative Platelet Binding Decreases in Stage 3-5 Chronic Kidney Disease Patients**<br>*Cynthia Pruss*<br><br>10:30<br>**Nitric Oxide attenuates the enhanced expression of Giα proteins in vascular smooth muscle cells from Spontaneously Hypertensive Rats: Molecular mechanisms**<br>*Oli Sarkar*<br><br>10:45<br>**Endoplasmic reticulum stress inhibition preserves endothelial-mediated vasodilation in SHR resistance blood vessels**<br>*Kaitlyn Werner*<br><br>11:00 - 11:30<br>**STATE OF THE ART**<br>**How do Ang II or aldosterone cause hypertension? Role of kidney and brain actions**<br>*Dr. Frans Leenen* | **CLINICAL # 2**<br>*Co-chairs: Dr. Slimon Bacon & Dr. Richard Lewanczuk*<br><br>10:00 - 10:30<br>**STATE OF THE ART**<br>**The role of the health system in hypertension control**<br>*Dr. Richard Lewanczuk*<br><br>10:30<br>**Subcutaneous resistance artery remodeling and function in chronic kidney disease patients**<br>*Julio C. Fraulob-Aquino*<br><br>10:45<br>**Undiagnosed Hypertension in the Emergency Department: The Significance of Elevated Blood Pressure Measurements at Emergency Department Triage**<br>*Brian Levy*<br><br>11:00<br>**Marginal structural models for estimating the causal relationships between physical activity, blood pressure, and mortality in a longitudinal cohort study: the Honolulu Heart Program**<br>*Amanda M. Rossi*<br><br>11:15<br>**The relationship between primary care physician utilization and hospitalizations or ED visits: is uncomplicated hypertension really an ambulatory care sensitive condition?**<br>*Robin Walker* | **PRIMARY CARE # 2**<br>*Co-chairs: Dr. Charlotte Jones & Dr. Guy Tremblay*<br><br>**Primary Prevention Updates for other risk factors**<br><br>10:00 - 10:30<br>**Smoking Cessation: Slaying Zombies!**<br>*Dr. Andrew Pipe*<br><br>10:30 - 11:00<br>**2013 Canadian Cholesterol Guidelines**<br>*Dr. Robert Hegele*<br><br>11:00 - 11:30<br>**Type 2 Diabetes Recommendations Update**<br>*Dr. Charlotte Jones* |
| 11:30 - 13:30 | **LUNCH SYMPOSIUM IN THE PETIT CHÂTEAU** | | |

# SCIENTIFIC PROGRAM

## FRIDAY, OCTOBER 17, 2014

| | BIOMEDICAL RESEARCH TRACK ROOM: CHAUDIÈRE A | CLINICAL/OUTCOMES/ POPULATION RESEARCH TRACK ROOM: CHAUDIÈRE B | PRIMARY CARE TRACK ROOM: CHAUDIÈRE C |
|---|---|---|---|
| 13:30 - 15:00 | **BIOMEDICAL SYMPOSIUM**<br>*Chair: Dr. Rob Gros*<br><br>**Identifiying new targets in treatment of hypertension and dyslipidemia: population genetics meets cellular biology**<br><br>13:30 - 14:00<br>**Surprising links between G Proteins and LDL cholesterol metabolism**<br>*Dr. Robert Hegele*<br><br>14:00- 14:30<br>**Can genetics help us to identify new targets for prevention and treatment of hypertension?**<br>*Dr. Pavel Hamet*<br><br>14:30 - 15:00<br>**Coronary artery disease in women: new targets, new mechanisms**<br>*Dr. Ross Feldman* | **HSF CIHR CHAIR IN HYPERTENSION PREVENTION AND CONTROL SYMPOSIA**<br>*Chair: Dr. Norm Campbell*<br><br>13:30 - 13:50<br>**Update on sodium reduction: new evidence, challenges and a way forward**<br>*Dr. Norm Campbell*<br><br>13:50- 14:10<br>**Intervening on the food supply in an isolated Northern Community. Lessons learned.**<br>*Ms. Nancy Perkins*<br><br>14:10 - 14:30<br>**The Canadian Hypertension Advisory Committee and Canadian Hypertension Framework. What we are doing and why.**<br>*Ms. Tara Duhaney*<br><br>14:30 - 15:00<br>**Panel discussion and questions**<br>*Dr. Norm Campbell, Ms. Nancy Perkins, Ms. Tara Duhaney, Dr. Sheldon Tobe, Dr. Debra Reid* | **PRIMARY CARE # 3**<br>ANGLOPHONE TRACK<br>*Co-chairs: Dr. Tavis Campbell & Luc Poirier*<br><br>13:30 - 14:15<br>**Motivational interviewing applied to risk factor modification**<br>*Dr. Tavis Campbell*<br><br>14:15 - 15:00<br>**How to incorporate ABPM into your practice**<br>*Dr. Martin Myers*<br><br>**ROOM: FRONTENAC ABC**<br><br>**PRIMARY CARE #3**<br>FRANCOPHONE TRACK<br>*Co-chairs: Prof. Lyne Cloutier & Dr. Denis Drouin*<br><br>13:30 - 14:15<br>**Techniques d'entretien motivationnelle appliquée à la modification des facteurs de risque**<br>*Dr. Kim Lavoie*<br><br>14:15 - 15:00<br>**Mise en place d'une pratique en vue de l'évaluation de la pression artérielle et des facteurs de risque de maladie athéroscléreuse**<br>*Prof. Lyne Cloutier* |
| 15:00 - 15:30 | **BREAK** | | |

# SCIENTIFIC PROGRAM

## FRIDAY, OCTOBER 17, 2014

| | BIOMEDICAL RESEARCH TRACK | CLINICAL/OUTCOMES/ POPULATION RESEARCH TRACK | PRIMARY CARE TRACK ROOM: CHAUDIÈRE C |
|---|---|---|---|
| 15:30 - 17:00 | **POSTER SESSION # 2**<br>Hallway & Foyer<br><br>**Biomedical Research Track**<br>**P15 - Angiotensin-II (Ang-II)-induced expression of the early growth response protein 1 (Egr-1) is mediated by a Calcium-CaMKII-II – dependent pathway in vascular smooth muscle cells (VSMC)** - *Estelle Rolande Simo Cheyou*<br>**P16 - Involvement of MAPKs and PKB pathways in Insulin-like growth factor 1 (IGF-1)-induced Early Growth Response protein-1 (Egr-1) expression in A10 Vascular smooth muscle cells (VSMC)** - *Viktoria Youreva*<br>**P17 - Retrospective analysis of international normalized ratio variability in healthy patients taking the Vitamin K Antagonist Warfarin** - *David Casey*<br>**P18 - De novo production of reactive oxygen species by endothelial microparticles** - *Maddison Turner*<br>**P19 - Function and regulation of HCaRG in kidney damage** - *Carole Campion*<br>**P20 - Impact of the (pro) renin receptor on adipose tissue structure and function** - *Zulaykho Shamansurova*<br><br>**Clinical/Outcomes/Population Research Track**<br>**P21 - Salt reduction as a healthy recommendation in lowering blood pressure** - *Samia L.L. Rizk*<br>**P22 - Effectiveness of patient programs designed to increase physical activity or decrease salt consumption on blood pressure control** - *Sajal Jain*<br>**P23 - The Successful Experience thought Rondon Project of Brazil: An Experience Report** - *Willian Roger Dullius*<br>**P24 - Improving hypertension detection and management in the community – a nation-wide approach through a grocery/pharmacy chain** - *Shelley Diamond*<br>**P25 - To study the prevalence and factors affecting treatment of resistant hypertension** - *Shaylika Chauhan*<br>**P26 - Resistance Hypertension: A simplified etiological classification** - *Tawfik Albassam*<br>**P27 - Electrocardiogram-Assisted Blood Pressure Estimation in Patients with Atrial Fibrillation and other Chronic Conditions** - *Saif Ahmad*<br>**P28 - Use of Web-based app to provide clinical decision support for hypertension at point of care** - *Rahul Mehta* | | **PRIMARY CARE # 4**<br>*Co-chairs: Dr. Ally Prebtani & Dr. Donna McLean*<br><br>**Update on the management of hypertension related complications**<br><br>15:30 - 16:00<br>**Enhancing longevity in CAD: Doing what we know**<br>*Dr. James Stone*<br><br>16:00 - 16:30<br>**Erectile Dysfunction**<br>*Dr. Gerald Brock*<br><br>16:30 - 17:00<br>**Atrial Fibrillation: A step-wise approach for primary care**<br>*Dr. Allan Skanes* |
| 17:00 - 19:00 | **DINNER IN THE PETIT CHÂTEAU** | | |

# SCIENTIFIC PROGRAM

## SATURDAY, OCTOBER 18, 2014

| | BIOMEDICAL RESEARCH TRACK ROOM: CHAUDIÈRE A | CLINICAL/OUTCOMES/ POPULATION RESEARCH TRACK ROOM: CHAUDIÈRE B | PRIMARY CARE TRACK ROOM: CHAUDIÈRE B |
|---|---|---|---|
| 07:00 - 8:00 | **BREAKFAST IN THE PETIT CHÂTEAU** | | |
| 08:00 - 09:30 | **BIOMEDICAL # 3**<br>*Co-chairs: Dr. Jeffrey Dickhout & Dr. Stephen Ferguson*<br><br>08:00 - 08:30<br>**STATE OF THE ART**<br>**GRK2 Targeted Knock-down Results in Spontaneous Hypertension and Altered Vascular GPCR Signaling**<br>*Dr. Stephen Ferguson*<br><br>08:30<br>**Mechanism implicated in the beneficial effects of (pro) renin/renin receptor blockade on weight gain and insulin sensitivity in obese mice**<br>*Paul Tan*<br><br>08:45<br>**Endothelin-1 overexpression exaggerates diabetes-induced endothelial dysfunction**<br>*Noureddine Idris-Khodja*<br><br>09:00<br>**Endoplasmic reticulum stress inhibition prevents chronic kidney disease independent of blood pressure lowering effects in the Dahl S raT**<br>*Rachel E. Carlisle*<br><br>09:15<br>**A somite-derived Sox2+ stem cell in the adult mouse aorta gives rise to myeloid cells**<br>*Sarah Steinbach* | **CLINICAL # 3**<br>*Co-chairs: Dr. Paul Timothy Pollak & Dr. Ross Feldman*<br><br>08:00<br>**SD-OCT Measurement of Serial Change in Retinal Macular Thickness During Normal Pregnancy: Proof-of-Concept Data for a Tool to Study Dynamic Changes in Function of the Microcirculation in Humans**<br>*Robert Herman*<br><br>08:15<br>**Arbitrary pharmacy switching between differing nifedipine osmotic delivery formulations leads to unexpected variability in blood pressure for majority of patients**<br>*Paul Timothy Pollak*<br><br>08:30<br>**Hypertension Treatment and Control in the Community: A novel program of surveillance for hypertension in a grocery-pharmacy setting**<br>*Shelley Diamond*<br><br>08:45<br>**Psychosocial work factors and ambulatory blood pressure: repeated exposure to demand-control and effort reward imbalance models over 5 years**<br>*Xavier Trudel*<br><br>09:00 - 09:30<br>*JACQUES DE CHAMPLAIN NEW INVESTIGATOR AWARD LECTURE*<br>**Arterial Stiffness and Hemodynamics: from Physiology to Clinical Practice**<br>*Dr. Stella Daskalopoulou* | |
| 09:30 - 10:00 | **BREAK** | | |

# SCIENTIFIC PROGRAM

## SATURDAY, OCTOBER 18, 2014

| | BIOMEDICAL RESEARCH TRACK ROOM: CHAUDIÈRE A | CLINICAL/OUTCOMES/ POPULATION RESEARCH TRACK ROOM: CHAUDIÈRE B | PRIMARY CARE TRACK ROOM: CHAUDIÈRE B |
|---|---|---|---|
| 10:00 - 11:30 | **BIOMEDICAL # 4**<br>*Co-chairs: Dr. Pierre Paradis & Dr. Robert Gros*<br><br>10:00<br>**Systemic Inflammation, Hypertension, and Changes in Middle Cerebral Artery Function**<br>*Amy Randell*<br><br>10:15<br>**c-Myb regulates specific Sca1+ adventitial cell populations following injury**<br>*Eric Shikatanl*<br><br>10:30<br>**ER stress inhibition decreases inflammatory response in a CKD mouse model**<br>*Zahraa Mohammed-Ali*<br><br>10:45<br>**Endothelin-1 overexpression preserves endothelial function in mice with vascular smooth muscle cell-specific deletion of PPAR-gamma**<br>*Sofiane Ouerd*<br><br>11:00 - 11:30<br>**STATE OF THE ART**<br>**An Independent Adrenergic System in Perivascular Adipose Tissue**<br>*Dr. Stephanie Watts* | | **2015 DRAFT CHEP RECOMMENDADTIONS SYMPOSIUM**<br>*Co-chairs: Luc Poirier & Dr. Raj Padwal*<br><br>10:00 - 10:05<br>**Opening Remarks**<br>*Luc Poirier*<br><br>10:05 - 10:20<br>**eLearning Initiatives**<br>*Dr. Denis Drouin & Dr. Guy Tremblay*<br><br>10:20 - 11:20<br>**New Evidence and the 2015 Draft CHEP Recommendations**<br>*Dr. Doreen Rabi, Luc Poirier & Dr. Raj Padwal*<br><br>11:20 - 11:30<br>**Q&A and Closing Comments**<br>*Dr. Raj Padwal* |
| 11:30 - 13:30 | **LUNCH IN THE PETIT CHÂTEAU** | | |

# SCIENTIFIC PROGRAM

## SATURDAY, OCTOBER 18, 2014

| | |
|---|---|
| 13:30 - 15:00 | **HYPERTENSION CANADA AWARDS LECTURE**<br><br>13:30 - 14:00<br>GEORGE FODOR AWARD LECTURE<br>**Salt**<br>*Bill Jeffery*<br><br>14:00 - 14:30<br>SENIOR INVESTIGATOR AWARD LECTURE<br>**Should publicly funded health care be managing obesity, its comorbidities, or both?**<br>*Dr. Raj Padwal*<br><br>14:30 - 15:00<br>SENIOR INVESTIGATOR AWARD LECTURE<br>**An eclectic voyage of discovery in hypertension**<br>*Dr. Alexander Logan*<br><br>Room: Chaudière B |
| 15:00 - 15:30 | **BREAK** |
| 15:30 - 17:30 | **ANNUAL GENERAL MEETING,** Dr. Ernesto Schiffrin, President and Chair<br>**AWARDS CEREMONY,** Dr. Ernesto Schiffrin, President and Chair<br>**CLOSING RECEPTION,** Dr. Ross Feldman, Congress Chair<br>Room: Chaudière A |

# NOTES

Congrès Hypertension Canada

Canadian Hypertension Congress

# AWARD RECIPIENTS

# AWARD RECIPIENTS

## 2014 GEORGE FODOR AWARD

**Bill Jeffery, LLB, National Coordinator**
Centre for Science in the Public Interest (CSPI)
Ottawa, Ontario

The 2014 George Fodor Award recognizes Bill Jeffery as the single most effective voice nationally, in keeping healthy eating, especially sodium reduction on the national agenda.

Since 2002 Mr. Jeffery directly helped draft, informed or influenced:

- Seven federal and provincial private member's bills,
- Two House of Commons motions,
- Three sets of framework federal regulations,
- One government bill,
- Two major federal/provincial/territorial Health Minister's reports,
- One meeting of First Ministers,
- Two high level United Nations processes,
- One WHO technical process,
- The coordination of networks to support Canadian and international networks representing up to 2,000 non-profit health and citizens groups on various proposals including; nutrition labeling, restaurant menu labeling, sodium reduction, restrictions on advertising to children, improved allergen labeling, school nutrition programs, and safeguard against conflicts of interest in nutrition policy, and
- Two major Codex Food Labeling Committee global standards.

## 2014 DISTINGUISHED SERVICE AWARD

**Simon W Rabkin MD, FRCPC, FACC**
Professor of Medicine
University of British Columbia
Cardiology Staff, Vancouver Hospital
Cardiology Staff Healthy Heart Program Saint Paul's Hospital
Vancouver, British Columbia

The 2014 Distinguished Service Award recognizes Dr. Rabkin's significant voluntary service contributions to Hypertension Canada and his prolonged contributions in particular to the Canadian Hypertension Society throughout his career. Over the years, Dr. Rabkin has served as a; member of the Abstract Review committee, Board Member, Chairman of the Awards committee, member of the Strategic Planning Group, Executive member and President of the Canadian Hypertension Society. He was instrumental in bringing the 23rd International Society of Hypertension Congress to Vancouver in 2010 and was President of this very successful meeting with approximately 2,500 attendees. This meeting raised the profile of Canadian hypertension research, and has given opportunity to many senior and junior Canadian scientists, as well as students, residents and postdoctoral fellows to communicate to an international audience.

# AWARD RECIPIENTS

## 2014 SENIOR INVESTIGATOR AWARD

**Alexander G. Logan, MD, FRCPC**
Professor of Medicine, University of Toronto
Lunenfeld-Tanenbaum Research Institute
Mount Sinai Hospital
Toronto, Ontario

The 2014 Senior Investigator Award recognizes Dr. Logan's 45 year commitment to hypertension research, education, diagnosis and treatment. Seeking deeper understanding of hypertension mechanisms, more rational and effective therapy, and its efficient implementation across vulnerable populations, Dr. Logan has over the course of his career, engaged the full spectrum of discovery science: genetic, molecular and cellular, integrative human, clinical trial, epidemiology, health care delivery, knowledge translation, evaluative and population health research.

Highlights of Dr. Logan's career include:

- His 181 peer-reviewed scientific publications, plus 3 Editorials, 6 Review Articles, and 24 non peer-reviewed articles
- The principal investigator on peer-reviewed grants
- The first or senior author of 9 publications in the leading hypertension focused journals
- Important national leadership positions within the hypertension community
- Decade's long interest in nutritional contributions to blood pressure
- Service on Editorial Boards
- Long standing commitment to knowledge translation
- Commitment to building research and clinical capacity in hypertension

# AWARD RECIPIENTS

## 2014 SENIOR INVESTIGATOR AWARD

**Raj S. Padwal, MD, MSc, FRCPC**
Internal Medicine and Clinical Pharmacology
Director, Hypertension Clinic
University of Alberta
Edmonton, Alberta

The 2014 Senior Investigator Award recognizes Dr. Padwal' s unique, dynamic and extensive background as a general internist, clinical pharmacologist, clinical epidemiologist and health services researcher with an interest in cardiovascular risk management and health care delivery. Since 2003 he has conducted research spanning the entire health services spectrum, including large scale administrative database analyses, cross-sectional analyses of Canadian national survey data, primary data collection and longitudinal data analysis, systematic review/meta-analysis, randomized controlled trials, as well as the creation of national clinical guidelines (CHEP).

Dr. Padwal's work includes:

- Co-investigator in the award-winning Care Transformation Initiative
- Co-investigator on a recently awarded CIHR grant to further evaluate Care Transformation
- Co-PI on a recently awarded PRIHS examining the impact of an elder-friendly surgical unit on health care outcomes
- Past-President of the Internal Medicine section of the Alberta Medical Association
- Medical Lead for the Capital Health Regional Obesity Initiative
- Lead of a worksite cardiovascular risk screening and prevention program in the oil sector
- Chair of the Recommendations  Task Force and Central Review Committee

In addition, Dr. Padwal has a proven track record of securing grant funds, for publishing over 150 papers and book chapters, for being cited nearly 5800 times, for supervising 20 research trainees and acting as a reviewer for a number of funding agencies.

# AWARD RECIPIENTS

## 2014 JACQUES DE CHAMPLAIN NEW INVESTIGATOR AWARD

**Styliani Stella Daskalopoulou**
**MD, MSc, Diploma of Imperial College (DIC), PhD**
General Internist
Fonds de la recherche en santé du Québec Chercheur-boursier clinicien Junior 2
Associate Professor (Tenured) in Medicine
McGill University Health Centre and McGill University
Montréal, Québec

The 2014 Jacques de Champlain New Investigator Award recognizes and celebrates Dr. Daskalopoulou' s great skills in leadership through her work with the MeasureBP Canadian Institutes of Health Research, the Canadian Hypertension Education Program BP measurement group and the Central Review Committee. Dr. Daskalopoulou is making significant contributions in the area of hypertension and vessel hemodynamics by the identification of early markers of vascular impairment and maintenance of vascular health. Despite the fact that she is in the early stage of her career development, she has demonstrated excellent productivity with more than 90 publications, 1775 citations and h-index of 25. Her level of research funding is equally impressive.

We acknowledge and thank Dr. Daskalopoulou for:

- Her interest in women's health during their lifespan and her studies in the evolution of blood pressure variability from the perimenopausal to the postmenopausal period
- Her randomized control trial to determine the effect of dietary calcium intake as compared to supplemental calcium on the vascular system
- Her research in the area of pre-eclampsia and hypertensive disorders of pregnancy
- Examining the effect of smoking and smoking cessation on arterial stiffness and vessels hemodynamics, including central blood pressure, as well as the endothelium, in young healthy individuals
- Bridging the gap between patient care, research, and teaching by establishing and directing the Vascular Health Clinic and co-directing the Hypertension Clinic at the McGill University Health Centre
- Leading the effort to create an electronic database including all clinical data gathered to provide unique research opportunities in the area of hypertension
- The research opportunities offered to over 70 trainees through your Vascular Health Unit

# AWARD RECIPIENTS

## 2014 CERTIFICATES OF EXCELLENCE

The Certificate of Excellence is awarded in recognition of outstanding efforts and contributions in Canada, to increase public awareness, prevention and control of hypertension.

**Charlotte Jones, PhD, MD., FRCPC**
Associate Professor of Medicine
Director of student research
Southern Medical Program
University of British Columbia,
Okanagan Campus

The 2014 Certificate of Excellence recognizes Dr. Jones' substantial contributions to clinical care and research in the area of hypertension. We applaud her work in developing and implementing CHAMP (Cardiovascular health, awareness and management program) programs, which target identification and management of uncontrolled cardiovascular risk factors in the community. As well, her efforts towards developing a collaborative inter-professional program uniting nursing, pharmacy and medical students from the Universities of Alberta and Calgary to plan and implement CHAMP programs, of which hypertension management and control are a major component. The fact the programs are being disseminated throughout the Province of Alberta, and beyond, in rural, urban, multicultural and worksite communities, truly brings hypertension identification and control to the high risk and difficult to reach populations.

Dr. Jones has been an active member of the CHEP program and hypertension guideline development, as well as a leader in the "train-the-trainer" programs offered as part of the KT strategies for dissemination and uptake of the guidelines.

**Hude Quan, MD**
Professor, Department of Community Health Services
University of Calgary
Calgary, Alberta

The 2014 Certificate of Excellence recognizes Dr. Quan's substantial contributions to research in the area of hypertension. We applaud the key study he led, funded by CIHR, to validate an algorithm to define hypertension using administrative data. This work enabled the study of hypertension using administrative data from across Canada.

As the PI for the Hypertension Outcomes Surveillance Team (HOST), Dr. Quan's work has greatly enhanced our understanding of the outcomes associated with hypertension, and informed intervention strategies to improve hypertension management and outcomes. His research has been published in major journals and has placed Canada as a leader in the field of health services research in hypertension.

# AWARD RECIPIENTS

## 2014 CERTIFICATES OF EXCELLENCE

**Carly Weeks**
Health Reporter
The Globe and Mail
Toronto, Ontario

The 2014 Certificate of Excellence recognizes Ms. Weeks' substantial contributions to the people of Canada as a health reporter. Her more than 300 articles that focus mainly on health and consumer issues shine a light on important issues that impact the health and well-being of Canadians. Specifically the following articles have increased knowledge of healthy eating and lifestyle choices that can promote healthy behavior changes that improve cardiovascular health.

- Reality check: All you think you know about fat is wrong
- Men with heart attack symptoms treated faster than women, study shows
- Belly size beats BMI for assessing fully health picture, Mayo Clinic study shows
- Experts blast Health Canada's approach to sodium reduction
- Healthy food policies at risk, scientists say
- Is your toddler eating too much salt?
- Canadians – and lots of them – want to lower sodium in food, survey finds

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Loblaw
COMPANIES LIMITED

**Loblaw Companies Limited**

The 2014 Certificate of Excellence recognizes their Blood Pressure Awareness Program launched in February 2014.

We salute this year round pharmacist-led intervention on blood pressure that includes:

- An in-store blood pressure reading
- A blood pressure tracker card
- A personalized assessment using a brief questionnaire and recommendations for current and future blood pressure management
- A phone follow up 8 weeks following the initial consultation to see if the person saw their physician, had a change or increased adherence to their medications, modified lifestyle, etc.
- A booklet provided, containing information on managing blood pressure, healthy eating for maintaining healthy blood pressure, shopping tips for reducing sodium, low-sodium recipes, and a sodium tracker tool
- A referral to an in-store dietitian where available

The pharmacist interventions will have a significant impact on Canadians, by creating awareness and understanding of an individual's blood pressure targets and the importance of monitoring it on a regular basis. It also increases knowledge of healthy eating and lifestyle choices that can promote healthy behavior changes, to improve and help control high blood pressure.

# AWARD RECIPIENTS

## 2014 CERTIFICATES OF EXCELLENCE

### Rogers Communications Inc.

The Certificate of Excellence awarded to Rogers Communications Inc. recognizes the excellent, well thought out and implemented bWell, Employee Wellness Program. This program initiated a variety of blood pressure programs reaching out to an estimated 100,000 Canadians (Rogers' employees and families across Canada). This aligns directly with a common vision that Hypertension Canada shares with many partners in the health field: Canadians will have the healthiest and best managed blood pressure in the world. We are impressed by the bWell website content, the annual national online health challenge to Rogers' employees, the annual bWell Health, Safety & Wellness Fair, and the availability of free, on-site 20 minute cardiovascular screening assessments. These initiatives will have a significant impact on Canadians, by creating awareness and understanding of an individual's blood pressure targets and the importance of monitoring it on a regular basis. It also increases knowledge of healthy eating and lifestyle choices that can promote healthy behavior changes, to improve and help control high blood pressure.

Congrès Hypertension Canada

Canadian Hypertension Congress

# ABSTRACT GUIDE

## BIOMEDICAL POSTER SESSION #1: 17:30 – 18:30: HALL & FOYER

### P1 - Effects of podocyte microparticles on cultured human proximal tubule cells

*Shareef Akbari, Maddison Turner, Dylan Burger. University of Ottawa, Ottawa Hospital Research Institute*

**Background:** Microparticles (MPs) are small (0.1-1.0), membranous vesicles shed from the cell surface following stress/injury. Our laboratory and others have shown that endothelial 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 in podocytes following mechanical stretch (a mimic of intraglomerular hypertension). However whether podocyte MPs interact with the proximal tubule and influence function is unknown. Methods: Cultured human proximal tubule cells were exposed to podocyte MPs (105/ml) and phosphorylation of p38, ERK 1/2, and JNK and expression of fibronectin were examined by Western blot analysis. Reactive oxygen species (ROS) production was assessed by lucigenin chemiluminescence. Results: p38 phosphorylation was increased (~3 fold) after 30 minutes exposure to podocyte microparticles. By contrast, JNK and ERK phosphorylation levels were unchanged over 24 hours. In addition, ROS production was shown increased by podocyte MP treatment at 16 hours (control: 7.2±1.6 vs treated 15.5±1.4 AU/mg protein). Expression of fibronectin was significantly increased following 72 hour treatment with pMPs ($p<0.05$, $n=6$).Fluorescence microscopy revealed cell surface binding of podocyte MPs to proximal tubule cells suggesting a paracrine effect. **Conclusion:** Our results suggest that podocyte MP interact with proximal tubule epithelial cells and induce intracellular signaling, ROS production, and fibrosis. Such effects may play a role in the development of tubular injury in hypertensive and diabetic nephropathy.

## BIOMEDICAL POSTER SESSION #1: 17:30 – 18:30: HALL & FOYER

### P2 - Cardio protective role of the cholinergic system

*Mouhamed Dakroub, Dr. Robert Gros, Departments of Medicine and of Physiology and Pharmacology, Western University, London, Ontario*

**Background:** The cholinergic system plays a crucial role in heart function. The vesicular acetylcholine transporter (VAChT) functions alongside the other cholinergic enzymes to secrete the parasympathetic neurotransmitter acetylcholine (ACh). Our lab has previously shown that VAChT knockdown mice suffer from a wide range of cardiovascular complications. The purpose of our research is to determine whether an overexpression of the VAChT gene can act as a cardio protective mechanism in aged mice. **Methods/Results:** Channel rhodopsin-2 (ChR2) transgenic mice express a two-fold increase in atrial and ventricular VAChT gene expression, as determined by real-time qPCR. This results in a two-fold increase in ACh in the atria of the heart. ChR2 mice show no metabolic differences when compared to aged-matched wild-type mice. However, when the mice are stressed, a multitude of cardiovascular differences are observed. When compared with wild-type mice, ChR2 mice run for longer periods of time when exercised on an animal treadmill until exhaustion. When implanted with telemeter electrocardiography devices, they also show greater vagal heart control after this exercise period. Ejection fraction and fractional shortening data obtained from echocardiogram recordings show ChR2 mice have overall better heart function when compared with wild-type mice. Wild-type mice show increased gene expression levels of ventricular cardiac damage markers such as atrial natriuretic factor and myosin heavy chain 7.
**Conclusion:** Our findings suggest that an increase in VAChT gene expression and subsequent elevation in ACh levels in the heart may be cardio protective and help counteract some of the detrimental effects that aging may cause to the heart.

# ABSTRACTS
## Thursday, October 16

## BIOMEDICAL POSTER SESSION #1: 17:30 – 18:30: HALL & FOYER

### P3 - Natriuretic peptide receptor-C agonist attenuates the expression of cell cycle proteins and proliferation of vascular smooth muscle cells from spontaneously hypertensive rats: role of Gi proteins and MAPkinase/ PI3kinase signaling

*Yuan Li, Jasmine El Andalousi; Madhu B. Anand-Srivastava*

**Background:** Vascular smooth muscle cells (VSMC) from spontaneously hypertensive rats (SHR) exhibit hyperproliferation and overexpression of cell cycle proteins. We earlier showed that small peptide fragments of cytoplasmic domain of natriuretic receptor-C (NPR-C) attenuate vasoactive peptide-induced hyperproliferation of VSMC. The present study investigated if C-ANP4-23, a specific agonist of NPR-C, could attenuate the hyperproliferation of VSMC from SHR by inhibiting the overexpression of cell cycle proteins and examine the underlying signaling pathways contributing to this inhibition. The proliferation of VSMC was determined by [3H] thymidine incorporation and the expression of proteins was determined by Western blotting. The hyperproliferation of VSMC from SHR and overexpression of cyclin D1, cyclin A, cyclin E, cyclin-dependent kinase 2 (cdk2), phosphorylated retinoblastoma protein (pRb), Giα proteins and enhanced phosphorylation of ERK1/2 and AKT exhibited by VSMC from SHR were attenuated by C-ANP4-23 to control levels. In addition, in vivo treatment of SHR with C-ANP4-23 also attenuated the enhanced proliferation of VSMC. Furthermore, PD98059, wortmannin and pertussis toxin, the inhibitors of MAP kinase, PI3kinase and Giα proteins respectively, also attenuated the hyperproliferation of VSMC from SHR and overexpression of cell cycle proteins to control levels. These results indicate that NPR-C activation by C-ANP4-23 attenuates the enhanced levels of cell cycle proteins through the inhibition of enhanced expression of Giαproteins and enhanced activation of MAPkinase/PI3kinase and results in the attenuation of hyperproliferation of VSMC from SHR. It may be suggested that C-ANP4-23 could be used as a therapeutic agent in the treatment of vascular complications associated with hypertension, atherosclerosis and restenosis (Supported by grant from Canadian Institutes of Health Research).

## BIOMEDICAL POSTER SESSION #1: 17:30 – 18:30: HALL & FOYER

### P4 - Role of vascular smooth muscle cell PPARγ in aldosterone-induced vascular injury

*Michelle Trindade, Hypertension and Vascular Research Unit, Lady Davis Institute for Medical , Department of Clinical Medicine, State University of Rio de Janeiro, Rio de Janeiro, Brazil; Sofiane Ouerd, Hypertension and Vascular Research Unit, Lady Davis Institute for Medical Research, Noureddine Idris-Khodja, Hypertension and Vascular Research Unit, Lady Davis Institute for Medical Research, Tlili Barhoumi, Hypertension and Vascular Research Unit, Lady Davis Institute for Medical Research, Stefan Offermanns, Department of Pharmacology, Max-Planck-Institute for Heart and Lung Research, Bad Nauheim, Germany, Frank J. Gonzalez, Laboratory of Metabolism, Division of Basic Sciences, National Cancer Institute, National Institutes of Health, Bethesda, Maryland, USA, Pierre Paradis, Hypertension and Vascular Research Unit, Lady Davis Institute for Medical Research, and Ernesto L. Schiffrin, Hypertension and Vascular Research Unit, Lady Davis Institute for Medical Research, Department of Medicine, Sir Mortimer B. Davis-Jewish General Hospital, McGill University, Montréal, Québec, Canada*

**Background:** Peroxisome proliferator activated receptor γ (PPARγ) agonists improve vascular remodeling and endothelial dysfunction in hypertensive rodents and humans. PPARγ activation in vascular smooth muscle cells (VSMC) may be responsible for the vascular protective effects of PPARγ agonists. We previously observed a protective role of VSMC PPARγ in angiotensin II-induced endothelial dysfunction and vascular remodeling. However, it is unknown whether VSMC PPARγ plays a similar protective role on adverse vascular effects of aldosterone. We hypothesized that inactivation of the Ppar gene in VSMC (smPparγ-/-) would exaggerate aldosterone-induced vascular injury.
**Methods/Results:** Using a tamoxifen-inducible Cre/loxP system, Pparγ was ablated in VSMC of adult mice. Thirteen week-old control and smPparγ-/- mice were infused or not with aldosterone (400 µg/kg/d, SC) for 14 days while receiving 1% NaCl/0.3% KCl in drinking water. Endothelial function and vascular remodeling were assessed in mesenteric arteries (MA) using pressurized myography. Endothelium-dependent relaxation (EDR) responses to acetylcholine were reduced to a similar extent in smPparγ-/- and aldosterone-treated control and smPparγ-/- compared to control mice (Emax: 62.5±8.7%, 50.8±8.6% and 56.8±7.9%, respectively, vs 86.4±3.2%, $P<0.05$). L-NAME, an inhibitor of nitric oxide (NO) synthase, completely blocked EDR in the four groups. Endothelium-independent relaxation response to the NO donor sodium nitroprusside and contractile responses to norepinephrine were similar in the four groups. Preliminary data indicated that aldosterone tended to increase MA stiffness in control mice, as shown by a leftward shift of the stress/strain curve (strain at 140 mmHg, 0.79±0.07 vs 0.89±0.03). Furthermore, Pparγ deletion induced an increase in MA stiffness compared to control mice, which was not worsened by aldosterone (strain at 140 mmHg, 0.67±0.01, 0.65±0.03). **Conclusion:** These results indicate that either VMSC Pparγ inactivation or aldosterone treatment induce vascular remodeling and endothelial dysfunction, which are not mutually exaggerated, suggesting that PPARγ and aldosterone act through different pathways.

## BIOMEDICAL POSTER SESSION #1: 17:30 – 18:30: HALL & FOYER

### P5 - SKP-derived vascular smooth muscle cells exhibit type II diabetes associated dysfunction

*SK Steinbach[1], TK Waddell[2], M Ouzounian[3], TM Yau[3], M Husain[1,4-7] [1]McEwenCentreforRegenerativeMedicine, Division of Experimental Therapeutics- [2]Respiratory and Critical Care, & [3]Cardiovascular, Departments of [4]Medicine, [5]Physiology, [6]Laboratory Medicine and Pathophysiology and the [7]Heart and Stroke Richard Lewar Centre of Excellence*

**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:** 125 patients were recruited for this study. 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, PIdren. 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. 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 vascular dysfunction.

## BIOMEDICAL POSTER SESSION #1: 17:30 – 18:30: HALL & FOYER

### P6 - MicroRNA and regulation of angiotensin II-induced vascular injury

*Kugeng Huo, [1] Tlili Barhoumi, [1] Chantal Richer, [3] Virginie Saillour, [3] Daniel Sinnett, Pierre Paradis, [1] Ernesto L. Schiffrin[1,2] [1]Hypertension and Vascular 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:** MicroRNAs (miRNAs) are master gene regulators that 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 miRNAs are involved in the development of vascular injury. Recent miRNA profiling studies using next generation sequencing (NGS) revealed that not all miRNAs had been identified. We hypothesize that miRNAs mediate vascular remodeling and plays an important role in the pathophysiology of hypertension. We aim to use NGS to profile known and novel miRNAs in resistance arteries of a mouse hypertensive model. **Method/Results:** Ten to twelve week-old male C57BL/6 mice were treated or not with angiotensin II for 7 or 14 days. Blood pressure was measured by telemetry. Total RNA was extracted from mesenteric arteries using the mirVANA kit. RNA quantity and quality were tested by NanoDrop 2000 spectrophotometer and Agilent 2100 Bioanalyzer. Total RNA was enriched in small RNAs using the PureLink miRNA kit and used to construct libraries for deep sequencing by the SOLiD 5500 NGS platform. Novel miRNAs and miRNA targets were predicted using miRDeep* and TargetScan, respectively.mBoth 7-day and 14-day angiotensin II-treated mice exhibited a ~40 mmHg systolic and diastolic blood pressure elevation (P5µg) of high-quality total RNA (RNA integrity number>8.0) was isolated to allow miRNA and mRNA profiling and RT-qPCR validation. In the preliminary test, 10.4 million mapped-reads were obtained, allowing miRNA sequencing in a discovery mode. The sequence analysis identified 395 known miRNAs. miRDeep* predicted 194 novel miRNAs. TargetScan predicted 42 to 9463 target mRNAs per miRNA. **Conclusions:** Sufficient high-quality RNA was isolated to allow miRNA and mRNA profiling and RT-qPCR validation. This preliminary NGS study demonstrates the feasibility of the research proposal and enables us to profile miRNA in a discovery mode.

# ABSTRACTS
## Thursday, October 16

## BIOMEDICAL POSTER SESSION #1: 17:30 – 18:30: HALL & FOYER

### P7 - Differences in renal pathological responses found between salt sensitive and salt insensitive hypertension

*Victoria Yum, Chao Lu, Jeffrey G. Dickhout Department of Medicine, Division of Nephrology, McMaster University, St. Joseph's Healthcare Hamilton, ON, Canada*

**Background:** Amongst hypertensives, there are populations that are relatively salt sensitive or insensitive. Salt sensitive individuals appear to have reduced preglomerular resistance in response to a high salt diet that leads to elevated glomerular capillary pressure and renal damage. We used two animal models to represent either group: the Dahl salt sensitive (Dahl S) rat and the spontaneously hypertensive rat (SHR). We studied whether differences in preglomerular vessel function contributes to organ protection in the salt insensitive SHR. **Methods:** Animals were fed a high salt diet (HS; 8% NaCl) or a normal salt diet (NS; 0.4% NaCl) for 4 weeks. Blood pressure was measured with tail cuff plethysmography and 24-hour urine collected for measurement of protein and albumin excretion. After 4 weeks kidneys were harvested. Arcuate arteries were dissected from kidneys and transferred to a pressurized myograph to assess myogenic constriction. Kidney sections were stained with PAS to observe renal pathology. **Results:** Regardless of diet, SHR animals demonstrated significantly higher blood pressure than Dahl S rats. Dahl S rats demonstrated blood pressure salt sensitivity after 4 weeks of HS, this was not found in the SHR. HS fed Dahl S rats demonstrated increased renal damage as shown by proteinuria, albuminuria, and protein cast formation. SHRs did not show increased renal pathology with HS feeding. Further, SHR demonstrated less protein and albumin excretion as well as less protein cast formation compared to Dahl S regardless of diet. HS feeding significantly attenuated arcuate artery myogenic constriction in both SHR and Dahl S. **Conclusions:** Despite greater elevation of blood pressure in SHRs, these animals were resistant to renal damage compared to the Dahl S. The myogenic response of arcuate arteries was attenuated in both strains by HS feeding, and therefore cannot solely explain the renoprotection found in the SHR on a HS diet.

## CLINICAL/OUTCOMES/POPULATION POSTER SESSION #1: 17:30 – 18:30: HALL & FOYER

### P8 - Are Home Blood Pressure Monitors Accurate Compared to Validated Devices?

*Eva Bruketa, Swapnil Hiremath Division of Nephrology The Ottawa Hospital, Marcel Ruzicka Division of Nephrology The Ottawa Hospital and Ayub Akbari Division of Nephrology The Ottawa Hospital*

**Background:** Hypertension remains the most common cause of morbidity and mortality from cardiovascular disease. Major guidelines recommend home blood pressure (BP) monitoring to guide diagnosis and treatment of hypertensive patients. Notwithstanding this, little data exists on the real-world accuracy of home BP monitors used by patients. The objective of our study was to compare BP measurements from Home BP monitors with validated mercury sphygmomanometers. **Method:** We conducted a retrospective chart review and abstracted data from the charts of patients who had their home blood pressure monitor validated in the hypertension clinic at a tertiary care academic centre. After casual measurements, 3 resting measurements are taken with the home monitor and a validated office mercury sphygmomanometer using standard methods. The means of these three measurements taken with each method were compared with the t-test. In addition, we analyzed the proportion of home BP readings within 5 and 10 mm Hg of the mercury readings. **Results:** We analyzed data from 210 patients (60% men, mean age 67 + 14 years). The mean body mass index was 29 kg/m^2, and the mean arm circumference was 32 + 5 cms. There was a significant difference between the resting mercury systolic blood pressure (SBP) (132.3 +18.5 mm Hg) compared to the home monitor resting SBP (133.4 mm Hg + 18.1 mm Hg), p = 0.011. For diastolic BP (DBP) the difference was 71.4 + 11.9 versus 68.9 + 12.7 mm Hg (p 5 mm Hg different and 16 (8%) were > 10 mm Hg different from the mercury SBP measurement. For DBP, these proportions were 32% (67/210) and 9% (18/210) respectively.

## CLINICAL/OUTCOMES/POPULATION POSTER SESSION #1: 17:30 – 18:30: HALL & FOYER

### P9 - Indexing Left Ventricular Mass

*Sharmila Udupa (Division of Cardiology, Department of Pediatrics, University of Ottawa, Children's Hospital of Eastern Ontario, Ottawa, Canada), Janusz Feber (Division of Nephrology, Department of Pediatrics, University of Ottawa, Children's Hospital of Eastern Ontario, Ottawa, Canada), Letizia Gardin (Division of Cardiology, Department of Pediatrics, University of Ottawa, Children's Hospital of Eastern Ontario, Ottawa, Canada), Nick Barrowman (Clinical Research Unit, CHEO Research Institute, Department of Pediatrics, University of Ottawa, Children's Hospital of Eastern Ontario, Ottawa, Canada), Robert Gow (Division of Cardiology, Department of Pediatrics, University of Ottawa, Children's Hospital of Eastern Ontario, Ottawa, Canada)*

**Background:** Assessment of LVH helps determine disease severity and guide management decisions. Alternate methods of quantifying LV mass have been developed. Our study examines the reproducibility of various indexing methods and evaluates for classification errors of LVH due to indexing method. **Methods/Results:** A total of 230 transthoracic echocardiograms of children without complex congenital heart disease or cardiomyopathy were randomly selected: 100 with structurally normal hearts, 100 with hypertension, and 30 with LVH. LV mass was indexed to body surface area (BSA), height and height2.7, and to lean body mass (LBM). The primary variable of interest was indexed LV mass Z score. LVH was defined as a Z score ≥1.65. Reliability was evaluated using intraclass correlation coefficient (ICC (3,1)). Agreement between indexing methods was explored using Bland-Altman analysis. Classification of LVH was evaluated by percent agreement and kappa coefficient. There were 126 males (54.7%); mean age was 9.83 years. Mean Z score varied significantly by method, from -0.64 (BSA) to 1.41 (height2.7). Reliability between methods showed an ICC of 0.62. Bland-Altman analyses of Z scores revealed bias (-2.16 to 1.56) and wide limits of agreement between methods. The indexing method determined the proportion of individuals classified as having LVH. The percentage of absolute agreement was 68.7% amongst the entire cohort, with higher agreement among the three methods in children under the age of 5 years (85.3%) compared to all four methods in children over the age of 5 (61.7%). Kappa analysis showed low agreement for classification. There was no significant difference in classification errors between the three populations ("normals," "hypertension," and "LVH"). **Conclusion:** Classification of LVH is heavily biased and depends directly on indexing technique. There is poor reliability and agreement between the four key indexing methods, which are not interchangeable. Moving between Z score indexing methods could lead to inaccurate clinical decisions.

## CLINICAL/OUTCOMES/POPULATION POSTER SESSION #1: 17:30 – 18:30: HALL & FOYER

### P10 - Immune sensitization and mortality on the waiting list for kidney transplantation

*Sapir-Pichhadze R (University of Toronto), Tinckam K (University of Toronto), Laupacis (University of Toronto), Logan AG (University of Toronto), Beyene J (McMaster University), Kim SJ (University of Toronto)*

**Background:** Cardiovascular mortality is the leading cause of death in end-stage renal disease (ESRD). While innate and adaptive immunity have been shown to play a role in cardiovascular disease, the triggers of immune activity culminating in vascular injury and the role of humeral immunity are unknown. We evaluated whether anti-human leukocyte antigen antibodies, measured as panel reactive antibodies (PRA), are related to mortality in ESRD. **Methods:** We conducted a retrospective cohort study in first-time adult kidney transplant candidates using data from the Scientific Registry of Transplant Recipients and Centers for Medicare & Medicaid Services. The relationships between time-varying PRA categories and cardiovascular and all-cause mortality were assessed using Cox proportional hazards models. The analysis was repeated in sub cohorts of transplant candidates at low cardiovascular risk (based on traditional risk assessment and dialysis vintage), who were activated after 2007, and who were unsensitized upon activation. Competing risk analyses were also conducted. **Results:** Among 161,308 kidney transplant candidates, Cox proportional hazards models showed an increase in hazard ratios (HR [95% confidence interval]) for cardiovascular mortality (HR 1.05 [1.00, 1.10], 1.11 [1.05, 1.18], and 1.21 [1.12, 1.31]) and all-cause mortality (HR 1.01 [0.95, 1.08], 1.11 [1.02, 1.21], and 1.17 [1.05, 1.31]) in PRA 1-19%, PRA 20-79%, and PRA 80-100% categories compared with PRA 0%, respectively. The relationship between PRA and study endpoints was accentuated in low risk patients and competing risks models. The relationship persisted in sub cohorts activated after 2007, and who were unsensitized at activation. **Conclusions:** Our findings suggest PRA is an independent predictor of mortality in waitlisted kidney transplant candidates. The mechanisms by which PRA confers an added risk for mortality and whether transplantation modifies this risk in sensitized patients, warrant further study.

# ABSTRACTS
## Thursday, October 16

## CLINICAL/OUTCOMES/POPULATION POSTER SESSION #1: 17:30 – 18:30: HALL & FOYER

### P12 - Formation and management of an interdisciplinary research network in vascular health

*Amanda van Beinum, MSc[1,2], Shirley Mei, PhD1,2, Duncan Stewart, MD[1,2], Peter Liu, MD[1,3], Katie Lafferty, MBA [1,4] [1]Canadian Vascular Network, [2]Ottawa Hospital Research Institute, [3]University of Ottawa Heart Institute, [4]Canadian Partnership for Stroke Recovery*

**Background:** Medical treatment and research has traditionally focused on silos of disease such as heart disease, renal disease/diabetes, hypertension, stroke, and cognitive decline, to name a few. Vascular diseases and vascular aging are common causal pathways for many of these devastating chronic conditions, and yet there remains limited focus on the study and prevention of vascular disease specifically. Due to their complexity, vascular diseases present a unique and multifaceted challenge for the medical and research communities. This is not a problem that can be addressed by a single research team, instead, we argue that the complexity, size, and overlap of approaches to addressing vascular disease makes it a problem ideally tackled by a network of that transcends traditional barriers between specialties, and creates a new way of working together towards improving the health of Canadians. **Methods/Results:** Development of a national network of researchers, stakeholders, clinicians, and allied health professionals interested in coming together to improve vascular health has involved a series of meetings, teleconferences, and development of a consensus document. In the early stages of the network focus has been on retaining momentum and engagement of the network, as well as improving network presence. Moving forward, a formal network analysis of current strengths and weaknesses will provide a baseline to help identify and foster important connections. At the completion of 3 years, a formal evaluation will be conducted with our major funding bodies. At the completion of 5 years we hope to show an improvement in Canada's approach towards improving vascular health for all Canadians. **Conclusion:** Research networks are new in the scientific world but hold tremendous potential for addressing large, complex, and stubborn health problems. The Canadian Vascular Network is already showing promise as a catalyst for forming important partnerships within the Canadian health research community.

## CLINICAL/OUTCOMES/POPULATION POSTER SESSION #1: 17:30 – 18:30: HALL & FOYER

### P13 - Practice Audit of Renal Denervation Procedure for Drug-resistant Hypertension

*Praveena Sivapalan, John S. Floras1,5, Douglas J. Ing1,5, George D. Oreopoulos2,3,5, Dheeraj K. Rajan3, Coimbatore Srinivas4, Duminda N. Wijeysundera4 and Alexander G. Logan1. 1Department of Medicine, University Health Network and Mount Sinai Hospital, Toronto, Ontario, Canada; 2Department of Surgery, University Health Network, Toronto, Ontario, Canada; 3Department of Interventional Radiology, University Health Network, Toronto, Ontario, Canada; 4Department of Anesthesia, University Health Network, Toronto, Ontario, Canada and 5Peter Munk Cardiac Center, University Health Network, Toronto, Ontario, Canada*

**Background:** While two seminal but unblinded trials demonstrated substantive reductions in office blood pressure (BP) following renal denervation (RDN) in patients with drug-resistant hypertension (DRH), this was not confirmed in the large American randomized, sham-controlled Symplicity HTN-3 trial. Thus, we suspended our institution\'s RDN program pending audit of our "real-world" experience with the procedure, which we report herein. **Methods:** In a retrospective office chart review, we assessed the effects of RDN in patients with \'true\' DRH at 6- and 12-months after the procedure. Office BP was measured sitting quietly alone 5 times using an automated device.
Results: Of 105 patients referred for initial evaluation, 21 were eligible and agreed to have the procedure. BP averaged 170.0±26.3/89.6±18.8 (mean ± SD) mm Hg at baseline, 148.6 ± 23.0/81.9±19.0 at 6 months ($p = 0.01$ and 0.18 for systolic BP and diastolic BP, respectively) and 156.5±22.4/83.0±18.3 mm Hg at 1 year ($p = 0.16$ and 0.27). 63 and 42% of patients achieved a systolic BP reduction of > 10 mm Hg at 6 and 12 months, respectively. There was no significant change in the average number of antihypertensive drugs used at 6 months or 1 year ($p = 0.18$, 0.22). Serum creatinine, which averaged 90.4±22.9 umol/L at baseline, rose to 106.8±42.3 umol/L at 1 year ($p = 0.03$). There were 9 peri-procedural complications, including a renal artery dissection and acute renal failure, which resolved. **Conclusions:** RDN had no effect on diastolic BP and only lowered systolic BP significantly at 6 months. Moreover, there was a wide variation in BP response and we observed several serious complications with a trend towards worsened renal function. Based on the outcomes of this audit, we are reserving RDN for patients who are adherent to antihypertensive drug treatment and suspected to have neurogenic DRH.

## CLINICAL/OUTCOMES/POPULATION POSTER SESSION #1: 17:30 – 18:30: HALL & FOYER

### P14 - Hospitalized Diabetes with Hypertension and Other Co-morbid Conditions in Canada

*Andreas Wielgosz, Sulan Dai, MD, PhD, Jennifer McCrea-Logie, PhD, Ece Yilmaz, Public Health Agency of Canada*

**Background:** Canadians with diabetes have an increased burden of comorbidities, including hypertension, and complications that affect the health care system. Their health-adjusted life expectancy (HALE) is more than 10 years lower than that of individuals without diabetes. Objectives: To report hospital mortality and identify comorbid conditions associated with patients of all ages, hospitalized with diabetes in 2011-12, the most recent year that data are available. **Research Design and Methods:** The most responsible diagnosis (MRDx) of diabetes (ICD-10-CA: E10-E11) was identified from 2011-12 acute care hospital separations (Discharge Abstract Database) in all Canadian jurisdictions except Quebec. Hospital separation rates were calculated by 5-year age groups and sex. The most common comorbid (secondary) diagnoses were identified for hospital separations with diabetes as MRDx and mean length of stay for each comorbidity was determined. **Results:** The total number of hospital separations in 2011-12 in Canada with diabetes as the MRDx in all ages was 738,625, of which 57.7% concerned men. The median age for all hospitalized Canadians with diabetes was 60 years. The average length of stay (LOS) in hospital was 10.5 days for patients with diabetes, compared with 6.5 days for all hospitalizations. On average, there were 3.8 comorbid conditions for each diabetes hospital separation, compared with 2.4 comorbid conditions for all hospital separations. For all patients hospitalized with an MRDx of diabetes, there were 2.4% deaths. Hypertension (ICD-10-CA: I100; 7.0%, 7,845 occurrences) was the most common diagnosis associated with diabetes followed by renal disease (N0835, N0839, N179, N189; 6.0%, 6,756 occurrences) and peripheral angiopathy (I792; 3.8%, 4,284 occurrences). **Conclusions:** Diabetes is associated with vascular comorbidities including hypertension being the most common. Prevention of shared predisposing factors should reduce the burden of hospitalizations associated with diabetes.

# ABSTRACTS
## Friday, October 17

## BIOMEDICAL ORAL SESSION #1: 08:00 – 08:15: CHAUDIÈRE A

### Implication of renal angiotensin-(1-7) axis in the development of preeclampsia in previously hypertensive mice

*Aida Kasaei Roodsari, Dominique S. Genest[1], Stéphanie Falcao[1], Catherine Michel[1], Sonia Kajla[1], Jolanta[1, 3] Gutkowska and Julie L. Lavoie[1, 2, 3]*
*[1]CHUM Research Center, Department of [2]Kinesiology and [3]Medicine, University of Montreal and [3]Montreal Diabetic Research Center, Montréal, Québec, Canada*

**Introduction:** Gestational hypertensive disorders, such as preeclampsia, affect 6% to 8% of all pregnancies in North America, and are the leading cause of maternal mortality in industrialized countries, accounting for 16% of all deaths. Women with hypertension have an increased risk (15% to 25%) of developing preeclampsia. **Objective:** Our aim was to investigate the potential role of the angiotensin-(1-7) axis in preeclampsia superimposed on chronic hypertension. Method: We used female mice overexpressing human angiotensinogen and human renin (R+A+) as a model of preeclampsia superimposed on chronic hypertension. As we have shown that exercise improves preeclampsia features in this mouse model, to assess the functional role of the different parameters studies, we trained mice by placing them in cages with free access to an exercise wheel 4 weeks prior to and throughout pregnancy. At the end of gestation, mice were sacrificed to harvest and weigh fetus, placentas and kidneys. Blood pressure was measured by telemetry. **Results:** We found that the Mas receptor was decreased in the kidney of pregnant sedentary transgenic mice compared to their control littermates whereas this was significantly increased in the trained group. This may cause an increase in angiotensin-(1–7) effects which may counter the negative effects of Angiotensin II. We also found increased renal angiotensin converting enzyme type 2 in trained transgenic mice compared with sedentary mice. Supporting the functional contribution of this modulation, we found that most of the pathological features observed in sedentary transgenic mice were prevented in trained mice such as increased blood pressure and, proteinuria. This study demonstrates that modulation of the angiotensin-(1-7) axis seems to be a key mechanism in the development of preeclampsia superimposed on chronic hypertension, which can be improved by exercise training to prevent disease features in an animal model.

## BIOMEDICAL ORAL SESSION #1: 08:15 – 08:30: CHAUDIÈRE A

### High glucose increases endothelial microparticle formation, pro-oxidative activity and pro-coagulant activity

*Dylan Burger, Kevin D Burns*
*Kidney Research Centre, Ottawa Hospital Research Institute, University of Ottawa*

**Background:** Diabetic subjects exhibit elevated plasma levels of endothelial microparticles (eMPs) which predict risk of cardiovascular events. Emerging evidence suggests that eMPs may influence pathophysiological processes in a paracrine fashion. However the impact of high glucose on the formation and biological activity of eMPs is unknown. **Methods:** Human dermal microvascular endothelial cells (ECs) were cultured in media containing normal D-glucose (5.6 mM), 15 mM D-glucose or 25 D-mM glucose, with L-glucose as osmotic control. eMP levels were assessed by flow cytometry (Annexin V labeling) and nanoparticle tracking analysis (NTA). eMPs were isolated from the media of ECs subjected to high glucose and effects on endothelial reactive oxygen species (ROS) formation were assessed by dichlorofluorescin fluorescence. Effects of high glucose on eMP pro-coagulant activity were assessed by a procoagulant activity assay. **Results:** Exposure of ECs to high D-glucose caused a dose-dependent increase in eMP formation relative to osmotic controls. At 24 hours, there was a ~2.5-fold increase in eMP release associated with 25 mM D-glucose exposure ($P<0.01$, n=4-6). eMPs formed in 25 mM D-glucose displayed a greater diameter compared with normal D-glucose osmotic controls (25 mM: 260±14 nm vs 5.6 mM: 203±7nm vs. $P<0.05$, n=6). eMPs generated in 25 mM D-glucose were more potent inducers of EC ROS production than eMPs generated in 5.6 mM D-glucose (~6-fold vs ~2.5 fold increase vs. control, respectively; $P<0.05$, n=4). High glucose eMPs caused a ~3-fold increase in pro-coagulant activity compared with an equivalent amount of normal glucose eMPs ($P<0.001$, n=4). **Conclusions:** Our results suggest that elevated glucose is a potent inducer of endothelial MP formation while also increasing the pro-oxidative effects of endothelial MPs. Such effects may contribute to progressive endothelial injury in diabetes.

## BIOMEDICAL ORAL SESSION #1: 08:30 – 08:45: CHAUDIÈRE A

### New Insights on Mechanisms of Foamy Macrophage Induction and Persistence

*Marian Laderoute, Ph.D., Immune System Management Clinic & Lab, Ottawa*

**Background:** Foamy macrophages (FM) are believed to be central to atherosclerosis initiation and progression. Accumulating evidence suggests infections may be causally connected to atherosclerosis but it remains unclear how this might contribute to the induction of FM. Serendipitously, an in vitro method to induce FM in human cells was discovered and appeared to involve induction of a novel, endogenous foamy virus (FV). **Methods/Results:** Immature retroviral particles were found to bud into vacuoles as visualized by electron microscopy. The endogenous retrovirus was identified as human endogenous retrovirus K102 (HERV-K102) by sequencing excised RT-PCR HERV-K HML-2 pol transcripts. RNA expression was confirmed with a newly designed real time ddCt ratio method for HERV-K102 pol. DNA also accumulated during culture and evidence for both cDNA and integrated DNA was found. HERV-K102 Env was expressed as shown by immunohistology in foamy cells, and the Env was processed as shown by transblotting immunoprecipitated antigens. Particles were isolated from plasma of patients with autoimmune and/or acute viral infections but not when these patients entered remission. In contrast no particles were isolated from 30/30 normal adults but were found in 2/4 normal cord blood plasma samples. High maximal levels of particles (about 10e12 per ml of plasma) were demonstrated commonly in viral infections but were 7 logs lower in HIV-1 patients who are at higher risk of atherosclerosis. A five-fold increased gene copy number was found to be associated with protection against HIV-1 transmission. HERV-K102 was found to have salient features of non-pathogenic FV. **Conclusion:** A common cause of FM in humans appears to be the induction of a novel, endogenous, FV as an innate immune protector response to viral infections. Induction but failed release of HERV-K102 particles in HIV-1 patients might suggest FM abnormally accumulate in immunosuppressed individuals leading to atherosclerosis.

## BIOMEDICAL ORAL SESSION #1: 08:45 – 09:00: CHAUDIÈRE A

### Angiotensin II-induced vascular injury is counteracted by FOXP3+ T regulatory lymphocytes

*Muhammad Oneeb Rehman Mian, Tlili Barhoumi [1], Marie Briet [3], Adriana Cristina Ene [1], Asia Rehman [1], Pierre Paradis [1] and Ernesto L. Schiffrin [1,2]. Affiliations: [1] Hypertension and Vascular Research Unit, Lady Davis Institute for Medical Research, and [2] Department of Medicine, Sir Mortimer B. Davis-Jewish General Hospital, McGill University, Montreal, Quebec, Canada, and [3] INSERM U1083, CNRS UMR 6214, Centre Hospitalo-Universitaire d'Angers, Université d\'Angers, Angers, France*

**Background:** T effector lymphocytes contribute to vascular injury in angiotensin II-induced hypertension, but the role of T regulatory cells (Treg) is unclear. Angiotensin II-induced hypertension is blunted in T and B cell-deficient (Rag1-/-) mice, and restored with reconstitution of T cells. We hypothesized that adoptive transfer of FOXP3-deficient (Scurfy) vs. wild-type T cells will exacerbate angiotensin II-induced vascular damage in Rag1-/- mice. **Methods/Results:** Eleven-week old male Rag1-/- mice were injected IV with vehicle, 10 million wild-type or Scurfy T cells, 1 million CD4+CD25+ Treg alone or with Scurfy T cells, and 2 weeks later were infused or not with angiotensin II (490 ng/kg/min, SC) for 14 days (n=3-8). Telemetric systolic and diastolic blood pressure (SBP and DBP), and mesenteric arteries (MA) function and structure, reactive oxygen species (ROS) production and fibronectin expression were assessed. Angiotensin II induced a 40 mmHg SBP rise in vehicle, wild-type and Scurfy T cells groups, but DBP rise was ~10 mmHg greater in wild-type and Scurfy T cell-injected mice than in vehicles ($P<0.01$). Wild-type Treg injection alone tended to reduce SBP rise but did not affect DBP compared to control. Adoptive transfer of wild-type T cells restored angiotensin II induced-endothelial dysfunction ($P<0.05$), which was exaggerated in Scurfy T cell-injected mice ($P<0.01$) but not in mice receiving Scurfy T cells + wild-type Treg ($P<0.05$). Angiotensin II increased ROS production in MA wall and perivascular fat in Scurfy T cell-injected mice ($P<0.01$), but not when co-transferred with wild-type Tregs. Angiotensin II induced vascular stiffness ($P<0.01$) and hypertrophic remodeling ($P<0.05$) in vehicle and Scurfy T cell-injected mice, but not in other groups. Angiotensin II increased MA fibronectin expression ($P<0.05$) only in vehicle and Scurfy T cell-injected mice. **Conclusion:** These results demonstrate that Foxp3+ T regulatory lymphocytes have a protective role against angiotensin II-induced vascular damage.

## CLINICAL/OUTCOMES/POPULATION ORAL SESSION #1: 08:30 – 08:45: CHAUDIÈRE B

### Relationship between Extracellular Fluid Movement during Sleep and Urine Sodium Excretion in Normotensive and Hypertensive Subjects


*Laura White, T. Douglas Bradley, Sleep Research Laboratory, Toronto Rehabilitation Institute; Centre for Sleep Medicine and Circadian Biology of the University of Toronto and Department of Medicine of the University Health Network Toronto General Hospital, Alexander G. Logan, Lunenfeld-Tanenbaum Research Institute, Mount Sinai Hospital and Faculty of Medicine, University of Toronto.*



**Background:** Extracellular fluid volume (ECV) relates closely to sodium balance. During the day gravitational forces increase leg ECV that is reabsorbed overnight and excreted by the kidneys. In hypertensive patients, defective renal sodium handling may alter this relationship. We hypothesized that unlike normotensives, overnight reduction in leg ECV would not be associated with urine sodium excretion (UNaV) in hypertensives, which would result in increased intravascular volume and blood pressure (BP). **Methods:** Normotensive (BP140/90 or on anti-hypertensive medications, excluding diuretics) subjects underwent bioelectrical impedance measurement of leg fluid volumes (LFV) at night and the following morning, same-day 24 hour urine collection divided into day and night-time samples and automated BP measurement. **Results:** Hypertensives (n=25) had higher 24-hour (217.0±124.6 vs 148.6±72.2 mmol, p=0.008) and night-time UNaV (9.13±5.68 vs 5.37±2.69 mmol/hour, p=0.008) but lower daytime UNaV (4.80±2.64 vs 6.18±2.81 mmol/hour, p=0.047), than normotensives (n=26). Hypertensives had lower evening leg intracellular fluid volume (ICV, 4.71±1.74 vs 5.49±1.30 L, p=0.014) and higher ECV/ICV ratio (0.86±0.21 vs 0.69±0.10, p=0.001) than normotensives. Overnight leg ECV change correlated inversely with night UNaV in normotensives ($r=-0.741$, $p<0.001$) but not hypertensives ($r=0.082$, $p=0.696$). On stepwise linear regression, including age, sex, BMI, systolic (S)BP and overnight leg ECV change, overnight leg ECV change was the only independent predictor of 24-hour UNaV ($r^2=0.374$, $p=0.001$), day UNaV ($r^2=0.206$, $p=0.023$) and night UNaV ($r^2=0.529$, $p<0.001$) in normotensives. In hypertensives, SBP was the only independent predictor of night UNaV ($r^2=0.497$, $p<0.001$), with no independent relationships between 24-hour or day UNaV and any of the variables. **Conclusion:** In normotensives, the correlation between UNaV and overnight change in leg ECV suggests a normal renal response to increased ECV. However, in hypertensive patients, the kidneys may be unable to regulate ECV via sodium excretion, resulting in elevated BP in order to excrete excess sodium overnight.

## CLINICAL/OUTCOMES/POPULATION ORAL SESSION #1: 08:45 – 09:00: CHAUDIÈRE B

### The Control of Hypertension in Pregnancy Study (CHIPS) randomized controlled trial

*Laura A Magee MD FRCPC MSc (1, 2, 3), Peter von Dadelszen MBChB, DPhil FRCSC (2, 3), Evelyne Rey MD MSc FRCPC (13), Susan Ross MBA PhD (10), Elizabeth Asztalos MSc MD FRCPC (6, 7, 14), Kellie E Murphy MSc MD FRCSC (7, 14), Jennifer Menzies MSc (2), Johanna Sanchez MIPH (14), Joel Singer PhD (3), Amiram Gafni BSc MSc DSc (4), Andrée Gruslin MD FRCSC (12), Michael Helewa MD FRCSC (9), Eileen Hutton MSc PhD (11), Shoo K Lee MD PhD FRCPC (6), Alexander G Logan MD FRCPC (8), J Wessel Ganzevoort MD PhD (15), Ross Welch MBBS DA MD (17), Jim G Thornton MBChB MD (16), and Jean-Marie Moutquin MSc MD FRCSC (5) for the CHIPS Study Group (1, 2). Affiliations: (1) Medicine, University of British Columbia, Canada, (2) Obstetrics and Gynaecology, University of British Columbia, Canada, (3) School of Population and Public Health, University of British Columbia, Canada, (4) Clinical Epidemiology and Biostatistics, McMaster University, Canada, (5) Obstetrics and Gynaecology, Université de Sherbrooke, Canada, (6) Pediatrics, University of Toronto, Canada, (7) Obstetrics and Gynaecology, University of Toronto, Canada, (8) Medicine, University of Toronto, Canada, (9) Obstetrics and Gynaecology, University of Manitoba, Canada, (10) Obstetrics and Gynaecology, University of Alberta, Canada, (11) Obstetrics and Gynaecology, McMaster University, Canada, (12) Obstetrics and Gynaecology, University of Ottawa, Canada, (13) Medicine and Obstetrics and Gynaecology, University of Montreal, Canada, (14) The Centre for Mother, Infant and Child Research, Sunnybrook Research Institute, University of Toronto, Canada, (15) Obstetrics and Gynaecology, University of Amsterdam, Netherlands, (16) Obstetrics and Gynaecology, University of Nottingham, United Kingdom, (17) Obstetrics and Gynaecology, Derriford Hospital, United Kingdom*

**Background:** Normalizing blood pressure in pregnancy may reduce maternal complications but increase adverse perinatal effects. CHIPS aimed to determine whether a higher (vs. lower) blood pressure target improves perinatal outcome, without compromising maternal safety in non-severe pregnancy hypertension. **Methods:** In an open pragmatic international multicentre trial, women at 14+0-33+6 weeks gestation with non-proteinuric pre-existing or gestational hypertension, office diastolic BP (dBP) 90-105 mm Hg (or 85-105 mm Hg if on antihypertensives) and a live fetus were randomized to 'less tight' (target dBP 100 mm Hg) or 'tight' control (target dBP 85 mm Hg). The composite primary outcome was pregnancy loss or high level neonatal care for >48h in the first 28d of life, and the secondary, serious maternal complications before 6 weeks postpartum. Outcomes were compared between groups using logistic regression adjusted for key prognostic factors. **Results:** Of 1030 women randomized, 987 (94 sites) were included in the analysis. 74.6% had pre-existing hypertension. Women in 'less tight' (n=497) compared with 'tight' (n=490) control had similar rates of adverse perinatal (31.4% vs. 30.7%; aOR 1.02 [0.77, 1.35]) and maternal outcomes (3.7% vs. 2.0%; aOR 1.74 [0.79, 3.84]), despite higher mean dBP by 4.6 mm Hg (95% confidence interval 3.7, 5.4). Severe hypertension (≥160/110 mm Hg) developed more frequently in 'less tight' (vs. 'tight') control (40.6% vs. 27.5%; aOR 1.80 [1.36, 2.38]). **Conclusions:** 'Less tight' (vs. 'tight') control did not improve perinatal outcome, but did result in more severe hypertension. Our results do not support 'less tight' control of non-severe pregnancy hypertension.

# ABSTRACTS
## Friday, October 17

## CLINICAL/OUTCOMES/POPULATION ORAL SESSION #1: 09:00 – 09:15: CHAUDIÈRE B

### Comparison of auscultatory and oscilometric normative values for blood pressure evaluation in children

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**Background:** The automated oscilometric blood pressure (BP) devices are widely used in children, but many physicians apply Fourth Task Force (4TF) normative values (Pediatrics 1994), which were obtained from auscultatory devices. This may yield incorrect BP percentiles/Z scores and consequently incorrectly identify hypertensive children. Recently, oscilometric pediatric BP normative values have become available (Neuhauser et al, 2011). **Aim:** To compare auscultatory and oscilometric normative values for office BP evaluation in children. **Methods:** We conducted a retrospective analysis of office and 24h BP measurements (ABPM) of 229 children (116 boys), median age 15.31 years (IQ range 12.94, 16.76). The office systolic BP (SBP) and diastolic BP (DBP) absolute values were converted into Z scores using auscultatory (4TF) and oscilometric (Neuhauser) normative data. Results were compared using correlation analysis and the Bland Altman test. The accuracy of both normative values for prediction of hypertensive values on ABPM was analyzed using the receiver operator curve (ROC). **Results:** The comparison of the two normative methods for BP evaluation showed their good correlation (r= 0.9773 for SBP, r=0.9627 for DBP). Bland Altman test revealed only minimal difference in Z-scores between 4TF and Neuhauser for SBP (bias=-0.06±0.38, 95% LOA=-0.82 to +0.70), but a significant proportional error for DBP (Neuhauser underestimated of DBP for DBP Z scores lower than 1.65 and overestimated DBP at DBP Z scores higher than 1.65; bias=0.18±0.60, 95%LOA=-1.0 to +1.36). However, 4TF and Neuhauser Z scores yielded similar ROC AUC i.e. did not differ in prediction of ABPM systolic or diastolic hypertensive BP values defined as ABPM Z scores ≥1.65. **Conclusion:** Auscultatory and oscilometric normative data are interchangeable for SBP evaluation, but significant differences (under- and overestimation) were noted for DBP. Both methods were similar in the prediction of hypertensive BP values on ABPM.

## CLINICAL/OUTCOMES/POPULATION ORAL SESSION #1: 09:15 – 09:30: CHAUDIÈRE B

### A randomized trial of the effect of pharmacist prescribing on improving blood pressure in the community: The Alberta clinical trial in optimizing hypertension (RxACTION)

*Ross T Tsuyuki (1), Sherilyn KD Houle (1,2)Theresa L Charrois (3), Michael R Kolber (1), Finlay A McAlister (1), Meagen M Rosenthal (1), Richard Lewanczuk (1), Dale Cooney (4). Affiliations: (1) University of Alberta, (2) University of Waterloo, (3) Curtin University, (4) Alberta College of Pharmacists*

**Background:** Hypertension control rates in Canada remain suboptimal. In Alberta, pharmacists may apply for Additional Prescribing Authorization, allowing them to prescribe drug therapy for patients. This provides a new option for improving access to hypertension care. The objective of this study was to determine the impact of pharmacist care (including prescribing) on systolic blood pressure (SBP) in patients with uncontrolled hypertension. **Method:** Design: Randomized controlled trial with patients as the unit of randomization, with care provided by 22 Alberta pharmacists with prescribing authorization. Patients: Adults with BP above recommended targets (>140/90 or >130/80mmHg if diabetic) based upon measurements over multiple visits. **Enhanced Care:** Pharmacist assessment, BP wallet card, education on hypertension, pharmacist prescribing of antihypertensive drugs and laboratory monitoring using current Canadian guidelines, and monthly follow-up visits dropping down to every 3 months if target BP was achieved for 2 consecutive visits. All patients were followed for 6 months. **Control:** Wallet card for BP recording, patient information materials on hypertension, and no study specific follow-up. **Outcomes:** Primary outcome was the difference in change in systolic BP between the intervention and control groups at 6 months. **Results:** A total of 248 patients were randomized to the study. The mean (SD) patient age was 63.5 (12.7) years, 48.8% were male, and the mean (SD) systolic/diastolic BP was 149.7(13.6)/83.4(11.5) mm Hg at baseline. The intervention group had an adjusted mean (SE) reduction in systolic BP at 6 months of 18.0 (1.4) mm Hg compared with 11.0 (2.1) mm Hg in the control group (p=0.005). **Conclusion:** Pharmacist prescribing for patients with uncontrolled hypertension resulted in a statistically and clinically significant reduction in systolic blood pressure when compared to usual care.

## BIOMEDICAL ORAL SESSION #2: 10:00 – 10:15: CHAUDIÈRE A

### Endothelin-1 Overexpression in Endothelial Cells Increases Blood Pressure In an Endothelin Type A Receptor-Dependent Manner

*Suellen C. Coelho, Yohann Rautureau1, Asia Rehman1, Stefan Offermanns3,4, Pierre Paradis1, Ernesto L. Schiffrin1,2. 1 Hypertension and Vascular Research Unit, Lady Davis Institute for Medical Research and 2Department of Medicine, Sir Mortimer B. Davis-Jewish General Hospital, McGill University, Montréal, Canada. 3Department of Pharmacology, Max-Planck-Institute for Heart and Lung Research, Ludwigstr. 43, 61231 Bad Nauheim, Germany 4Medical Faculty, J.W. Goethe University Frankfurt, Theodor-Stern-Kai 7, 60590 Frankfurt, Germany*

**Background:** The mechanisms of blood pressure (BP) regulation by endothelin (ET)-1 produced by endothelial cells (EC) are complex and remain unclear. Transgenic mice with constitutive EC-specific human ET-1 (EDN1) overexpression presented vascular injury but no change in BP, which could be due to adaptation to life-long high ET-1 exposure. We have now generated an inducible EC-restricted EDN1 overexpressing mouse (ieET-1) in order to demonstrate the effects of ET-1 on BP regulation independent of developmental effects. **Method/Results:** Two transgenic mouse lines (C134 and C170) expressing chloramphenicol acetyltransferase and EDN1 before and after Cre-mediated excision, respectively, were crossed with mice expressing tamoxifen-inducible CreERT2 under the control of Tie2 promoter (ieCre) to generate ieET-1 mice. Mice were treated with tamoxifen (1 mg/kg/day, SC) or vehicle for 5 days and sacrificed 16 days later. Additional mice were treated with 5 or 10 mg/kg/day PO of ET type A receptor blocker, atrasentan, from day 9. BP was determined by telemetry, plasma ET-1 levels by ELISA and ET type A (Ednra) and B (Ednrb) receptors expression in the kidney by quantitative RT-qPCR. Tamoxifen induced a 10-fold increase of plasma ET-1 in ieET-1-C134 and 13-fold in ieET-1-C170 ($P<0.01$), compared to control ieET-1 and tamoxifen-treated ieCre. ET-1 overexpression increased night systolic BP by ~20 mmHg in ieET-1-C134 and ieET-1-C170 compared to tamoxifen-treated ieCre, which was reversed partially or completely by 5 or 10 mg/kg/day of atrasentan, respectively ($P<0.01$). Tamoxifen-treated ieET-1-C134 presented a 17-fold increase in Ednrb expression in renal cortex ($P<0.05$) and no change in renal medulla compared to control ieET-1, whereas renal Ednra expression was unchanged. **Conclusion:** This new inducible EC-restricted EDN1 overexpressing mouse exhibits ET-1-dependent elevated BP mediated by ET type A receptors. Increased ET type B receptor expression in renal cortex could play a role in ET-1-induced BP rise.

## BIOMEDICAL ORAL SESSION #2: 10:15 – 10:30: CHAUDIÈRE A

### von Willebrand Factor Synthesis and Circulating Half Life Are Progressively Elevated, but Relative Platelet Binding Decreases in Stage 3-5 Chronic Kidney Disease Patients


*Cynthia M. Pruss, PhD1, Spencer Barr1, Julie Grabell2, Shawn Tinlin2, Angie Tuttle2, Michael A. Adams, PhD1, Jocelyn S. Garland, MD2, Paula James, MD2 and Rachel M Holden, MD2. 1Department of Biomedical and Molecular Sciences, and 2Department of Medicine, Queen's University, Kingston, ON, Canada.*



**Background:** Introduction: von Willebrand Factor (VWF) is essential for primary hemostasis by binding platelets at the site of injury, is a biomarker for endothelial dysfunction, and increased VWF levels are linked to cardiovascular disease. VWF and osteoprotegerin (OPG), an inhibitor of vascular calcification, are co-secreted by the endothelium and remain associated in circulation. Chronic kidney disease (CKD) patients have high rates of cardiovascular disease, not explained by traditional Framingham risk factors. This study closely evaluates pre-dialysis CKD individuals longitudinally for VWF and OPG. **Methods:** VWF antigen (VWF:Ag), VWF propeptide (VWFpp), VWF multimer structure, VWF platelet binding (VWF:Rco) and OPG were measured at baseline and at 4 years in a cohort with stage 3-5 CKD (N=55, 61.5±12.9 years, 44% female) and controls (N=49, 67.4±7.9*years, 67% female). Percentage high molecular weight VWF multimers (HMWM) determined via densitometry of modified western blot. Values are mean±SD., one-way-ANOVA with Tukey's post test; *: P<0.05 . **: P<0.01, ***: P<0.001 vs. control. †††: P<0.001, CKD initial vs. 4 years. **Results:** VWF:Ag, VWFpp, and OPG significantly increased from CKD baseline to 4 years. VWF multimer structure showed a small increase in %HMWM. VWFpp/VWF:Ag and VWF:Rco/VWF:Ag ratios were significantly lower in CKD patients, indicating an increase in VWF circulating half life and a loss in relative VWF platelet binding activity. **Conclusions:** This study demonstrates that significant changes in VWF level and function occurs and progresses in stage 3-5 CKD. The impact these changes may have on cardiovascular health and hemostasis warrant further study. Supported by Queen's Department of Medicine, and Kidney Foundation of Canada.


| | Controls | CKD, Initial | CKD, 4 years |
|---|---|---|---|
| VWF:Ag (U/ml) | 1.15±0.42 | 1.42±0.62††† | 1.91±0.71*** |
| VWFpp (U/ml) | 1.28±0.36 | 1.29±0.43††† | 1.76±0.46*** |
| VWFpp/VWF:Ag | 1.16±0.23 | 0.98±0.27** | 0.99±0.30** |
| VWF:RCo (U/ml) | 1.41±0.47 | 1.40±0.67 | 1.41±0.70 |
| VWF:RCo /VWF:Ag | 1.23±0.3 | 1.04±0.4**††† | 0.73±0.2*** |
| VWF HMWM (%) | 19.9±4.9 | 19.7±5.3† | 21.2±5.2 |
| OPG (ng/ml) | 1.5±1.4 | 3.0±1.6*††† | 7.9±4.2*** |

## BIOMEDICAL ORAL SESSION #2: 10:30 – 10:45: CHAUDIÈRE A

### Nitric Oxide attenuates the enhanced expression of Giα proteins in vascular smooth muscle cells from Spontaneously Hypertensive Rats: Molecular mechanisms

*Oli Sarkar, Ravinder Pal, Madhu B. Anand-Srivastava, University of Montreal*

**Background:** We have previously shown that the nitric oxide donor, SNAP, decreased the expression of Giα proteins and associated functions in A10 vascular smooth muscle cells. The present study was undertaken to investigate if SNAP can also decrease the expression of Giα proteins and associated signaling in aortic vascular smooth muscle cells (VSMC) from 12-week-old Wistar-Kyoto (WKY) and Spontaneously Hypertensive rats (SHR) and to further explore the underlying mechanism(s). Treatment of VSMC with SNAP (100 μM) for 24h decreased the expression of Giα-2, -3 proteins. This decrease was subverted by inhibiting peroxynitrite with either Mn (III) tetrakis (4-benzoic acid) porphyrin (MnTBAP) or uric acid, and by inhibiting MEK with PD98059. However, inhibition of guanylate cyclase by 1H-(1, 2, 4) oxadiazolo (4, 3-a) quinoxalin-1-one (ODQ) was unable to restore the SNAP-induced decrease in Giα-2, -3. In addition, the enhanced activity of NADPH oxidase and protein levels of its subunits (Nox-4, p47phox and p22phox) were decreased by SNAP. The decreased expression of NADPH oxidase subunits was restored to near-control levels by MnTBAP. Furthermore, SNAP treatment decreased the enhanced levels of O2-, TBARS and protein carbonylation in SHR to control levels. SNAP also attenuated the increased phospholyration of PDGFR, EGFR and c-Src. These results suggest that SNAP decreased the enhanced expression of Giα proteins in VSMC from SHR by attenuating the increased oxidative stress, growth factor receptor activation and ensuing ERK1/2 signaling and not by a cGMP-dependent mechanism.

## BIOMEDICAL ORAL SESSION #2: 10:45 – 11:00: CHAUDIÈRE A

### Endoplasmic reticulum stress inhibition preserves endothelial-mediated vasodilation in SHR resistance blood vessels.

*Kaitlyn Werner, Victoria Yum, Chao Lu, Rachel E. Carlisle1, Yejin No and Jeffrey G. Dickhout, Nephrology, St Joseph's Healthcare Hamilton and McMaster University, Hamilton, Ontario, Canada*

**Background:** Endoplasmic reticulum (ER) stress has been observed in blood vessel dysfunction. Studies have shown that ER stress can be resolved using the chemical chaperone, 4-phenylbutyrate (4-PBA). Thus, we tested the ability of 4-PBA to correct blood vessel dysfunction in the spontaneously hypertensive rat (SHR). **Methods/Results:** 12-week-old male SHRs with established hypertension and normotensive WKY rats were treated with 4-PBA for 5 weeks (1g/kg/day, orally). Baseline SHR blood pressure (BP), measured using tail cuff plethysmography 1 week prior to 4-PBA treatment, was 181/118 mmHg. Animals were randomized into 4-PBA or vehicle treatment groups. Mesenteric resistance vessels were collected after 5 weeks for functional and structural analysis. Final BPs were measured directly through carotid artery cannulation.
We found that 4-PBA treatment significantly lowered BP in the SHR but not in the WKY (vehicle 206.1±4.4 vs. 4-PBA 179.0±3.1, systolic; vehicle 143.6±4.4 vs. 4-PBA 121.2±4.3 mmHg, diastolic). ER stress, characterized by GRP78 and CHOP expression, was significantly reduced in SHR resistance vessels by 4-PBA treatment. 4-PBA diminished contractility and augmented endothelial vasodilation in SHR resistance vessels, but not WKY. 4-PBA treatment had no effect on SHR or WKY sodium nitroprusside-mediated vasodilation, indicating reduced nitric oxide bioavailability. Normotensive resistance vessels, treated with the ER stress-inducing agent tunicamycin, showed decreased vasodilation as well as increased GRP78 and CHOP expression - characteristics similar to the SHR; this was resolved with 4-PBA treatment. DHE staining revealed that ER stress induction and the accumulation of misfolded proteins resulted in superoxide generation, a mechanism that reduces nitric oxide bioavailability. **Conclusion:** ER stress causes endothelial-mediated vascular dysfunction leading to hypertension. 4-PBA reduces blood pressure in the SHR by alleviating ER stress and improving resistance blood vessel endothelial-dependent vasodilation without changing smooth muscle mass or vessel structure.

# ABSTRACTS
## Friday, October 17

## CLINICAL/OUTCOMES/POPULATION ORAL SESSION #2: 10:30 – 10:45: CHAUDIÈRE B

### Subcutaneous resistance artery remodeling and function in chronic kidney disease patients

*Julio C. Fraulob-Aquino, Marie Briet, INSERM U1083, CNRS UMR 6214, Centre Hospitalo-Universitaire d'Angers, Université d\\\'Angers, Angers, France; Tlili Barhoumi, Vascular and Hypertension Research Unit, Lady Davis Institute for Medical Research, Jewish General Hospital, McGill University, Montréal, Québec, Canada; Carmine Savoia, Cardiology Unit, Second Faculty of Medicine, Sant'Andrea Hospital, Sapienza University of Rome, and ‡Research Center, Fatebenefratelli San Pietro Hospital, Rome, Italy; Pierre Paradis,Vascular and Hypertension Research Unit, Lady Davis Institute for Medical Research, Jewish General Hospital, McGill University; Ernesto L. Schiffrin, Vascular and Hypertension Research Unit, Lady Davis Institute for Medical Research and Department of Medicine, Jewish General Hospital, McGill University, Montréal, Québec, Canada*

**Background:** Chronic kidney disease (CKD) is associated with cardiovascular (CV) complications. However, interventional trials targeting classical CV risks factors have been often unsuccessful in advanced stage CKD, which emphasizes the need to better understand CKD-associated vascular disorders. Resistance arteries are a key determinant of blood pressure (BP) and their changes in different CV conditions contribute to target organ damage. The aim of the present study was to characterize resistance artery remodelling and function in CKD patients, compared to vessels from hypertensive (HTN) subjects. **Method/Results:** Twenty-two stage 4 CKD patients (aged 63.6±3.1 years) and 16 HTN subjects (45.6±16.1 years) were included in the present study. They all underwent a subcutaneous biopsy under local anaesthesia. Small artery remodelling and function were studied on a pressurized myograph, and subcutaneous fat CD3 infiltration and media fibronectin expression by immunostaining. Vascular smooth muscle cells (VSMCs) were counted after hematoxilin-eosin staining. CKD systolic BP was similar to HTN (133±18vs.143±10 mmHg, respectively). Vasodilatory responses to acetylcholine were lower in CKD compared to HTN (maximal relaxation (%), 74.3±3.4vs.87.5±2.7, P<0.05). Media/lumen at 60 mmHg was lower in CKD than in HTN (6.7±0.5 vs 8.8±0.7, P<0.05). Resistance artery stiffness was lower in CKD compared to HTN (strain at 120 mmHg, 0.845±0.126vs0.585±0.099, P<0.05). Fibronectin staining in resistance arteries was lower in CKD than HTN (8.2±0.8 vs 23.3±1.7 RFU/µm2, P<0.001). Less VSMCs were present in the arterial wall of CKD compared to HTN (5.4±0.4vs7.2±0.5 cells/µm2, P<0.05). Subcutaneous fat presented fewer CD3+ cells in CKD than HTN (12.8±4.1 vs 23.7±12.8 cells/mm2, P<0.05). **Conclusion:** Despite higher levels of BP, resistance arteries isolated from CKD patients exhibited no vascular remodelling and lower arterial stiffness compared with HTN patients. These results are in line with the maladaptive hypotrophic remodelling observed in large vessels in CKD, suggesting a generalized vascular defect in mechanotransduction in CKD.

## CLINICAL/OUTCOMES/POPULATION ORAL SESSION #2: 10:45 – 11:00: CHAUDIÈRE B

### Undiagnosed Hypertension in the Emergency Department: The Significance of Elevated Blood Pressure Measurements at Emergency Department Triage

*Brian Levy, Ian Preyra, MD FRCP(C), Mathew Mercuri, Ph.D, Wendy Suapapol Ph.D. PGY-5 Emergency Medicine Resident at McMaster University/ Hamilton Health Sciences*

**Background:** Relatively little is known about the implications of incidentally encountered elevated blood pressure (BP) in the emergency department (ED). We sought to better characterize the relationship between emergency department triage blood pressure and a patient's likely ambulatory baseline BP. We also examined common perceptions concerning pain and blood pressure. **Methods:** We performed a prospective observational study of 425 patients in a large suburban Canadian emergency department. We surveyed awareness of subjects' current health status prior to taking vital signs at triage. Subjects identified with elevated triage BP were retested later in their stay utilizing a serial blood pressure measurement device. Our analysis allowed us to characterize the potential significance of triage BP relative to ambulatory BP in the community. **Results:** The sample prevalence of previously diagnosed hypertension among patients presenting in the emergency department was 34.8%, plus incremental prevalence of 16.9% for previously undiagnosed hypertension, even after allowing ~ 85 minutes for normalization prior to serial blood pressure device confirmation. Over 33% of those newly diagnosed suffered from stage 2 hypertension. Only 42.5% of previously diagnosed hypertensive patients encountered were controlled to guidelines. Only a small sub segment of typically normotensive people (~ 16%) demonstrated precipitous BP declines on repeated measurement (-21mmHg/-10mmHg), while most patients with elevated triage BP evidence only a slight drop (-6.6 mmHg/-1.2mmHg) on serial measurement. Of patients presenting with elevated triage BP, over 76% continued to be hypertensive on serial measurement. We found no association between pain and elevated blood pressure. **Conclusion:** The incidence of undiagnosed and/or uncontrolled hypertension in this population is significant. The notion that elevated ED triage BP is attributable to pain, stress, or 'white coat syndrome' is not supported by our results. It is apparent that incidentally elevated ED triage BP usually signals underlying disease and warrants referral for further evaluation.

## CLINICAL/OUTCOMES/POPULATION ORAL SESSION #2: 11:00 – 11:15: CHAUDIÈRE B

### Marginal structural models for estimating the causal relationships between physical activity, blood pressure, and mortality in a longitudinal cohort study: the Honolulu Heart Program

*Amanda M. Rossi, Denis Talbot, Université du Québec à Montréal; Geneviève Lefebvre, Université du Québec à Montréal; Juli Atherton, Université du Québec à Montréal; Simon L. Bacon, Concordia University*

**Background:** The purpose of this study was to evaluate the separate causal relationships of physical activity and blood pressure (BP) on mortality and major adverse cardiovascular events (MACE) and the causal relationship of physical activity on BP. **Methods and Results:** This study comprised secondary analyses of a longitudinal, observational study, the Honolulu Heart Program (n=8,006 men). Physical activity (measured by self-report questionnaire) and BP were both assessed at three time points; Exam 1 (1965-68), Exam 2 (1968-71), and Exam 4 (1991-1993). Marginal structural Cox models and Marginal structural models for repeated measures were used to estimate: 1) the separate causal relationships of physical activity and BP on mortality and MACE; and 2) the causal relationship of physical activity on BP. Being physically active reduced the rate of mortality (Hazard Ratio (HR) = 0.68, 95% confidence interval (CI) = 0.60 to 0.76) and MACE (HR= 0.84, 95%CI: 0.75 to 0.93) by 32% and 16%, respectively. Blood pressure was shown to have a dose-dependent, causal relationship with both mortality and MACE whereby increasing BP was related to more events. Active participants showed a significant decrease of 2.47 mmHg (95%CI, -3.46 to -1.48) in systolic BP compared to the inactive group. No change in diastolic BP was observed. **Conclusions:** This is the first study to simultaneously examine the time-varying effect of physical activity on blood pressure, mortality, and MACE, demonstrating a causal relationship with all variables. The results suggest that BP may be a mediator of the relationship between physical activity and mortality.

## CLINICAL/OUTCOMES/POPULATION ORAL SESSION #2: 11:15 – 11:30: CHAUDIÈRE B

### The relationship between primary care physician utilization and hospitalizations or ED visits: is uncomplicated hypertension really an ambulatory care sensitive condition?

*Robin Walker, G Chen, F McAlister, N Campbell, B Hemmelgarn, E Dixon, W Ghali, D Rabi, K Tu, N Jette, H Quan for the Hypertension Outcome and Surveillance Team, University of Calgary, University of Alberta, University of Toronto*

**Purpose:** Hospitalizations for ambulatory care sensitive conditions (ACSC) represent an indirect measure of access and quality of community care. The purpose of this study was to examine primary care physician (PCP) utilization among patients hospitalized for one ACSC, uncomplicated hypertension. **Methods:** A cohort of patients with hypertension was identified using administrative databases in Alberta, Canada. We applied the Canadian Institute for Health Information's case definition to detect all patients who had an ACSC hospitalization and/or emergency department (ED) visit for uncomplicated hypertension. We assessed hypertension-related and all-cause PCP visits. **Results:** The overall adjusted rate of ACSC hospitalizations and ED visits for uncomplicated hypertension was 7.1 and 13.9 per 10,000 hypertensive patients, respectively. The likelihood of ACSC hospitalization for uncomplicated hypertension was associated with age, household income quintile, region of residence, and Charlson comorbidity status (all $p< 0.0001$). The adjusted rate of ACSC hospitalizations for uncomplicated hypertension increased from 4.8 per 10,000 hypertensive patients for those without hypertension-related PCP visits prior to diagnosis to 10.5 per 10,000 hypertensive patients for those with 5 or more hypertension-related PCP visits. The rate of ACSC hospitalizations and/or ED visits for uncomplicated hypertension increased as the number of hypertension-related PCP visits increased even after stratifying by demographic and clinical characteristics. **Conclusion:** As the frequency of hypertension-related PCP visits increased, the rate of ACSC hospitalizations and/or ED visits for uncomplicated hypertension increased. This suggests that ACSC hospitalization for uncomplicated hypertension may not be a particularly good indicator for assessing access to primary care.

# ABSTRACTS
## Friday, October 17

## BIOMEDICAL POSTER SESSION #2: 15:30 – 17:00: HALL & FOYER

### P15 - Angiotensin-II (Ang-II)-induced expression of the early growth response protein 1 (Egr-1) is mediated by a Calcium-CaMKII-II –dependent pathway in vascular smooth muscle cells (VSMC)

*Estelle Rolande Simo Cheyou, Ashok Srivastava University of Montreal, Department of Medicine, CRCHUM*

**Background:** Egr-1 is a transcription factor implicated in the regulation of several growth-related cascades. It has been suggested to participate in the development of hypertension and vascular disease processes such as intimal thickening following vascular injury, atherosclerosis and cardiac hypertrophy. Up regulation of Egr-1 was recently shown to be a key contributing factor in stimuli-induced vascular smooth muscle cells (VSMC) proliferation and hypertrophy. Ang-II, a potent vasoactive peptide with pathogenic role in vascular disease, has been demonstrated to contribute to aberrant remodeling of the vessel wall. This is mainly due to the ability of Ang-II to activate hypertrophic and proliferative pathways such as the mitogen-activated protein kinase and the phosphatidylinositol-3-kinase pathways in VSMC. It has been shown earlier that Ang-II requires H2O2 generation to activate these signaling pathways and we have shown that Ca2+, calmodulin (CaM) and Ca2+/CaM-dependent protein kinase II (CaMKII), play a critical role to trigger H2O2-induced effects in VSMC. We have also shown that endothelin-1, another mitogenic vasoactive peptide, requires CaMKII to up regulate Egr-1 expression and to mediate its physiological responses. However, Ang-II-induced modulation of Egr-1 expression in VSMC and the implication of CaMKII in this process remain unexplored. Therefore these studies were undertaken to characterize the effect of Ang-II on Egr-1 expression in A10 VSMC and to examine the role of Ca2+, CaM and CaMKII on this response. Ang-II, at 100nM, induced an increase in Egr-1 expression in a time-dependent fashion in the nucleus of A10 VSMC. Pharmacological blockade of CaM and CaMKII by calmidazolium and KN-93 respectively, significantly attenuated Ang-II-induced Egr-1 expression. These results demonstrate that Egr-1 expression is up regulated in response to Ang-II via CaM/CaMKII- dependent pathways in VSMC. (Supported by CIHR grant)

## BIOMEDICAL POSTER SESSION #2: 15:30 – 17:00: HALL & FOYER

### P16 - Involvement of MAPKs and PKB pathways in Insulin-like growth factor 1 (IGF-1)-induced Early Growth Response protein-1 (Egr-1) expression in A10 Vascular smooth muscle cells (VSMC)

*Viktoria Youreva and Ashok K. Srivastava, Department of Medicine, University of Montreal and University of Montreal Hospital Center (CRCHUM), La Tour Viger, Montreal, Quebec, H2X 0A9*

**Background:** Egr-1 is a transcription factor that plays an important role in vascular biology. Egr-1 expression has been shown to be up regulated in cardiovascular disease (CVD). IGF-1, a potent mitogen and vasoactive factor, is believed to contribute to the development of CVD, such as atherosclerosis, through the hyper activation of mitogenic and growth promoting pathways, including mitogen-activated protein kinase (MAPK) and protein kinase B (PKB) pathways, as well as regulation of multiple transcription factors. Reactive oxygen species (ROS) have been shown to mediate the effects of IGF-1 and has been suggested to contribute to the pathogenesis of vascular abnormalities. We have previously shown that IGF-1 can induce the expression of Egr-1 in VSMC; however, the signaling pathways involved in this process remain unexplored. Therefore, we have investigated the involvement of MAPK, PKB and ROS in IGF-1-induced Egr-1 expression. Treatment of VSMC with IGF-1 enhanced Egr-1 protein levels in a time and dose-dependent fashion. Pharmacological blockade of RNA synthesis and protein synthesis by Actinomycine D and Cycloheximide, respectively, inhibited IGF-1-induced Egr-1 expression. AG1024, a selective pharmacological inhibitor of IGF-1R, also attenuated IGF-1-induced Egr-1 expression. Moreover, PD98059 and SP600125, two selective inhibitors of MEK/ERK1/2 and JNK family of MAPKs, respectively, significantly decreased IGF-1-stimulated increase in Egr-1 expression in VSMC. In addition, pharmacological blockade of PI3K/PKB pathways by Wortmannin/SC-66 respectively, also suppressed IGF-1-induced Egr-1 expression. Moreover, DPI (Diphenyleneiodonium), an NAD (P) H oxidase inhibitor, also blocked the Egr-1 expression in response to IGF-1. In summary, these data demonstrate that ERK1/2/JNK, PI3K/PKB and ROS-dependant signaling events play a critical role in IGF-1 induced expression of Egr-1 in VSMC.

## BIOMEDICAL POSTER SESSION #2: 15:30 – 17:00: HALL & FOYER

### P17 - Retrospective analysis of international normalized ratio variability in healthy patients taking the Vitamin K Antagonist Warfarin

*David Casey, MD, Department of Medicine, Western University,dcasey4@uwo.ca; Ute Schwartz, MD PhD, Department of Medicine, Western University,Ute.Schwarz@lhsc.on.ca; George Dresser, MD PhD, Department of Medicine, Western UniversityGeorge.Dresser@lhsc.on.ca*

**Introduction:** Response to the commonly used anticoagulant warfarin is affected by well-characterized genetic variations in hepatic metabolic enzymes cytochrome P450 (CYP) 2C9 and 4F2, and the pharmacological target of warfarin, vitamin K epoxide reductase complex 1 (VKORC1). A recent meta-analysis showing variants of VKORC1 and CYP 2C9 require lower warfarin dose and variant 4F2 alleles confer significantly higher dose requirements (1) contrasts a recent study showing decreased bleeding events with variant CYP4F2 alleles but no change with variant CYP2C9 or VKORC1 alleles (2). Further, the association of allele variants with adequacy of anticoagulation has never been studied in genomics-guided warfarin therapy. We determined local trends in time in therapeutic INR range (TTR) in patients receiving genomics-guided therapy at LHSC. **Methods:** Using the PG-WF database, this retrospective chart review was performed in stable out-patients with previously determined CYP2C9, CYP4F2, and VKORC1 genotype. TTR was assessed using the Rosendaal linear interpolation method. **Results:** We identified 56 patients with complete genomic data and available TTR data. We found no association of variant CYP2C9, VKORC1, or CYP4F2 alleles and impairment in TTR. Further analyses of variant alleles and other factors affecting TTR, such as therapeutic turbulence, will be presented. **Discussion:** Although our study is under powered, the lack of association between variant alleles and impairment in TTR suggests that genomics-guided warfarin therapy may negate the detrimental impact of variant alleles. Our preliminary data actually suggests an inverse trend supporting the hypothesis that genomics-guided warfarin therapy favours variant alleles.

## BIOMEDICAL POSTER SESSION #2: 15:30 – 17:00: HALL & FOYER

### P18 - De novo production of reactive oxygen species by endothelial microparticles

*Maddison Turner, Shareef Akbari, Dylan Burger, Ottawa Hospital Research Institute*

**Background:** Microparticles (MPs) are small (0.1-1.0), membranous vesicles shed from the cell surface following stress/injury. Our laboratory and others have shown that endothelial MPs are also potent autocrine/paracrine signals for a variety of cellular responses including inflammation, apoptosis, and senescence. Endothelial MPs have also been shown to induce reactive oxygen species (ROS) production in target cells however there is a paucity of information on whether endothelial MPs themselves may produce ROS. **Methods:** Endothelial MPs were isolated from media of cultured human umbilical vein endothelial cells (HUVECs) by differential centrifugation. The high speed supernatant obtained during microparticle isolation served as a control. Reactive oxygen species production was assessed by lucigenin chemiluminescence and HPLC. **Results:** Endothelial MPs displayed ROS significantly higher ROS production compared with high speed supernatant as determined by lucigenin (1519±477 vs 3.71±5.57 AU/mg protein, $P<0.05$, $n=3$) and HPLC (2-HE/DHE ratio 58±13 vs 4±2, $n=4$, $p<0.05$). **Conclusion:** Our results suggest that endothelial MPs themselves produce ROS. Such processes may contribute to the paracrine effects of endothelial MPs.

## BIOMEDICAL POSTER SESSION #2: 15:30 – 17:00: HALL & FOYER

### P19 - Function and regulation of HCaRG in kidney damage

*Carole G. Campion, department of Medicine, University of Montreal, Quebec, Canada, Hiroyuki Matsuda, Department of Internal Medicine, Nihon University School of Medicine, Tokyo, Japan, Johanne Tremblay, Department of Medicine, University of Montreal, Quebec, Canada.*

**Background:** The kidneys play a central role in the regulation of arterial pressure and the development of hypertensive disorders. Hypertension-related, Calcium-Regulated Gene (HCaRG), also named COMMD5, was identified and cloned by our group, from the spontaneously hypertensive rat (SHR) parathyroid gland. HCaRG mRNA levels are more abundant in kidneys of SHR than of the normotensive control rat. Transgenic mice overexpressing human HCaRG recovered renal function faster than controls after ischemia/reperfusion. HCaRG accelerates tubular repair after ischemic kidney injury by facilitating redifferentiation of tubular epithelial cells. **Methods/Results:** Overexpression of HCaRG in mouse renal adenocarcinoma cells led to the inhibition of tumor progression. In this model, HCaRG acted as a natural inhibitor of the ErbB signaling pathway by the inhibition of EGFR/ErbB receptors phosphorylation. EGFR activation is as an important mediator of renal repair following injury. On the other hand, constitutive/chronic ErbB activation is thought to be important in the evolution of renal diseases and cancer. In this project, we focus our attention on the role of HCaRG in the down-modulation of ErbB receptors. Recent studies suggest that regulation of the ubiquitin pathway may be the basis of many of the functions of the COMMD proteins. We hypothesize that HCaRG is implicated in the ubiquitination and proteasomal degradation of the ErbB receptors. We will study impact of HCaRG expression on ErbB receptors activation using normal kidney cell's model, with different treatments leading to cell injuries (Cisplatin, Angiotensin II). We will use inhibitors of the ErbB signaling pathway. Co-localization of HCaRG and ErbB receptors in endosomal compartments will be determined by confocal microscopy analysis. **Conclusion:** The mechanisms that down regulate the activity of ErbB receptors received a lot of attention, particularly when their activities need to be terminated to prevent chronic inflammation and tissue damage. Consequently, HCaRG could be a target for improving repair after renal injury.

## BIOMEDICAL POSTER SESSION #2: 15:30 – 17:00: HALL & FOYER

### P20 - Impact of the (pro) renin receptor on adipose tissue structure and function

*Zulaykho Shamansurova, Basma A.-M. Ahmed (3), Sonia Kajla (3), Catherine Michel (3), Ondrej Seda (3), Julie L. Lavoie (1,2,3); 1- Université de Montréal, 2-Montreal Diabetes Research Centre, 3-Centre de Recherche CHUM*

**Introduction:** Mice lacking (pro) renin/renin receptor [(P) RR] in adipose tissue present with smaller body weight (BW) and fat masses. However, the impact of (P) RR deficiency on fat tissue structure and function has not been studied. **Material/Methods:** Specific adipose tissue (P) RR gene knock-out mice - AP2 (P) RR-KO [(KO)] were created by cre/loxp technology, and produced male homozygous and female heterozygous KO. Mice were kept for 10 weeks on regular chow (ND) and female mice were additionally exposed to a high-fat/high-carbohydrate diet (HF/HC). At the end of protocol, animals were sacrificed and blood, the subcutaneous (SCF) and perirenal (PRF) fat were collected. Tissues were fixed and paraffin-embedded. Using hematoxylin-eosin colored slices the cell numbers, and mean cell surface area were calculated in pictures taken by optic microscope Olympus600 with "Q-capture" software program, analyzed with the Image-J application. Circulating adiponectin and insulin were measured by immunoassay AlphaLisa kits. **Results:** Circulating adiponectin levels were significantly increased in all KO mice and were associated with decreased circulating insulin levels. These results were more pronounced in male KO mice and were accentuated with HF/HC diet in females. Moreover, clear differences could be observed when comparing KO and wild-type mice looking at both SCF and PRF by histology. We found that KO mice on ND had higher cell numbers and lower mean cell surface area. Similarly to circulating adiponectin levels, the histology results were more pronounced in male KO mice. **Conclusion:** Our results suggest that deletion of the (P) RR in adipose tissue produces a healthier adipose tissue structure and function with increased circulating adiponectin levels. As such, (P) RR gene suppression may improve insulin sensitivity and cardiometabolic health.

## CLINICAL/OUTCOMES/POPULATION POSTER SESSION #1: 17:30 – 18:30: HALL & FOYER

### P21 – Salt reduction as a healthy recommendation in lowering blood pressure

*Samia L.L. Rizk*
*Ain Shams University, Cairo, Egypt*
*Potsdam University, Potsdam, NY, USA*

**Introduction:** Reduction in salt intake lowers blood pressure in both hypertensive and normotensive subjects. Low salt intake affects release of other hormones as plasma renin aldosterone, noradrenaline, and adrenaline. Salt reduction also affects lipids of the body as total cholesterol, low density lipoprotein and high density lipoprotein as well as triglycerides. The goal is the less salt intake protects against cardiovascular diseases.
**Method:** 3 groups were chosen, and were asked to decrease salt intake for at least 4 weeks or more. Each group included 100 persons.
The first group was hypertensive.
The second group chosen randomly, normotensive and were asked to take the usual salt intake.
The third group, normotensive and were asked to reduce the salt intake from 9-12g/day to 5-7 g/day each.
All subjects, aged 18 and above, no pregnant women, no other diseases other than hypertension e.g. no diabetes or heart failure with non-pharmacological intervention,.
After 4 weeks, blood pressure was measured together with 24 hours urinary sodium.
**Results:**

- The mean age of subjects was 50(22-73).
- The median blood pressure was 141/86 mmHg.
- The decrease in urinary sodium was 75 mmol, range 40-118 mmol, equivalent to a reduction of salt intake of 4.4g/day(range 2.3-6.9g/day)
- The study varied between 4 weeks and 6 months.
- 24 hour urinary sodium was measured and the median was 160 mmol equivalent to a salt intake of 9.4g/day.
- The first group had a drop of urinary sodium from 190to 108mmol/24 hour.
- The second group had a drop of urinary sodium from 145 to 65mmol/24 hour.
- The third group had a drop of urinary sodium between 139 to 64mmol/ 24 hour.
- The results showed that the systolic blood pressure was reduced by - 4.18 mmHg, P

## CLINICAL/OUTCOMES/POPULATION POSTER SESSION #1: 17:30 – 18:30: HALL & FOYER

### P22 – Effectiveness of patient programs designed to increase physical activity or decrease salt consumption on blood pressure control

*Sajal Jain, DR Birbrager, Oshawa Medical Centre Ontario, Canada, HA Boyrazian, Scarborough Hospital, Ontario, Canada, JS Sampalis, Faculty of Medicine, McGill University, JSS Medical Research Inc., Montreal, Quebec, Canada for PROTECT II/SHAKE THE HABIT (STH) Investigators.*

**Objective:** To assess the effect on blood pressure (BP) of: (i) an educational program aiming to increase physical activity, and; (ii) a salt intake support program; in patients with mild-to-moderate hypertension managed in a real-life setting by Canadian general practitioners.
**Design/Method:** PROTECT II and STH were open-label, randomized, multi-center studies. All patients were treated with perindopril for 16 weeks. In PROTECT II patients were randomized to: Routine Care (RC), reminders for physical activity (PA); medication adherence (AD) and AD+PA. Patients in STH were randomized into RC or a salt intervention (SI) receiving information on salt sources, reading food labels, reduce salt intake and were encouraged to follow the Canadian guidelines for upper intake limit of 1500 mg/day. **Results:** 12,187 and 12,697 patients were recruited in PROTECT II and STH, respectively, with mean ± SD baseline age of 61±13 years and 60±13 years. The majority of patients were male (54.3% and 55.3%, respectively) and Caucasian (76.8% / 78.2%). In PROTECT II the mean change in SBP/DBP was (-21.1/-11.1 mmHg; Pion, in addition to treatment with perindopril, are more beneficial in achieving BP control.

# ABSTRACTS
## Friday, October 17

## CLINICAL/OUTCOMES/POPULATION POSTER SESSION #1: 17:30 – 18:30: HALL & FOYER

### P23 – The Successful Experience thought Rondon Project of Brazil: An Experience Report

*Willian Roger Dullius, Carla Piardi, Undergraduate Student. Carolina Camine, Undergraduate Student. Déborah Ramos, Undergraduate Student. Dilana Ferreira, Undergraduate Student. Fernanda Busnello, Undergraduate Student. Fernanda Gnoatto, Undergraduate Student. Laura Sandoval, Undergraduate Student. Vera Da Rosa Haas, MSc. Professor of Biology. Ana Maria Bellani Migott, Dr. Nursing.*

The Rondon Project, coordinated by the Ministry of Defense of Brazil, is a project of social integration that involves the voluntary participation of academic students to find solutions that contribute to sustainable development of poor communities and expand the welfare of the population. During 22 days of July, 2013, the operation was realized in Ipixuna do Pará, where 8 students coordinated by 2 professors developed actions to combat Hypertension and other cardiovascular diseases.

**Object:** to report a successful experience thought Rondon Project as an essential part of undergraduate formation. **Method/ Results:** the actions of operation were developed in three distinct time. Before-operation: visit in loco, elaboration of plan action, training of students, and preparation of audiovisual materials. During: application of the plan, which consisted of to transmit knowledge between local population and students such as how cardiovascular diseases could be prevented, treated, and how local health professional could develop actions of health promotion using the technique wheel conversation. **After-operation:** elaboration of reports to Federal government of Brazil. As results for students, this was an opportunity to live the real situation of communities because just to look at the map of Brazil opened on the table work or nailed to the wall of our home is not enough. It is necessary to walk on its to feel close to people\'s anxieties, their hopes, their dramas or tragedies; their history, and their faith in the destiny of nationality. For those communities that are marginalized because their remote location, they had moments to find answers for their health doubts and to make a critical thinking about their daily practices that trigger diseases. In brief, the Rondon Project is a milestone for everyone involved in the project because it provides unique moments in the formation of involved citizen with public health.

## CLINICAL/OUTCOMES/POPULATION POSTER SESSION #1: 17:30 – 18:30: HALL & FOYER

### P24 – Improving hypertension detection and management in the community – a nation-wide approach through a grocery/pharmacy chain

*Shelley Diamond, Tsuyuki RT (Department of Medicine, University of Alberta), Kaczorowski J (Department of Family and Emergency Medicine University of Montreal and CRCHUM), Syron L (Princess Margaret Cancer Foundation), Berg A (Hypertension Canada), Farrell J (Hypertension Canada), Padwal R (Department of Medicine, University of Alberta), Feldman RD (Departments of Medicine and of Physiology & Pharmacology University of Western Ontario)*

**Background:** Undiagnosed, untreated, and undertreated hypertension remains a significant public health burden. Additional methods of detection and management are needed. Community pharmacies are accessible, visited frequently and are staffed with health professionals that can play an important role in hypertension care. **Methods/Results:** We conducted a before-after study in 470 Loblaw/DrugStore pharmacies across Canada from February 1-28, 2014. In-store signage and newspaper ads offered to any individual the opportunity to have a blood pressure (BP) consultation from a pharmacist. We followed CHEP-recommended procedures for BP measurement and used the validated PharmaSmart PS2000. All patients received feedback and recommendations based on their results as well as educational material endorsed by Hypertension Canada. Significantly elevated BP results were communicated to the subject's family physician according to a standardized protocol. In some locations we had a dietitian available to discuss low-sodium food choices and in some we performed a 60 day follow-up in subjects with SBP>150mmHg. We assessed 21,708 individuals (average age 58.7 (SD 16.8) y, 53% female). Average BP was 134.4 (SD 16.6)/78.3 (SD 11.3) mmHg, heart rate was 75.9 (SD 12.6), 58% self-reported taking antihypertensive medications in the past month and 21% had diabetes. In those without diabetes, 85% were at the BP target of <140/90 mmHg. In those with diabetes, 31% were at the BP target of <130/80 mmHg. A total of 3315 (15%) had a systolic BP >150 mmHg, for which 499 had pharmacy follow-up completed. Upon follow-up, 45% self-reported a reduction in BP, and 71% agreed or strongly agreed that in-pharmacy BP measurement had a positive impact on their health. **Conclusions:** In a >21,000-subject community-based screening program, >26% had BP levels above target. Pharmacy-based BP measurement is feasible, reaches many individuals in the community, and identifies those needing better hypertension care. Patients felt that the program improved their health.

## CLINICAL/OUTCOMES/POPULATION POSTER SESSION #1: 17:30 – 18:30: HALL & FOYER

### P25 – To study the prevalence and factors affecting treatment of resistant hypertension

*Shaylika Chauhan (M.B.B.S), Arundhati Kanbur (M.D Medicine)*
*# Clinical Research Associate, Department of Medicine, Rajiv Gandhi Medical College, India*
*# Ex-Department Chair, Department of Medicine, Kaushalya Medical Foundation and Trust Hospital*

**Introduction:** The magnitude of the burden of resistant hypertension needs an increase in awareness, treatment, and control of this condition. Recommendations for the pharmacological treatment of resistant hypertension remain largely empiric due to the lack of systematic assessments of 3 or 4 drug combinations, so this study was undertaken in 1192 hypertensive patients over a period of two years to assess prevalence and factors affecting treatment of resistant hypertension. **Methods:** Resistant hypertension was diagnosed by criteria as defined by WHO-ISH guidelines. All patients more than 18 years old, surgical and medical, admitted to the hospital with BP >150/90 and diabetic and chronic renal disease patients with BP >130/80 in spite of taking 3 antihypertensive drugs were diagnosed as resistant hypertension and formed the study cohort. Results: 233 patients (19.54%) out of a total of 1192 hypertensive patients admitted to the hospital in the study period were found to have resistant hypertension. In this study Diuretics was the most commonly used drug class (28.73% of all drugs used). 84 patients in all were taking 2 drugs as fixed dose combination medications, 13 patients were taking 4 drugs as fixed dose combination medications (2+2). 3 drugs fixed dose formulations were taken by 2 patients only. 66.67% of patients taking 7 drugs were unaware of the dose of their medications. 26.77% of patients who were taking three antihypertensive drugs were unaware of the doses of these medications. In all 23.17% were unaware of the doses of their antihypertensive medications. 82.83% of the study population gave a history of being compliant to their antihypertensive medications. 175 patients were taking analgesics which can potentially contribute to prevalence of resistant hypertension. Of these 175 patients 71 patients were on aspirin. **Conclusion:** Several classes of pharmacological agents which can increase blood pressure and contribute to treatment resistance should be avoided. Simplified prescribed regimens, longer acting combination drugs and diuretics in adequate dosage should be utilized.

## CLINICAL/OUTCOMES/POPULATION POSTER SESSION #1: 17:30 – 18:30: HALL & FOYER

### P26 – Resistance Hypertension: A simplified etiological classification

*Tawfik Albassam, Bayan Waez, Medical Reference Clinic, Jeddah, KSA (Kingdom of Saudi Arabia)*

**Background:** Several etiological classification of resistance hypertension (RH) is available. Most guidelines agree on the definition of RH. However, the terminology is confusing and may complicate the workup plan. Pseudo resistance, refractory, spurious, apparent RH are such terms which may confuse the topic. There is a need to simplify the etiological classification so that physicians can use in their daily work easily and formulate a workup plan accordingly without feeling confused.
**Methods:** we reviewed the most recent etiological classification and causes used for describing RH in the most recent guidelines. We attempted to avoid several confusing terms mentioned in the guidelines.
**Results:** We only used the terms primary and secondary RH analogues with hypertension. Therefor we suggest the following simplified etiological classification for RH:

1. Primary Resistance Hypertension: this corresponds to true RH .This is only diagnosed after exclusion of all causes mentioned below. Only this group should be considered for interventional therapy.
2. Secondary Resistance Hypertension: subdivided into :
    i) Disease related; include secondary causes like renal, endocrine, obstructive sleep apnea, coarctation of the aorta and other forms of secondary hypertension. Also included here is Pseudo Hypertension.
    ii) Not disease related ; subdivided into ;
        (1) Patient related : (a) White coat effect (b) Poor Adherence (c) Life style factors (d) Interfering agents or substances
        (2) Physician related : (a) Ineffective regimen (b) Measurement artifact

**Conclusion:** our suggested simplified etiological classification of resistance hypertension may be easier to follow at a busy clinic. Only two terms are used; primary and secondary RH. It includes all causes mentioned in the guidelines. It may be easily used as a frame or a foundation for work up of RH in the clinics. Only primary RH patients should be considered for interventional therapy. However, it needs to be tested in real clinical world.

# ABSTRACTS
## Friday, October 17

## CLINICAL/OUTCOMES/POPULATION POSTER SESSION #1: 17:30 – 18:30: HALL & FOYER

### P27 – Electrocardiogram-Assisted Blood Pressure Estimation in Patients with Atrial Fibrillation and other Chronic Conditions

*Saif Ahmad, Izmail Batkin, Health Parametrics Inc./University of Ottawa; Miodrag Bolic, Health Parametrics Inc./University of Ottawa; Hilmi R. Dajani, Health Parametrics Inc./University of Ottawa; Voicu Groza, Health Parametrics Inc./University of Ottawa; and Sanjeev Chander, Ottawa Cardiovascular Centre*

**Background:** Blood pressure (BP) is an important vital sign characterizing cardiovascular health. Therefore, BP management through accurate monitoring is critical for reducing risk of life-threatening conditions like stroke and myocardial infarction. Automated non-invasive BP (NIBP) devices are increasingly recommended in clinical practice and home monitoring. However, these devices tend to be unreliable in patients with chronic conditions like atrial fibrillation (AF), atherosclerosis, and obesity – resulting in inefficient BP management and hence increased risk. Unreliability arises because NIBP monitors estimate BP by analyzing arterial pulses alone and these patients may present weak, erratic, and/or unpredictable arterial pulses. **Method/Results:** Health Parametrics Inc., a University of Ottawa spinoff, is investigating a novel technology for increasing the accuracy and reliability of automatic NIBP estimation. Briefly, we have developed a simple method for simultaneous acquisition of electrocardiogram (ECG) and arterial pulse data within the automatic NIBP monitoring paradigm. Algorithms analyze arterial pulses with the assistance of ECG data, which tends to be less affected by the above conditions, to improve BP estimation accuracy. We recently conducted a pilot clinical investigation in which 13 patients (N=13) with chronic conditions including AF and obesity were recruited. For each patient, in about 30 minutes, 6 BP measurements taken by our prototype were compared with 6 BP measurements taken by BpTRU, a commonly used clinical NIBP device (78 measurements/device for N=13). The average systolic and diastolic BP measured by our prototype and BpTRU was statistically similar (Student\'s t-test, $p>0.05$). Moreover, standard deviation of systolic and diastolic BP measured by our device was lower than that of BpTRU in 77% and 69% of the patients respectively. **Conclusion:** These initial results suggest that our technology has the potential to improve the accuracy and reliability of NIBP estimation in patients with chronic conditions – leading to improved BP management and therefore reduced risk.

## CLINICAL/OUTCOMES/POPULATION POSTER SESSION #2: 15:30 – 17:00: HALL & FOYER

### P28 - Use of Web-based app to provide clinical decision support for hypertension at point of care

*Rahul Mehta, MD, University of Calgary, Aravind Ganesh, MD, Oxford University, Hisham Al-Shurafa BSc, Aleem Bharwani, MD, FRCPC Clinical Assistant Professor, University of Calgary*

**Background:** Medical research has exploded over the last few decades it almost become impossible for physician to keep up with the latest studies. For a general practitioner there are over 850 guidelines that they need to know and implement. Multiple studies have confirmed that less than 50% of care delivered in North America is as per the guidelines. We have a developed a Web and Ipad based software which allows for point of care delivery of latest Hypertension Guidelines. The tool has been deployed in multiple clinics and pharmacies in Ontario & Alberta as part of an evaluation study to see if it allows for more evidence based care. Consistent with our overall goal of creating a sustainable intervention, we will next mount an independent randomized controlled trial of the intervention, comparing knowledge/literacy and attitudinal outcomes between physicians and patients who will be randomized to an intervention group or control group. We intend to assess knowledge about screening and diagnosis, goals of treatment and treatment options for hypertension among the users before and after using the app.

## BIOMEDICAL ORAL SESSION #3: 08:30 – 08:45: CHAUDIÈRE A

### Mechanism implicated in the beneficial effects of (pro) renin/renin receptor blockade on weight gain and insulin sensitivity in obese mice

*Paul Tan1,2,5, Carolane Blais2, Catherine Michel1, Sonia Kajla1, Thi M.-D. Nguyen6, Peter W. Schiller6, Jolanta Gutkowska1,3 and Julie L. Lavoie1,4,5 1Centre de Recherche du Centre Hospitalier de l'Université de Montréal (CRCHUM) 2Department of Biochemistry and Molecular medicine, 3Medecine, and 4Kinesiology of the Université de Montréal, 5Montreal Diabetes Research Center, 6Institut de Recherches Cliniques de Montréal, and Montréal, Québec, Canada*

**Background:** We have previously found that the (pro) renin receptor [(P) RR] is increased in adipose tissue with obesity in mice. Obese mice treated with the handle region peptide (HRP), a (P) RR blocker, gained less weight and had decreased visceral adipose tissue. Improved glucose homeostasis was also observed. The aim of the study is to elucidate the mechanisms implicated in these beneficial effects.
**Methods/Results:** Mice were fed a normal or a high fat/high carbohydrate diet for 10 weeks in concomitance with saline or the HRP. Peri-gonadal fat (PGF), peri-renal fat (PRF) and blood were collected in mice at the end of the treatment. Diglyceride acytransferase 1 (DGAT1) expression, an enzyme implicated in the last step of triglyceride synthesis, decreased by 60% and 40% in PRF and PGF respectively in obese mice treated with the HRP. While dipeptidyl peptidase-4 (DPP-4) mRNA expression was found to be increased by 1.3-fold in PRF of obese mice independently of the treatment, protein levels were decreased by around 50% in both PGF and PRF of obese mice without any effect of the HRP. In addition, circulating DPP-4 activity seems to increase in obese mice while the HRP normalized it. **Conclusion:** Our results suggest that the HRP may reduce body weight and visceral fat pad size by lowering enzymes implicated in the synthesis of triglycerides. Decreased DPP-4 protein with obesity in adipose tissue has raised the possibility that the enzyme is secreted into the circulation where it degrades glucagon-like peptide-1 (GLP-1), a hormone that stimulates insulin secretion. Normalized circulating DPP-4 activity with the HRP suggests that this treatment may increase the half-life of postprandial circulating GLP-1 and thus improving glucose homeostasis.

## BIOMEDICAL ORAL SESSION #3: 08:45 – 09:00: CHAUDIÈRE A

### Endothelin-1 overexpression exaggerates diabetes-induced endothelial dysfunction

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

**Background:** Vascular disease associated with endothelial dysfunction is a major cause of morbidity in patients with type-1 diabetes. Endothelin (ET)-1 plays a role in diabetes-induced vascular complications, since ET-1 type A receptor blockade reduces diabetes-induced vascular injury. However, whether ET-1 contributes to diabetes-induced endothelial dysfunction remains unproven. We hypothesized that vascular ET-1 overexpression 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 mice and in mice overexpressing ET-1 restricted to the endothelium (eET-1). Mice were studied 14 weeks later. Blood was collected to determine glucose. Mesenteric artery reactivity and remodeling were evaluated using pressurized myography and aortic fibronectin expression by immnunofluorescence. STZ-induced diabetes was confirmed by a 3-fold increase in glycaemia in wild-type mice and eET-1 Diabetes impaired endothelium-dependent relaxation (EDR) reponses to acetylcholine by 27% in wild-type mice ($P<0.05$) and by 40% in eET-1 ($P<0.001$). Diabetes-induced EDR impairment was exaggerated in eET-1 compared to wild-type mice ($P<0.05$). Meclofenamic acid, an inhibitor of cyclooxygenase, increased EDR by 18% in eET-1 compared to wild-type mice ($P <0.01$), which was not observed in diabetic mice. L-NAME, an inhibitor of nitric oxide (NO) synthase, completely blocked EDR in wild-type, eET-1 and diabetic wild-type mice, but reduced by 45% EDR in diabetic eET-1 ($P<0.05$). Apamin plus Tram34, inhibitors of endothelium-dependent hyperpolarization, inhibited EDR in the four groups. Endothelium-independent relaxation to sodium nitroprusside, a NO donor, was similar in the four groups. Diabetes reduced media/lumen by 25% in both wild-type mice and eET-1 ($P<0.05$). Diabetes decreased by 38% aortic fibronectin expression in wild-type mice ($P<0.05$) and 55% in eET-1 ($P<0.05$). **Conclusion:** ET-1 contributes to alterations in several pathways mediating endothelium-dependent relaxation in type-1 diabetes, leading to exaggerated endothelial dysfunction.

# ABSTRACTS
## Saturday, October 18

## BIOMEDICAL ORAL SESSION #3: 09:00 – 09:15: CHAUDIÈRE A

### Endoplasmic reticulum stress inhibition prevents chronic kidney disease independent of blood pressure lowering effects in the Dahl S rat

*Rachel E. Carlisle, Katelyn A. Colwell, Savas D. Kanaroglou, Chao Lu, Victoria Yum, Jeffrey G. Dickhout, Department of Nephrology, St Joseph's Healthcare Hamilton and McMaster University, Hamilton, Ontario, Canada*

**Introduction:** The Dahl salt sensitive (DSS) rat is commonly used as a model of proteinuric chronic kidney disease (CKD). Our previous research demonstrated that inhibiting endoplasmic reticulum (ER) stress using 4-phenylbutyrate (1 g/kg/day), a small molecular chaperone, lowered blood pressure (BP) and prevented kidney damage in DSS rats on a high salt diet (HSD; 8% NaCl). It remained unclear whether the kidneys were protected from damage via inhibition of ER stress or due to the lowering of BP. Thus, we investigated the effects of lowering BP with a non-specific vasodilator, hydralazine, which has no effects on ER stress. **Method/Results:** 12-week old male DSS rats were fed a normal salt diet (NSD; 0.4% NaCl), a HSD, or a HSD supplemented with the vasodilator hydralazine (15 mg/kg/day) for 4-weeks. HSD-fed rats experienced a significant increase in BP, as measured by tail cuff plethysmography, and directly through cannulation of the carotid artery. This increase in BP was prevented in HSD-fed rats treated with hydralazine, which lowered BP to a similar degree as 4-phenylbutyrate treatment. Markers of CKD that were elevated in the HSD-fed DSS rats included proteinuria, albuminuria, increased protein cast formation, and increased renal interstitial fibrosis. Using urinalysis, as well as periodic acid-Schiff-stained, Masson's Trichrome stained, and a-smooth muscle actin-stained kidneys, we established that reducing BP using hydralazine treatment did not prevent any of these markers of CKD. **Conclusion:** Lowering BP with the non-specific vasodilator hydralazine does not prevent CKD in the DSS rat. However, ER stress inhibition with 4-phenylbutyrate, which lowers BP to a similar degree as hydralazine, prevented CKD in the DSS rat, as demonstrated through reduced proteinuria, albuminuria, protein cast formation, and renal interstitial fibrosis. Thus, it appears that ER stress inhibition has independent effects to inhibit hypertension and prevent CKD induced by proteinuria.

## BIOMEDICAL ORAL SESSION #3: 09:15 – 09:30: CHAUDIÈRE A

### A somite-derived Sox2+ stem cell in the adult mouse aorta gives rise to myeloid cells

*Sarah K. Steinbach, Martha H. Carruthers2, Eric A. Shikatani6, Caleb C. Zavitz3, David Smyth3, Clinton S. Robbins3,6-8 and Mansoor Husain1,2,4-6,8 1McEwen Centre for Regenerative Medicine, and Divisions of 2Experimental Therapeutics and 3Advanced Diagnostics, Toronto General Research Institute, 101 College Street, Toronto, Ontario, Canada, M5G 1L7. Departments of 4Medicine, 5Physiology, 6Laboratory Medicine & Pathobiology, 7Immunology and the 8Heart and Stroke Richard Lewar Centre of Excellence, University of Toronto, 1 Kings College Circle, Toronto, Ontario, Canada, M5S 1A8*

**Background:** The vasculature is derived from many different sources during embryonic development, including neural crest, and mesoderm of splanchnic, pharyngeal and somitic origin. Vascular smooth muscle cells (VSMC) of the aorta are similarly derived from several embryonic sources; however the developmental and phenotypic heterogeneity of their progenitors is poorly understood. **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. Intriguingly, a population of lineage-marked cells that express high levels of the reporter tdTomato (tdT-hi) is uniquely expressed in the aorta, and not found in blood, bone marrow or spleen. In Myf5- and T-marked lineages, this tdT-hi population gave rise to clonally-derived spheres that differentiated into VSMC, adipocytes, S100β-positive neural cell and macrophages in vitro. Consistent with the observed progenitor activity of the tdT-hi population, a subset of these cells co-express Sox2 and Sca1, known adult stem/progenitor cell markers. Further evidence of progenitor activity was demonstrated in an in vitro CFU assay where tdT-hi cells gave rise to F4/80+CD11b+ macrophages and CD11b+Ly6G+ neutrophils. Complementary analyses performed in mice demonstrated that leukocytes such as macrophage, and neutrophils were found to be lineage-marked cells suggesting derivation from somitic mesoderm. Indeed, somite-derived leukocytes were enriched over 6-fold in the proximal aorta vs. the blood, bone marrow and spleen of these mice. Parabiosis experiments suggest that somite-derived aortic progenitors are not readily replaced by circulating precursors and bone marrow transplant of Myf5-cretdTomato mice show that these progenitors are radiosensitive. Transplantation of tdT-hi 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. **Conclusion:** The aorta harbours a resident Sox2+ stem cell that gives rise to macrophages and neutrophils in the steady state. The role of this progenitor in aortic pathology such as atherosclerosis is currently being investigated.

## CLINICAL/OUTCOMES/POPULATION ORAL SESSION #3: 08:00 – 08:15: CHAUDIÈRE B

### SD-OCT Measurement of Serial Change in Retinal Macular Thickness During Normal Pregnancy: Proof-of-Concept Data for a Tool to Study Dynamic Changes in Function of the Microcirculation in Humans

*Robert J. Herman, MD, T. Lee-Ann Hawkins, MSc, MD, R. Geoff Williams, BSc, MD, Nancy Clayden, RA, Anshula Ambasta, BSc, MD. University of Calgary and Ward of the 21st Century.*

**Background:** The microcirculation is the largest and most functionally dynamic portion of the vascular tree. A recent report from single SD-OCT measurement of retinae in normal pregnancy demonstrated that the subfovial region of the macula thins whereas the subfovial choroid layer increases in thickness1. These likely represent physiologic adaptations to change in cardiac output and permeability that accompany pregnancy in a tissue which auto regulates its microcirculation. Our objective was to validate these findings by sequential measurements in patients as they progressed though pregnancy and to look for possible mal-adaptation of this response in preeclampsia (PET). **Methods/Results:** We enrolled 29 normal pregnant and 20 pregnant patients at high-risk for PET and sequentially examined both retinae at gestational ages ≤ 20 wks., 20-40 wks., at delivery and up to 12wks post-delivery. The Test/Re-test Coefficient of Reproducibility of our Zeiss Cirrus 4000 SD-OCT was examined on baseline images and found to be ±4.0μm for all segments on the conventional Early Treatment of Diabetic Retinopathy Study (ETDRS) macular scale. Thus, within eye variation ≥ ±5μm in any single segment or ≥ ±4μm in 3 or more contiguous segments was considered potentially meaningful. We observed 5-10μm macular thinning in many ETDRS segments in 7 of 8 profiles from normal pregnant females (others are not yet at a stage where this can be evaluated) and 3 of 3 high-risk patients that did not go on to develop PET. Macular thinning occurs early (baseline scans after 15 wks. often miss it), but show recovery of normal retinal thickness following delivery. One normal pregnant patient developed PET; she 'wet' her macula at 30 wks. **Conclusions:** 1) the macula thins in normal pregnancy; 2) SD-OCT can reliably detect these changes and may be a tool for examining micro vascular integrity in health and disease. (1. Hypertens in Preg; DOI:10.3109/10641955.2013.877924)

## CLINICAL/OUTCOMES/POPULATION ORAL SESSION #3: 08:15 – 08:30: CHAUDIÈRE B

### Arbitrary pharmacy switching between differing nifedipine osmotic delivery formulations leads to unexpected variability in blood pressure for majority of patients

*Paul Timothy Pollak, Navdeep Dehar, Robert J. Herman, Kelly B. Zarnke, Ross D. Feldman. Departments of Medicine, University of Calgary and Western University*

**Background:** Switching between "branded" and generic antihypertensive medications is almost universal in the current Canadian market. Regulatory approval of generic formulations is based solely on non-time-based pharmacokinetic parameters. Health Canada does not consider effectiveness. Concerns have been raised about assessment methods for generic versions of modified-release drugs. Anecdotal reports of BP differences between Mylan-nifedipine ER (MyN) and "branded" Adalat XL (AdN) formulations are consistent with in vitro dissolution studies suggesting MyN 24-hour drug release is only ~80% that of AdN. Further, based on MyN's first-order drug release profile vs. AdN's zero-order drug release, a less sustained antihypertensive effect would be expected, especially at the end of the dosing interval. **Methods/Results:** To determine whether these considerations translate into reduced antihypertensive effectiveness, we used a cross-over design to study 10 subjects receiving daily morning AdN 60 mg treatment vs. matching dose MyN. At the end of each 2-week dosing period, 24-hr ambulatory BP was assessed. SBP, both for 24 hours and for the last 8 hours (22:00 h - 06:00 h) was examined. Blood pressures were significantly lower in patients while treated with AdN. Mean ± SE 24-hour SBP was 135 ± 2.6 mmHg with AdN, and 139 ± 2.5 mmHg with MyN (p=0.03). For the last 8 hours of the dosing interval, mean ± SE nocturnal SBP was 128 ± 4.0 mmHg with AdN and 134 ± 2.9 mmHg with MyN (p=0.02). **Conclusion:** Mean 24-h SBP was statistically significantly higher in patients when taking MyN, than when taking AdN. Although deemed bioequivalent, differences in both: extent of drug delivery; and timing of delivery of a drug with only a 2-hour half-life, which could allow important concentration fluctuations are likely responsible for observed SBP differences. Our results indicate that arbitrary nifedipine switching by pharmacies may lead to unexpected variability in BP control.

# ABSTRACTS
## Saturday, October 18

## CLINICAL/OUTCOMES/POPULATION ORAL SESSION #3: 08:30 – 08:45: CHAUDIÈRE B

### Hypertension Treatment and Control in the Community: A novel program of surveillance for hypertension in a grocery-pharmacy setting

*Shelley Diamond, Tsuyuki RT (Department of Medicine, University of Alberta), Kaczorowski J (Departments of Family and Emergency Medicine, University of Montreal and CHCHUM), Syron L (The Princess Margaret Cancer Foundation), Berg A (Hypertension Canada), Farrell J (Hypertension Canada), Padwal RS (Department of Medicine, University of Alberta), Feldman RD (Departments of Medicine, Physiology and Pharmacology, University of Western Ontario)*

**Introduction:** Undiagnosed, untreated, and undertreated hypertension remains a significant public health burden. We need ongoing community-based methods of surveillance. Pharmacies located in grocery stores are visited frequently by the public, and represent a unique opportunity for blood pressure (BP) screening and awareness activities. **Methods/Results:** We conducted BP consultations in 470 Loblaw/DrugStore pharmacies across Canada from February 2013 to February 2014. In-store signs and newspaper ads offered individuals the opportunity to have a blood pressure (BP) consultation from a pharmacist. Blood pressure measurements were performed using the well-validated PharmaSmart PS2000 kiosk, and followed Canadian Hypertension Education Program (CHEP)-recommended procedures for BP measurement. All patients received feedback and recommendations based on their results as well as educational material endorsed by Hypertension Canada. Significantly elevated BP results were communicated to the subject's family physician according to a standardized protocol. We assessed 53,027 individuals (average age 59 (SD 16.9) years, 51% female). Average BP was 133 (SD 16.6)/77.9 (SD 11.4) mmHg, heart rate was 76.2 (SD 12.9) beats/minute. A total of 52% reported taking antihypertensive medications in the past month and 18.4% had diabetes. In those 43,552 subjects without diabetes, 42% achieved the BP target of <140/90 mmHg. In the 9,475 subjects with diabetes, 16.5% achieved the BP target of <130/80 mmHg.
**Conclusions:** In this ongoing screening program, we screened over 50,000 community-dwelling adults. BP treatment and control in 2013-2014 is no better than that reported in Ontario in 2006 and the National Population Health Survey from 2007-2009. Indeed, 58% and 84% of subjects without and with diabetes, respectively, were above recommended BP levels. Pharmacy-based interventions through major pharmacy chains offer a novel approach in the assessment and implementation of new management approaches in the treatment of hypertension.

## CLINICAL/OUTCOMES/POPULATION ORAL SESSION #3: 08:45 – 09:00: CHAUDIÈRE B

### Psychosocial work factors and ambulatory blood pressure: repeated exposure to demand-control and effort reward imbalance models over 5 years

*Xavier Trudel, Chantal Brisson Université Laval, Alain Milot, Université Laval, Benoit Masse Université de Montréal, Michel Vézina Université Laval*

**Background:** Two main theoretical models have been used to assess the impact of psychosocial work factors on blood pressure (BP): the demand-control model (DC) and the effort-reward imbalance (ERI) model. Previous studies have mostly used a single-time point exposure to examine this association. **Objective:** to examine the effect of baseline and repeated job strain and ERI exposure on 1) ambulatory BP (ABP) evolution over 5 years and 2) hypertension incidence over 5 years. **Method:** The design is a prospective cohort study. The study population was composed of 1,586 white-collar workers (662 men and 924 women). They were assessed three times during a 5-year period (Year 1, 3 and 5). At each time, psychosocial work factors were measured using validated scales and ABP was measured every 15 minutes during a working day. **Results:** In the repeated exposure analyses, men who were always exposed to an active job had higher systolic and diastolic ABP increases (+2.7/+2.5 mmHg) and a higher cumulative incidence of hypertension (RR = 2.20, 95% C.I. 1.50–3.23), compared to never exposed men. In women, ERI exposure onset was associated with higher increases in systolic (+2.8 mmHg) and diastolic (+1.6 mmHg) ABP, compared to never exposed women. **Conclusion**: Repeated exposure to psychosocial work factors from the DC and the ERI models over 5 years led to ABP increases and hypertension incidence, while no such deleterious effect was found using a single time-point exposure. Results from the present study highlight the need to consider repeated exposure in future studies.

## BIOMEDICAL ORAL SESSION #4: 10:00 – 10:15: CHAUDIÈRE A

### Systemic Inflammation, Hypertension, and Changes in Middle Cerebral Artery Function

*Amy Randell, Waghmare, K., Daneshtalab, N., School of Pharmacy, Memorial University of Newfoundland*

**Introduction:** Patients with autoimmune disease, such as rheumatoid arthritis (RA), have a higher incidence of hypertension and stroke than the normal population. Hemorrhagic stroke (HS) is associated with loss of cerebrovascular function; the vessels cannot constrict in response to increases in systemic blood pressure, leading to micro-vessel burst, and hemorrhage. We believe chronic inflammation causes the cerebrovasculature to lose the ability to respond properly to pressure, leading to increased incidence of HS. We investigated this link using an animal model. **Methods:** We established a hypertensive-arthritis model in spontaneously hypertensive rats (SHR), and compared them to non-inflamed SHR and stroke-prone SHR (SHRsp, upon stroke development) (n=3-7/group). Arthritis was induced by intradermal injection of Complete Freund's adjuvant (CFA) into the left paw of the rats. High salt diet was used to induce accelerated hypertension. The rats were monitored for signs of inflammation and hypertension. At the end of treatment, the animals were sacrificed and the middle cerebral artery (MCA) isolated. The ability of the MCAs to undergo pressure dependent constriction (PDC) and react to vasoactive peptides was evaluated. **Results and Discussion:** All three groups exhibited increase in blood pressure with the high salt diet. MCAs of post-stroke SHRsps failed to undergo PDC, or react to vasoactive peptides compared to healthy animals. The MCA of animals inflamed with CFA also exhibited decrease in ability to undergo PDC, and a diminished degree of response to vasoactive peptides. **Conclusion:** It is possible that inflammatory mediators are involved in weakening the MCA's and thus decreasing its ability to respond to PDC. This in turn would increase the chances of HS development.

## BIOMEDICAL ORAL SESSION #4: 10:15 – 10:30: CHAUDIÈRE A

### c-Myb regulates specific Sca1+ adventitial cell populations following injury

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**Background:** We have shown that the proto-oncogene c-myb regulates the proliferation and differentiation of vascular smooth muscle cells (VSMCs). Previously, we showed that hypomorphic (h/h) mice harboring a non-lethal point mutation in c-myb resulting in defective c-Myb activity have decreased neointimal hyperplasia versus wild-type (wt) mice following carotid injury (CI). The adventitia contains a population of stem cell antigen (Sca1) positive progenitor cells that are able to differentiate into VSMCs. These cells can contribute to neointimal remodeling following injury. It remains unknown if c-Myb regulates the differentiation of vessel-resident progenitor cells. **Hypothesis:** c-Myb regulates the proliferation and differentiation of vessel-resident adventitial Sca1+ (AdvSca) progenitor cells. **Methods/Results:** Histological analysis of wt and h/h carotid arteries showed no differences in adventitial Sca1 expression. Flow cytometric analysis of enzymatically digested carotid arteries shown no differences in the number of CD45- Lin- Sca1+ cells in wt and h/h arteries, suggesting that c-myb does not regulate the development of AdvSca cells. We examined the expression of markers previously shown to be expressed on AdvSca cells, and found that co-expression of cKit, CD34 and Flk1 did not significantly differ between wt and h/h AdvSca cells. In wt carotid arteries D8 post-injury, Sca1+cKit+ cells were increased ~10 fold compared to uninjured arteries. However, in h/h arteries, Sca1+cKit+ cells were only increased ~3 fold compared to uninjured arteries, suggesting a defect in the expansion of cKit+ AdvSca cells. No differences in Sca1+CD34+ or Sca1+Flk1+ cells were found at D8 between wt and h/h arteries. **Conclusion:** While c-myb appears not to regulate the development of AdvSca cells, c-myb does regulate the expansion of specific subsets of ckit+ Sca1+ AdvSca cells following injury.

## BIOMEDICAL ORAL SESSION #4: 10:30 – 10:45: CHAUDIÈRE A

### ER stress inhibition decreases inflammatory response in a CKD mouse model

*Zahraa Mohammed-Ali, Rachel E. Carlisle, Chao Lu, Kjetil Ask, Jeffrey G. Dickhout, Medicine, Division of Nephrology, McMaster University and St. Joseph's Healthcare Hamilton*

**Background:** Immune cell infiltration in the kidney is reported in animal models of salt-sensitive and Angiotensin (Ang) II-induced hypertension. Hypertensive patients have increased levels of C-reactive protein, TNFα, IL-6, MCP-1 and adhesion molecules. NFκB activation increases in patients with glomerulonephritis, diabetic nephropathy and acute kidney injury and results in disease progression. Endoplasmic reticulum (ER) stress is an imbalance between protein-folding capacity and protein demand and is important in the pathogenesis of chronic kidney disease (CKD). ER stress markers GRP78 and CHOP are up regulated in kidneys from glomerulonephritis and nephrotic syndrome patients. Activation of ER stress pathways has been shown to mediate proinflammatory transcriptional programs regulated by NFκB. Therefore, we hypothesized that inhibiting ER stress using 4-phenylbutyrate (4-PBA), a molecular chaperone, would reduce the chronic inflammatory response in CKD thereby halting disease progression. **Methods/Results:** CKD was induced in male C57BL/6 mice by uninephrectomy and subcutaneous implantation of an Ang II osmotic infusion pump and a slow release deoxycorticosterone acetate (DOCA) pellet. Mice were placed on 1% sodium chloride in their drinking water and 4-PBA-treatment was administered in drinking water. Ang II/DOCA mice treated with 4-PBA experienced a significant decrease in hypertension and qRT-PCR analysis showed decreased GRP78, CHOP, sXBP1 levels compared to non-treated Ang II/DOCA mice. 4-PBA-treatment also led to lower T cell and macrophage infiltration. Nanostring analysis showed that 4-PBA treatment in the AngII/DOCA model led to decreased pro-inflammatory gene expression of NFκB and IP-10 and an increase in anti-inflammatory genes, IL-10 and Foxp3, compared to non-treated Ang II/DOCA mice. **Conclusion:** Our findings show an association between ER stress inhibition and down regulation of inflammatory mediators and immune cell infiltration. 4-PBA treatment also correlated with induction of regulatory agents, IL-10 and Foxp3. By identifying molecular targets and pathways in CKD, these findings will allow development of therapeutics to reduce disease progression.

## BIOMEDICAL ORAL SESSION #4: 10:45 – 11:00: CHAUDIÈRE A

### Endothelin-1 overexpression preserves endothelial function in mice with vascular smooth muscle cell-specific deletion of PPAR-gamma

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**Background:** Peroxisome proliferator-activated receptor γ (PPARγ) agonists reduce blood pressure (BP) and vascular injury in hypertensive rodents and humans. Pparγ inactivation in vascular smooth muscle cells (VSMC) using a tamoxifen inducible Cre-Lox system enhanced angiotensin II-induced vascular remodeling and endothelial dysfunction. Transgenic mice overexpressing endothelin (ET)-1 selectively in the endothelium (eET-1) exhibit endothelial dysfunction, increased oxidative stress and inflammation. We hypothesized that inactivation of the Ppar gene in VSMC (smPparγ-/-) will exaggerate ET-1-induced vascular damage. **Methods/Results:** Eleven week-old male control, eET-1, smPparγ-/- and eET-1/smPparγ-/- mice were used. BP was determined by telemetry, mesenteric artery (MA) reactivity and structure by pressurized myography, reactive oxygen species (ROS) by dihydroethidium staining and expression of inflammatory markers by immunofluorescence. Systolic BP was 10 to 20 mmHg higher in eET-1 and eET-1/smPparγ-/- compared to control and smPparγ-/- ($P<0.05$). Endothelium-dependent relaxation (EDR) responses to acetylcholine were impaired 37% in smPparγ-/- ($P<0.05$) but not in eET-1 and eET-1/smPparγ-/- compared with control. Endothelium-independent relaxation responses to the nitric oxide donor, sodium nitroprusside, were similar in all groups. Media/lumen at 45 mmHg was increased 20% in eET-1/smPparγ-/- compared with control ($P<0.05$). A similar increase in MA stiffness was observed in eET-1, smPparγ-/- and eET-1/smPparγ-/- compared to control, as indicated by a leftward displacement of the stress-strain curves ($P<0.05$). ROS levels were 1.7-fold greater in eET-1, 2.2-fold in smPparγ-/- and 2.8-fold in eET-1/smPparγ-/-compared with control ($P<0.05$). Monocyte chemoattractant protein-1 levels were 1.7-fold higher in smPparγ-/- compared with control ($P<0.05$), which was not exaggerated by ET-1 overexpression. Monocyte/macrophage specific antigen-2-positive cells in perivascular fat were ~2-fold higher in eET-1 and in smPparγ-/- compared with control ($P<0.05$), which was further increased 2.0-fold in eET-1/smPparγ-/- ($P<0.05$). **Conclusion:** These results suggest that increased ET-1 paradoxically preserves endothelial function in mice with inactivated VSMC Pparγ, despite enhanced oxidative stress and inflammation.

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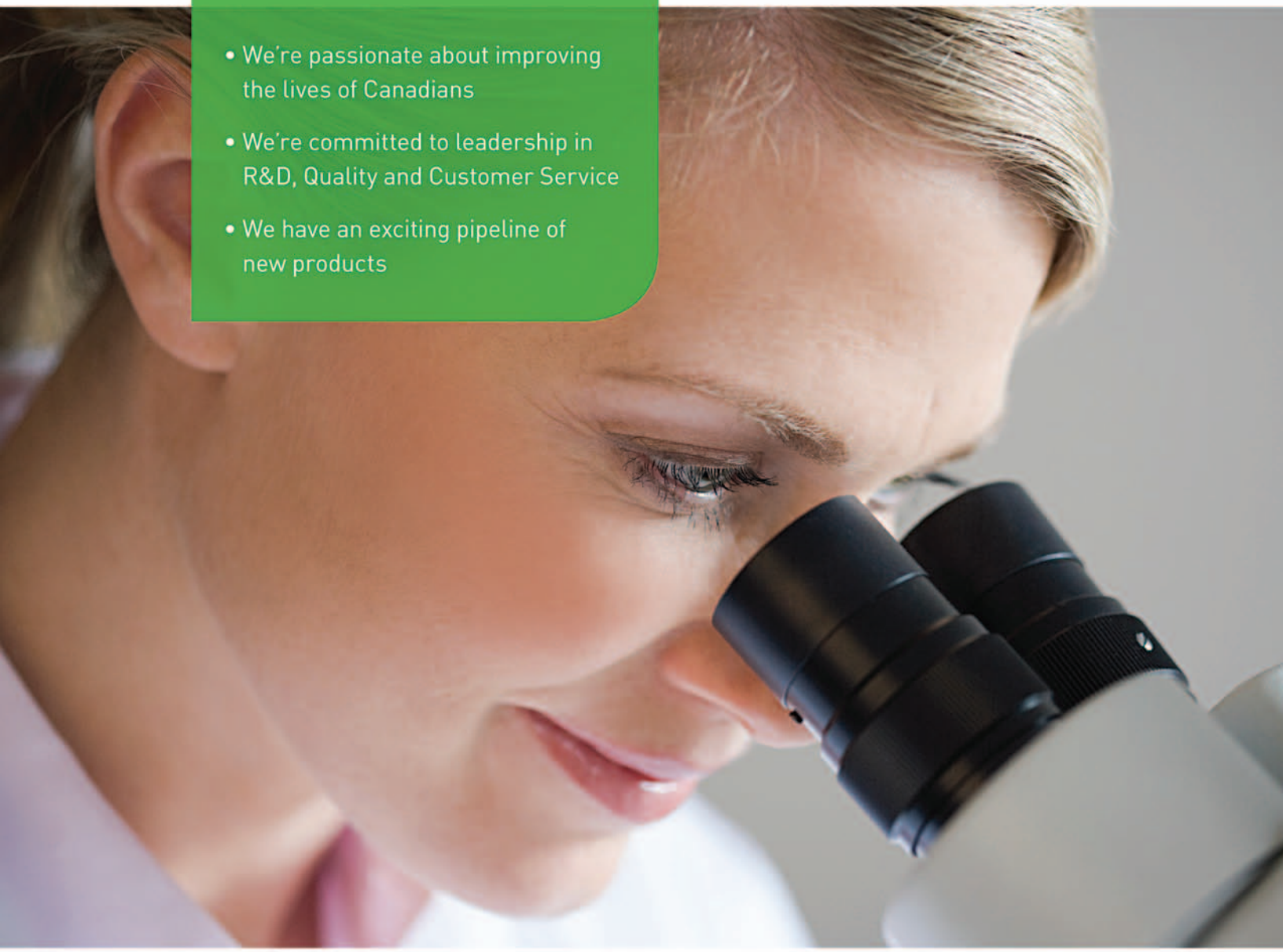

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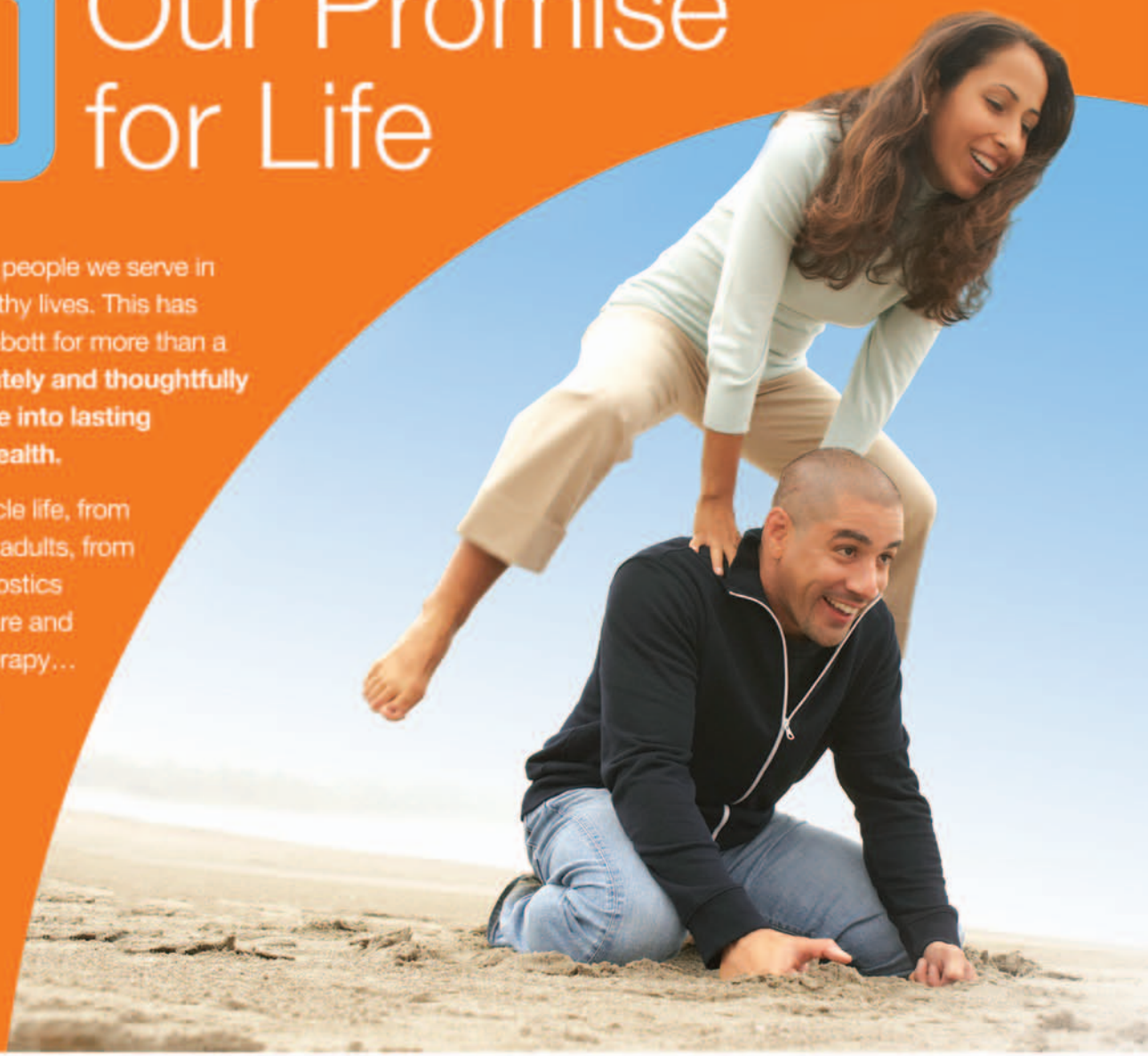

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