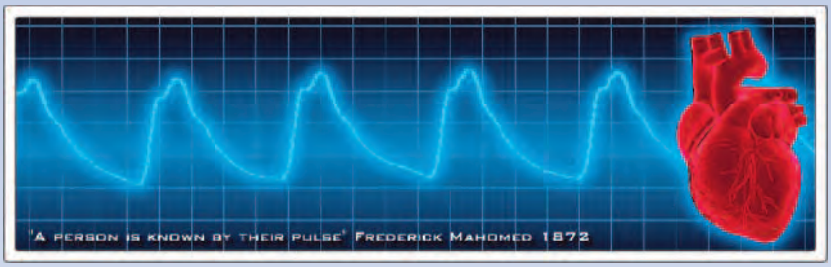
NORTH AMERICAN ARTERY



FINAL PROGRAM

THIRD ANNUAL MEETING

Arterial Hemodynamics: Next Steps Towards Clinical Applications

September 6-7, 2013

Hyatt Regency O'Hare

Rosemont, IL

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ABOUT THE NORTH AMERICAN ARTERY SOCIETY

Mission Statement

The Mission of North American Artery Society is to:

- Support education on arterial structure and function appropriate to the various medical communities, such as scientific researchers, clinical specialists, primary care specialists, medical students, and pharmaceutical researchers, as well as the patient community;
- Develop mechanisms and venues for disseminating information on the understanding and application of arterial structure and function and its measurement among the various medical communities;
- Participate in and encourage the study of improved application of technologies in the measurement of arterial structure and function;
- Participate in and encourage clinical trials that develop the understanding of how arterial structure and function and its measurement can guide and inform patient selection and treatment;
- Guide and support efforts to standardize arterial structural and functional measurements for clinical practice and clinical/scientific studies;
- Direct efforts to include arterial structure and function measurements in appropriate national guidelines;
- Formulate a consensus statement regarding what is known in regards to arterial structure and function.

Society Objectives

North American Artery is a non-profit, non-partisan professional society dedicated to the encouragement, support, and understanding of vascular structure and function and its application to clinical medicine, research and pharmaceutical and medical device development. The Society Objectives are to:

- Support education on arterial mechanics appropriate to the various medical communities, such as scientific researchers, clinical specialists, primary care specialists, and pharmaceutical researchers, as well as the patient community;
- Develop mechanisms and venues for disseminating information on the understanding and application of arterial mechanics and its measurement among the various medical communities;
- Participate in and encourage the study of arterial mechanics in basic and applied research to further especially the clinical applications derived from an improved understanding of arterial mechanics;
- Participate in and encourage clinical trials that develop the understanding of how arterial mechanics and its measurement can guide and inform patient treatment;
- Guide and support efforts to standardize arterial mechanics measurements for clinical practice and clinical/scientific studies;
- Direct efforts to include arterial mechanics measurements in appropriate national guidelines;
- Provide the knowledge for the critical understanding and application of technologies to measure arterial mechanics.

ACKNOWLEDGEMENT

The North American Artery Society wishes to acknowledge the following organizations for their support of the Third Annual Meeting.

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TABLE OF CONTENTS

NAA Application for Membership is available at the Registration Desk.

PRESIDENT'S WELCOME

NORTH AMERICAN ARTERY

North American Artery
12734 Cimarron Path
San Antonio, TX 78249

Dear Colleagues,

I'd like to personally welcome each of you to our Third Annual Meeting, "Arterial Hemodynamics: Next Steps towards Clinical Applications". The field of arterial hemodynamics is an exciting area and the North American Artery Society will continue to meet and bring inspired people together in forums like this, to ensure the NAA remains at the cutting edge of research and the practical applications for technologies that measure arterial structure and function.

I appreciate the efforts of our Conference Co-Chairs, Bo Fernhall and Gary Pierce, as well as Peter Feig and Dean Winter for working tirelessly with me to put together this program. We are also indebted to Hansen Global Event Management for such excellent logistical support.

I look forward to meeting our members, both old and new who will be here. For those of you attending who are not members, there is information about the NAA included within this book, and membership applications are available at the registration desk. I encourage you to think about being a part of our new and exciting organization.

This conference would not be possible without the generous support of our Platinum sponsors, AtCor Medical, Inc. and I.E.M. GmbH, and our Gold sponsors, Fukuda Denshi, HealthSTATS International, and Omron Healthcare, Inc. The NAA is grateful to each of them for their support of our organization. Please visit with them in the exhibit area during our breaks.

In closing, I would like to thank each of you for attending the conference and bringing your expertise to our gathering. Your vision, knowledge, and experience will help us pave the way for future developments in arterial studies. Throughout this conference, you are encouraged to engage faculty and sponsors. My personal respect and thanks to all!

Sincerely,

Raymond R. Townsend, M.D.
NAA President

PROGRAM COMMITTEE CO-CHAIRMEN WELCOME

NORTH AMERICAN ARTERY

Arterial Hemodynamics: Next Steps Towards Clinical Applications

The 3rd Annual Meeting of the North American Artery Society

Friday, September 6 – Saturday, September 7, 2013

Hyatt Regency O'Hare · Rosemont, IL

Dear Colleagues,

On behalf of the North American Artery Society (NAA), it is our distinct pleasure to welcome you to the 3rd Annual meeting of NAA, "Arterial Hemodynamics: Next Steps Towards Clinical Applications". The NAA is a multidisciplinary society dedicated to understanding vascular structure and function and its application to clinical medicine, basic/translational research, and pharmaceutical and medical device development. The breadth of this year's program clearly reflects these objectives with presentations focusing on clinical perspectives and prognosis, impact of drugs on blood pressure and arterial hemodynamics, new devices for determining central arterial hemodynamics, lifestyle interventions, reimbursement issues, and basic and translational science. This exciting program is delivered through main lectures, debates, oral and poster abstract presentations and exhibits.

We truly hope you will enjoy the 2013 NAA meeting at the Hyatt Regency O'Hare and that you take the opportunity to meet our speakers, delegates and exhibitors. We would especially like to thank our sponsors AtCor Medical, I.E.M., Fukuda Denshi, HealthSTATS and Omron Healthcare for making this exciting and scientifically enriching conference possible. Thank you for participating and helping to move the NAA forward as our organization continues to grow.

Sincerely,

Bo Fernhall, Ph.D.

Gary Pierce, Ph.D.

GENERAL INFORMATION

Meeting Venue/Headquarters Hotel
Hyatt Regency O'Hare

The sophisticated, upscale hotel provides registrants with effortless convenience to downtown Chicago and O'Hare airport. Recently refreshed, the hotel has a long tradition of service and extraordinary convenience and now features new amenities, newly refreshed guestrooms, and contemporary styling. With cutting-edge design and stunning lobby with impressive atrium, the hotel offers a striking modern environment.

Attendees staying at the hotel receive complimentary internet access in their rooms.

Airport Shuttle

The Hyatt Regency O'Hare offers 24 hour, complimentary shuttle service, which runs every 15 minutes. Follow the red Shuttle signs at the airport to the designated pick-up area, O'Hare Bus / Shuttle Center Door One. Shuttle buses are blue with white signage that reads Hyatt Regency O'Hare.

Valet and Self-Parking

Self-Parking – $20 daily or $22 overnight.
Valet Parking – $30 daily or $32 overnight.

Hotel Amenities

- StayFit Fitness Center—24/7 state of the art gym
- Perks Coffee & Gift Shop—On-site convenience/gift shop that is open 22 hours/day
- Room Service available 24 hours
- Full Service FedEx Office & Business Center open 24 hours daily
- Complimentary high-speed wireless access in all public spaces

Restaurants

- Oh' American Grill – American Cuisine
- Red Bar – Sushi Bar; Innovative cocktails & food

Meeting Registration – Rosemont Ballroom Foyer

All Conference materials including badges can be picked up from the registration desk during the following hours:
September 6, 2013 2:00 PM - 8:00 PM
September 7, 2013 6:30 AM - 12:00 PM

Badges are required for entry to all functions.

Conflict of Interest Disclosure

The North American Artery Society strives to ensure balance, independence, objectivity, and scientific rigor in its educational activities. Faculty members and Program Committee Chairpersons have disclosed to the Society financial relationships with commercial interests or manufacturers with products associated with or discussed in their presentation, in existence over the past 12 months. All Disclosure Statements are available to meeting attendees in the Program Book.

Posters on Display

Posters will be on display throughout the conference. Presenters will be available to discuss their posters during the Lunch on Saturday.

Exhibits

Meal functions, except the dinner, will be held in the exhibit hall as shown below.

Friday, September 6, 2013

Opening Reception	3:00 to 4:00 PM

Saturday, September 7, 2013

Breakfast	7:30 to 8:15 AM
Coffee Break	9:00 to 9:30 AM
Lunch	1:00 to 2:30 PM

Third Annual Meeting Sponsors

The North American Artery Society wishes to acknowledge the following Corporate Sponsors for their generous support of the NAA Third Annual Scientific Meeting.

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CHAIRPERSONS AND FACULTY DISCLOSURES

Julio A. Chirinos, MD, PhD, Assistant Professor of Medicine, University of Pennsylvania and Director of Non-Invasive Cardiac Imaging, Philadelphia VA Medical Center, has no conflict of interests to disclose.

Stella Daskalopoulou, MD, MSc, DIC, PhD, Associate Professor in Medicine and Director, Vascular Health Unit, Department of Medicine, Division of Internal Medicine, McGill University, McGill University Health Centre, has no conflict of interests to disclose.

Daniel Duprez, MD, PhD, Professor of Medicine, Donald and Patricia Garofalo Chair in Preventive Cardiology, Professor of Epidemiology and Community Health, University of Minnesota
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Shareholder & Employee: PF Pharmaceutical Development, LLC

Bo Fernhall, PhD, Dean, College of Applied Health Sciences, Professor of Kinesiology, University of Illinois at Chicago, has no conflicts of interest.

Bradley S. Fleenor, PhD, Assistant Professor, Department of Kinesiology and Health Promotion, SAHA Cardiovascular Research Center, University of Kentucky , has no conflict of interests.

Edward G. Lakatta, MD, Director of the Laboratory of Cardiovascular Science, National Institute on Aging, National Institutes of Health, has no conflict of interests.

Gary F. Mitchell, MD, President of Cardiovascular Engineering Inc.
Consultant: Novartis, Merck
Grant/Research Support: NIH, AHA
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Wilmer W. Nichols, PhD, Director of Basic Cardiovascular Research, Division of Cardiology, Department of Medicine, University of Florida
Consultant: Millar Instruments

Gary L. Pierce, PhD, Assistant Professor, Department of Health and Human Physiology, University of Iowa
Grant/Research Support: American Heart Association, NIH

Leopoldo Raij, MD, Director, Hypertension and Nephrology Division, Vice Chair, Vascular Biology Institute, University of Miami School of Medicine and Chief, Nephrology-Hypertension Section, Miami Veterans Affairs Medial Center, has no conflicts of interest.

Norman Stockbridge, MD, PhD, Director, Division of Cardiovascular and Renal Products, Office of Drug Evaluation, U.S. Food and Drug Administration, has no conflicts of interest.

Hirofumi Tanaka, PhD, Professor and Director, Cardiovascular Aging Research Laboratory , Department of Kinesiology & Health Education at the University of Texas at Austin
Grant/Research Support: Dairy Research Institute

Raymond R. Townsend, MD, Chief, Hypertension, Department of Medicine, University of Pennsylvania Medical School
Consultant: Medtronic, Janssen Pharmaceuticals
Grant/Research Support: NIH

Elaine M. Urbina, MD, MS, Professor of Pediatrics (Cardiology), Director of Preventive Cardiology, Department of Pediatrics , Cincinnati Children's Hospital Medical Center
Consultant: Midmark Medical and Unilever
Grant/Research Support: NIH, AtCor Medical

5:00 - 5:30 pm Clinical/Translational Lecture in Hypertension—Julio A. Chirinos, MD, PhD

Are There Differences in the Way Current Drugs Affect Central Arterial Hemodynamics and Blood Pressure as Compared with Brachial Blood Pressure?

Vasoactive drugs have different effects on central vs. peripheral pressures. Available data indicates that beta blockers do not reduce central systolic pressure as effectively as calcium-channel blockers, ACE inhibitors and/or diuretics. The combination of amlodipine/perindopril was shown to be significantly more effective in the reduction of central BP as compared to atenolol/bendroflumethiazide, despite similar brachial BP reduction. The most notable class of drugs that exert profound effects on central hemodynamics are organic nitrates. These drugs have important effects on the central pressure waveform, reducing late systolic hypertension and pressure augmentation. Before central pressures can be implemented as a therapeutic target in clinical practice, randomized controlled trials testing the potential benefit of selectively lowering central pressures, on clinically meaningful outcomes, are required.

6:00 - 6:30 pm Clinical/Translational Lecture in Hypertension—Norman Stockbridge, MD, PhD

Validation of New Parameters into Validated (or Surrogate) Biomarkers and What Studies Are Needed to Enhance Their Validation?

Surrogacy status may come, but it is seldom bidden.

9:30-10:00 pm Historical Perspectives Lecture—Wilmer W. Nichols, PhD

History of Central Hemodynamics and Wave Reflection: Translation from Early Experimental Studies to Contemporary Clinical Medicine

The external load (afterload) the left ventricle (LV) pumps against during contraction and blood ejection is composed of a steady component (associated with arteriolar caliber) and a pulsatile component (associated with aortic stiffness and wave reflection from the lower body). The steady load component can be obtained from any arterial pressure waveform (central or peripheral) while the pulsatile component can only be obtained from the central aortic pressure waveform because of systolic (and pulse) pressure amplification from central to peripheral measuring sites.

Arterial blood pressure was first measured (invasively) in animals in 1733. Over the next 230 years invasive techniques were developed that could accurately record the central aortic pressure waveform in both animals and humans. These invasive measurements were necessary for information gathering, equation development and model testing but were limited because they could not be used in large groups of patients. The application of Fourier analysis to invasively measured arterial pressure waveforms over the next 30 years led to the development of a generalized transfer function that allowed the accurate calculation of the central aortic pressure waveform from the non-invasively measured radial artery pressure waveform. These non-invasive measurements of central hemodynamics and LV external load (steady and pulsatile components) can be used to collect and evaluate data in large scale studies, clinical trials and clinical practice.

TAKEAWAY MESSAGES - SATURDAY, SEPTEMBER 7, 2013

8:30 - 9:00 am Basic Science Lecture—Edward G. Lakatta, MD

Molecular Mechanisms of Aortic Deterioration with Aging and Disease

Interaction among aging, disease, genetics, lifestyle and environment define reality.

As life expectancy increases, a systematic approach is needed to ensure that we have a healthy old age.

A mandate to future biomedical researchers and health care physicians: PREVENT the age-associated EPIDEMIC of cardiovascular diseases, i.e. hypertension, atherosclerosis, and diabetes, that lead to chronic heart, brain and kidney failure by retarding cardiovascular aging.

9:30-10:00 am Clinical Translational Lectures in Aging, Disease and Youth—Stella Daskalopoulou, MD, MSc, DIC, PhD

Central Pulsatile Hemodynamics, Arterial Stiffness and Target Organ Damage

Examples of clinical applications of measurements of arterial stiffness and hemodynamics will be provided, with particular emphasis on certain conditions, such as smoking, pre-eclampsia and obstructive sleep apnea. Smoking is well known to affect arterial stiffness and hemodynamics at rest. Recent results have demonstrated that even light smoking in young, healthy individuals affects arterial stiffness and hemodynamics. The harmful effects were revealed after acute physical stress, while resting values were not significantly different from non-smokers. The "arterial stress test" will be introduced. Arterial stiffness and hemodynamics as potential predictors of pre-eclampsia will be presented, and the available evidence will be summarized. Furthermore, clinical evidence will be discussed on the association between arterial stiffness and hemodynamics and obstructive sleep apnea, as well as the beneficial effect of continuous positive airway pressure (CPAP).

10:30-11:00 am Clinical Translational Lectures in Aging, Disease and Youth—Bradley S. Fleenor, PhD

Large Artery Stiffness with Aging and Disease: From Molecular Mechanisms to Novel Translational Interventions in Humans

Periaortic adipose tissue contributes to arterial stiffness in animal models of aging and disease. Whether this fat depot has a unique secretion profile and is of clinical relevance remains unknown. However, novel nutraceutical interventions attenuate arterial stiffness in these conditions and further investigation in humans is warranted.

11:00-11:30 am Clinical Translational Lectures in Aging, Disease and Youth—Elaine M. Urbina, MD, MS

Utility of Arterial Stiffness as a Marker of Clinical Cardiovascular Risk in Youth

Measuring arterial stiffness in youth is a useful method to more accurately assess future risk for CV events.

11:30 am-12:00 pm Clinical Translational Lectures in Aging, Disease and Youth—Hirofumi Tanaka, PhD

Effect of Lifestyle Interventions on Central Arterial Function in Aging and Disease

For most risk factors for cardiovascular disease (CVD), the first-line approach for prevention and treatment for development of CVD is lifestyle modifications. Given the role of arterial stiffness as a critical precursor of CVD, it is important to recognize the effects of lifestyle modifications for the prevention and treatment of arterial stiffening. Available evidence indicates that lifestyle modifications, in particular aerobic exercise and sodium restriction, appear to be clinically efficacious therapeutic interventions for preventing and treating arterial stiffening. This lecture highlights the previous intervention studies that focused on lifestyle modifications on arterial stiffening so that the attendees can make informed decisions regarding what lifestyle changes would be beneficial as a destiffening therapy.

2:30 - 3:00 pm Debate/Counterpoint Presentation—*Gary F. Mitchell, MD*

Central Systolic Blood Pressure and Pulse Pressure IS NOT a Better Predictor of CVD Risk than Brachial Systolic and Pulse Pressure

At the end of this presentation, attendees will understand available data regarding relations between central as compared to peripheral pulse pressure and cardiovascular disease events. Attendees will understand that antihypertensive therapy and cardiovascular disease prevention should be focused primarily on the major distinction between abnormalities in mean versus pulse pressure rather than the relatively minor distinction between central and peripheral pulse pressure.

FRIDAY, SEPTEMBER 6, 2013

3:00 - 4:00 pm **Opening Reception (Exhibit Hall)**

4:00 - 4:10 pm **Welcome Remarks**

Bo Fernhall, PhD, University of Illinois at Chicago

Gary L. Pierce, PhD, University of Iowa

4:10 - 4:20 pm **President's Opening Statement**

Raymond R. Townsend, MD, University of Pennsylvania

4:20 - 5:00 pm **Opening Plenary Lecture**

Do Novel Central Arterial Hemodynamic Parameters Improve Over Conventional Brachial BP as Predictor of CV Outcomes?

Raymond R. Townsend, MD, University of Pennsylvania

5:00 - 6:30 pm **Clinical/Translational Lectures in Hypertension**

5:00 pm — Are There Differences in the Way Current Drugs Affect Central Arterial Hemodynamics and Blood Pressure as Compared with Brachial Blood Pressure?
Julio A. Chirinos, MD, PhD, University of Pennsylvania School of Medicine

5:30 pm — Prime Time to Target Central Arterial Hemodynamics beyond Brachial BP, Which Drugs and Devices are Available?
Daniel Duprez, MD, PhD, University of Minnesota

6:00 pm — Validation of New Parameters into Validated (or Surrogate) Biomarkers and What Studies Are Needed to Enhance Their Validation?
Norman Stockbridge, MD, PhD, U.S. Food and Drug Administration

6:30 - 7:45 pm **Oral Communications**

6:30 pm
OR-01 — Resistance Exercise-Induced Increases in Carotid Artery Stiffness Do Not Affect Cerebral Blood Flow Pulsatility

Kevin S. Heffernan, Wesley K. Lefferts, Jacqueline A. Augustine; Department of Exercise Science, Syracuse University, Syracuse, NY

6:45 pm
OR-02 — Circulating AGE/RAGE Biomarkers and Aortic Structure and Function in Older Adults

Tina E. Brinkley, PhD[1], Xiaoyan Leng, PhD[2], Dalane W. Kitzman, MD[3], Jingzhong Ding, PhD[1], Barbara J. Nicklas, PhD[1], Stephen B. Kritchevsky, PhD[1], W. Gregory Hundley,MD[3]; [1]Department of Internal Medicine, Section on Gerontology and Geriatric Medicine, [2]Department of Biostatistical Sciences, Division of Public Health Sciences, [3]Department of Internal Medicine, Section on Cardiology, Wake Forest School of Medicine, Winston-Salem, NC

(Continued on page 12)

AGENDA—SEPTEMBER 6 & 7, 2013

7:00 pm OR-03	A Monoclonal Antibody to an Endogenous Steroidal Na/K-Atpase Ligand, Marinobufagenin, Reverses Expression of Profibrotic Genes in Aged Rats *Olga V. Fedorova*,[1] Victoria Shilova,[1] Valentina Zernetkina,[1] Yongqing Zhang,[1] Courtney A. Marshall,[1] Elin Lehrmann,[1] Kevin G. Becker,[1] Edward G. Lakatta,[1] Alexei Y. Bagrov[1]; [1]National Institute on Aging, NIH, Baltimore, USA
7:15 pm OR-04	Racial Differences in Pressure Responses Following Peak Exercise: Insight from Pressure Wave Separation Analysis *Rosenberg, A.J.*, Wee, S. O., Ranadive, S., Lane, A., Kappus, R., & Fernhall, B.; University of Illinois at Chicago, Chicago, IL
7:30 pm OR-05	Differential Association of the Forward and Reflected Pulse Waves with Aortic Diameter in a Community-Dwelling Population of Normotensive and Untreated Hypertensive Men and Women *Majd Alghatrif, MD**[1,2,3] Marco Canepa, MD*[1,2,4], James B. Strait, MD, PhD[1,2], Hao-Min Cheng, MD[5,6], Shao-Yuan Chuang, PhD[7], Chen-Huan Chen, MD[5,6],Claudio Brunelli, MD[3], Luigi Ferrucci, MD, PhD[2], Edward G. Lakatta, MD[1]; [1] Laboratory of Cardiovascular Sciences, Human Cardiovascular Studies Unit, National Institute on Aging, NIH, Baltimore, MD, USA. [2] Longitudinal Studies Section, Translational Gerontology Branch, National Institute on Aging, NIH, Baltimore, MD, USA. [3] Department of Medicine, Johns Hopkins Bayview Medical Center, Johns Hopkins School of Medicine, Baltimore, MD, USA. [4] Division of Cardiology, Research Center of Cardiovascular Biology, University of Genova, Genova, Italy. [5] Department of Medical Research And Education, Taipei Veterans General Hospital, Taipei, Taiwan. [6] Department of Medicine, National Yang-Ming University, Taipei, Taiwan. [7] Division of Preventive Medicine and Health Service, Research Institute of Population Health Sciences, National Health Research Institutes, Miaoli, Taiwan.
8:00-10:00 pm	**Dinner and Historical Perspectives Lecture**
8:00 - 9:20 pm	Dinner in United AB Room
9:30 pm	History of Central Hemodynamics and Wave Reflection: Translation from Early Experimental Studies to Contemporary Clinical Medicine *Wilmer W. Nichols, PhD, University of Florida*

SATURDAY, SEPTEMBER 7, 2013

7:30 - 8:15 am	**Breakfast (Exhibit Hall)**
8:30 - 9:00 am	**BASIC SCIENCE LECTURE**
	Molecular Mechanisms of Aortic Deterioration with Aging and Disease *Edward G. Lakatta, MD, National Institute on Aging*
9:00 - 9:30 am	**Coffee Break (Exhibit Hall)**

9:30 -12:00 pm **Clinical Translational Lectures in Aging, Disease and Youth**

9:30 am — Central Pulsatile Hemodynamics, Arterial Stiffness and Target Organ Damage
Stella Daskalopoulou, MD, MSc, DIC, PhD, McGill University Health Center

10:00 am — Hypertension, Endothelium and Anti-Hypertensives: Insights from Experimental Hypertension
Leopoldo Raij, MD, University of Miami School of Medicine

10:30 am — Large Artery Stiffness with Aging and Disease: From Molecular Mechanisms to Novel Translational Interventions in Humans
Bradley S. Fleenor, PhD, University of Kentucky College of Medicine

11:00 am — Utility of Arterial Stiffness as a Marker of Clinical Cardiovascular Risk in Youth
Elaine M. Urbina, MD, MS, Cincinnati Children's Hospital Medical Center

11:30 am — Effect of Lifestyle Interventions on Central Arterial Function in Aging and Disease
Hirofumi Tanaka, PhD, The University of Texas at Austin

12:00-12:20 pm **Reimbursement Issues for Arterial Stiffness and Central Blood Pressure Measurements**
Mr. Michael Beebe, ADVI

12:20 - 1:00 pm **Platinum Sponsor Presentations**

12:20 pm — Assessment of Central Blood Pressure – Single, Sequential and Ambulatory (Sponsored by I.E.M. GmbH)
Mr. Achim Schwarz, IEM GmbH

12:40 pm — SphygmoCor XCEL and the Use of Pulse Waveform Analysis in the Management of Blood Pressure (Sponsored by AtCor Medical, Inc.)
Dean C. Winter, PhD, AtCor Medical, Inc.

1:00 - 2:30 pm **Poster Session and Lunch**
Lunch served in Exhibit Area
Presenters at their Posters 1:30 - 2:30 pm

2:30 - 3:00 pm **Debate/Counterpoint Presentation**

Central Systolic Blood Pressure and Pulse Pressure **IS** a Better Predictor of CVD Risk than Brachial Systolic and Pulse Pressure
Richard B. Devereux, MD, Weill Cornell Medical College

Central Systolic Blood Pressure and Pulse Pressure **IS NOT** a Better Predictor of CVD Risk than Brachial Systolic and Pulse Pressure
Gary F. Mitchell, MD, Cardiovascular Engineering, Inc.

3:00 pm **Awards Presentations**

3:10 pm **Conclusions from the Meeting and Future Direction of NAA**

POSTERS

PO-01 **EFFECTS OF GENDER AND RACE ON INTERLEUKIN-6 PRODUCTION IN RESPONSE TO CRP IN HUVECS**

Chenyi Ling, Jan Kretzschmar, Heather Grimm, Michael Brown; Department of Kinesiology and Nutrition, University of Illinois at Chicago, Chicago, IL

PO-02 **ENDOTHELIAL MICROPARTICLES SHOW A BLUNTED EXERCISE RESPONSE IN AFRICAN AMERICANS WITH HIGH LEVELS OF INFLAMMATION**

Kretzschmar, Jan[1,2]; Babbitt, Dianne M.[1]; Diaz, Keith M.[1,3]; Feairheller, Deborah L.[1,4]; Sturgeon, Kathleen M.[1,5]; Perkins, Amanda M.[1,6]; Veerabhadrappa, Praveen[1,7]; Williamson, Sheara T.[1,8]; Ling, Chenyi[1,2]; Lee, Hojun[1]; Grimm, Heather[1,2]; Thakkar, Sunny R.[1]; Crabbe, Deborah L.[9]; Kashem, Mohammed A.[1,9]; Brown, Michael D.[1,2]; Temple University, Philadelphia, PA, USA[1], University of Illinois at Chicago, Chicago, IL, USA[2], Columbia University Medical Center, New York, NY, USA[3], Ursinus College, Collegeville, PA, USA[4], University of Pennsylvania, Philadelphia, PA, USA[5], Missouri State University, Springfield, MO, USA[6], Shippensburg University, Shippensburg, PA, USA[7], Notre Dame University of Maryland, Baltimore, MA, USA[8], School of Medicine, Temple University, Philadelphia, PA, USA[9]

PO-03 **THE EFFECT OF AEROBIC EXERCISE TRAINING ON ENDOTHELIAL HEALTH STATUS IN PRE- AND POST-MENOPAUSAL AFRICAN AMERICAN WOMEN**

Heather Grimm[1], Jan Kretzschmar[1], Sunny Thakkar[5], Chenyi Ling[1], Keith M. Diaz[2], Kathleen Sturgeon[4], Deborah L. Feairheller3, Amanda Perkins[7], Hojun Lee[5], Dianne Babbitt[5], Sheara T. Williamson[5], Michael D. Brown[1]; [1]University of Illinois at Chicago, IL, [2]Columbia University, New York, NY, [3]Health Technology Assessment Group, Plymouth Meeting, PA, [4]University of Pennsylvania, Philadelphia, PA, [5]Temple University, Philadelphia, PA, [6]Missouri State University

PO-04 **RACIAL DIFFERENCES IN BLOOD FLOW DURING ACUTE DYNAMIC EXERCISE**

Rebecca M. Kappus; University of Illinois at Chicago, Chicago, IL

PO-05 **THE HEMODYNAMIC EFFECTS ON BRACHIAL AND CENTRAL BLOOD PRESSURE WITH THE USE OF 5-HOUR ENERGY® DRINK ON HEALTHY ADULTS**

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PO-06 **SEX DIFFERENCES IN POST EXERCISE HYPOTENSION**

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PO-07 **DO CENTRAL ARTERIAL HEMODYNAMICS CONTRIBUTE TO WALKING PERFORMANCE IN OLDER ADULTS?**

Gonzales, J.U.[1], Shephard, J. [1], Defferari, E. [1], Proctor, D.N.[2]; Texas Tech University[1], Pennsylvania State University[2]

PO-08 **EFFECTS OF CAFFEINE SUPPLEMENTATION ON THE RELATIONSHIP BETWEEN TOTAL ARTERIAL COMPLIANCE AND HEMODYNAMIC PARAMETERS DURING EXERCISE RECOVERY**

Bunsawat, K., Rosenberg, A.J., Wee, S.O., Kappus, R.M., Hultgren, K., Fernhall, B., & Baynard, T.; University of Illinois at Chicago, Chicago, IL

PO-09 **COMPARISON OF STRUCTURAL AND FUNCTIONAL VASCULAR INDICES BETWEEN AMBULATORY AND NON-AMBULATORY ADULTS WITH CEREBRAL PALSY**

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PO-10 **SPIRONOLACTONE REVERSES PULSE WAVE VELOCITY IN PATIENTS WITH RESISTANT HYPERTENSION**

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PO-11 **AORTIC FIBROSIS INDUCED BY A STEROIDAL ENDOGENOUS NA/K-ATPASE INHIBITOR IS REVERSED BY ALDOSTERONE ANTAGONIST CANRENONE**

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STELLA S. DASKALOPOULOU, MD, MSc, DIC, PhD is a tenured Associate Professor of Medicine, Department of Medicine at McGill University.

Dr. Daskalopoulou is an Internist with a special interest in Vascular Medicine. She obtained her MD and PhD degrees at the University of Athens. Following further studies for her MSc in Vascular Medicine and Technology at Imperial College London and a second PhD in Vascular Medicine at Imperial College London, and further clinical training in vascular disease prevention in London, she took up a postdoctoral position at McGill University where she studied Cardiovascular Evaluation and Outcomes, and Pharmacoepidemiology.

Dr. Daskalopoulou established the Vascular Health Unit at the McGill University Health Centre (clinical and wet-bench labs), where she conducts her vascular research projects, including arterial stiffness studies in subjects with different cardiovascular risk factors, such as smoking, hypertension, diabetes, and dyslipidemia. The theme of her research program is the identification of early markers of vascular impairment and maintenance of vascular health. Her intention is to integrate biomedical technology into an innovative cardiovascular research

(Continued on page 16)

program aimed at unraveling the arteriosclerotic and atherosclerotic process, and identifying early markers of severity, progression of vascular disease, and efficacy of treatment. Dr. Daskalopoulou has numerous publications and holds several research grants and personal awards.

RICHARD B. DEVEREUX, MD is a Professor of Medicine at Weill Cornell Medical College and Director of the Adult Echocardiography Laboratory at the Weill Cornell Center of the New York Presbyterian Hospital.

He has a long-standing clinical and research interest in cardiovascular connective tissue diseases, and has served as Chair of the Professional Advisory Committee of the National Marfan Foundation. In addition to expertise in all aspects of clinical echocardiography, Dr. Devereux conducts extensive research using cardiac ultrasound and other techniques to improve understanding of hypertensive heart disease, valvular heart diseases, cardiac effects of obesity diabetes, the prevalence and mechanisms of cardiovascular diseases in population-based samples and identify genes linked to and potentially responsible for a spectrum of cardiovascular diseases. Dr. Devereux earned his MD degree from the University of Pennsylvania School of Medicine.

DANIEL DUPREZ, MD, PhD is a Professor of Medicine, Professor of Epidemiology and Community Health, and recipient of the Donald and Patricia Garofalo Chair in Preventive Cardiology at the University of Minnesota.

From research studies to clinical practice, Dr. Duprez's cardiology interests span the prevention of cardiovascular disease in the general population to its occurrence in patients with organ transplants and patients with HIV. Dr. Duprez is the author of more than 250 articles and 55 book chapters, and more than 1000 lectures nationally and internationally on a wide range of cardiology topics.

An internationally-renowned expert in vascular biology, Dr. Duprez's current research involves arterial mechanics and stiffness, biomarkers for early cardiovascular disease, lipoproteins, neurohormonal factors and hypertension, HIV and cardiovascular disease, and clinical-trials events evaluation.

BRADLEY S. FLEENOR, PhD is an Assistant Professor, Department of Kinesiology and Health Promotion at the University of Kentucky.

Dr. Fleenor received his Ph.D. in Biomedical Sciences from the University of Missouri. Thereafter, he completed a postdoctoral fellowship in translational cardiovascular physiology in Dr. Doug Seals' Integrative Physiology of Aging Laboratory at the University of Colorado – Boulder.

Dr. Fleenor currently is an assistant professor at the University of Kentucky with appointments in Kinesiology and Health Promotion and the Graduate Center for Nutritional Sciences. His newly established laboratory studies the mechanisms by which novel translatable interventions improve cardiovascular physiology and pathology.

EDWARD G. LAKATTA, MD is the founder and Director of the Laboratory of Cardiovascular Science, National Institute on Aging, National Institutes of Health. He also holds adjunct appointments as Professor, Department of Physiology, University of Maryland School of Medicine, and Professor, Cardiology Division, Johns Hopkins School of Medicine.

He has made a sustained 30-plus-year commitment to a broad-based research career. His studies range from molecules to humans, including translation of novel findings into the clinical realm. The overall goals of his research program are 1) to identify age associated changes that occur within the cardiovascular system and to determine the mechanisms for these changes; 2) to determine how aging of the heart and vasculature interacts with chronic disease states to enhance the risk for CV diseases in older persons; 3) to study basic mechanisms in excitation-contraction coupling and how these are modulated by surface receptor signaling pathways in cardiac cells; 4) to elucidate mechanisms of pacemaker activity in sinoatrial nodal cells; 5) to elucidate mechanisms that govern cardiac and vascular cell survival; 6) to establish the potentials and limitations of new therapeutic approaches such as changes in lifestyle, novel pharmacologic agents or gene or stem cell transfer techniques in aging or disease states.

Dr. Lakatta is recognized both nationally and internationally as an expert in cardiovascular research. He has authored over 450 original publications in top peer-reviewed cardiovascular journals, written over 250 invited reviews/book chapters, and delivered over 450 invited lectures. He is a member of multiple scholarly societies and journal editorial boards. Based upon his accomplishments, Dr. Lakatta has received numerous awards, among which are the Allied Signal Achievement Award in Aging, the Novartis Prize in Gerontology, the Irving Wright Award of Distinction of the American Federation for Aging Research (AFAR), the Frank J. O'Hara Alumni Award from the University of Scranton, and the Distinguished Leader Award of the International Society of Heart Research (ISHR).

GARY F. MITCHELL, MD is the founder and President of Cardiovascular Engineering, Inc.

Dr. Mitchell is a cardiologist and internationally acknowledged leader in the field of vascular stiffness and pulsatile hemodynamics. He received his medical degree from Washington University in St. Louis and did his medicine and cardiology training at Brigham and Women's Hospital in Boston, where he served as a staff cardiologist until 1998.

Dr. Mitchell left the Brigham in 1998 to become founder and president of Cardiovascular Engineering, Inc., which is an NIH-funded small business that designs and develops innovative devices for measuring arterial stiffness and uses those devices to explore the genetic and environmental correlates of arterial stiffness and the role that arterial stiffness plays in the pathogenesis of hypertension and target organ damage.

(Continued on page 17)

He joined the Framingham Heart Study as a Framingham Investigator in 1999 and became a collaborator on the AGES-Reykjavik study in 2006 and the Jackson Heart Study in 2010. Using devices designed and built by Cardiovascular Engineering, Dr. Mitchell has performed detailed assessments of arterial stiffness and pulsatile hemodynamics in more than 20,000 research participants, including participants in all 3 generations of the Framingham Heart Study as well as participants in the AGES-Reykjavik study, the REFINE study, and the Jackson Heart Study.

WILMER W. NICHOLS, PhD is an Adjunct Professor of Medicine in the Department of Medicine in the College of Medicine and in the Division of Cardiovascular Medicine at the University of Florida.

He served as the Director of Basic Cardiovascular Research, Division of Cardiology, Department of Medicine at the University of Florida for nearly 30 years. Dr. Nichols, who earned his PhD degree from the University of Alabama Medical School at Birmingham, Alabama, co-authored the book, *McDonald's Blood Flow in Arteries: Theoretic, Experimental and Clinical Principles* and has authored more than 200 articles that have appeared in numerous journals.

Dr. Nichols' major interests are related to pulsatile pressure and flow in arteries. His investigations over the years have dealt with sophisticated measurements of aortic impedance and left ventricular responses to acute changes in afterload in man. He is also interested in Hemodynamics, Cardiovascular Aging, Hypertension, Exercise, and Cardiovascular Disease in Women.

LEOPOLDO RAIJ, MD is a Professor of Medicine, Director of the Hypertension Nephrology/Hypertension Division, and Vice Chair of the Vascular Biology Institute at the University of Miami School of Medicine and as of June 2008 – Vice Chair of the Scientific Advisory Committee at the University of Miami Medical School. He is also the Chief of the Nephrology-Hypertension Section of the Veterans Affairs Medical Center in Miami

Dr. Raij received his MD from Universidad Nacional del Litoral (UNL) in Argentina, and completed his internship and residency at University Hospital, UNL. He has been a member of the Merit Review Board of the Veterans Administration, the Scientific Research Board of the American Society of Hypertension, the National Kidney Foundation Council on Hypertension, and the Scientific Advisory Board of the National Kidney Foundation.

(Continued on page 18)

Dr. Raij is a member of the editorial boards of Hypertension and Kidney International, is Associate Editor of the Journal of the American Society of Hypertension, and was a member of the Executive Committee of the Council on the Kidney in Cardiovascular Disease of the American Heart Association from July 1994 to 1996.

Dr. Raij currently serves on the Board of Directors of the American Society of Hypertension and is CO-PI of the NIH funded, Miami component of the Hispanic Community Health Study. A recipient of the American Heart Association Lewis Dahl Award, Dr. Raij has published over 180 articles in peer-reviewed publications.

ACHIM SCHWARZ is a Key Account Manager at I.E.M. GmbH.

Mr. Schwarz dedicates his professional career to the application of innovative technologies in cardio-vascular research. Among other activities, his special fields of interest relate to the support of studies in the fields of non-invasive capture of central hemodynamic indices, arterial stiffness evaluation, and central blood pressure measurement. Beyond his support activities, Mr. Schwarz has provided consultancy services for the application of remote monitoring systems in not only a research environment, but also daily medical practice.

I.E.M. GmbH is a Germany based enterprise, specialized in the combination of services in screening, diagnostics, and follow-up management of arterial hypertension. Mr. Schwarz is in charge of the I.E.M. scientific research program.

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HIROFUMI TANAKA, PhD is a Professor and the Director of the Cardiovascular Aging Research Laboratory at the University of Texas at Austin.

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Dr. Tanaka's research interests revolve around preventive cardiology and preventive gerontology, with primary research interests involving lifestyle modifications, aging, and vascular function.

He has published over 180 research articles in journals such as *Circulation* and the *Journal of Physiology*. He is an elected fellow of various professional organizations including the American Heart Association, the American College of Sports Medicine, and the Gerontological Society of America.

RAYMOND R. TOWNSEND, MD is a Professor of Medicine in the Renal Division and Associate Director of the Clinical & Translational Research Center (CTSA) at the University of Pennsylvania. He is also the Director of the Penn Hypertension Program.

Dr. Townsend received his MD degree from Hahnemann University Hospital. His formal certifications are in internal medicine (ABIM), nephrology (ABIM), clinical pharmacology (ASCP) and hypertension (ASH). He is a Fellow of the American Heart Association and a Fellow of the Council for High Blood Pressure Research. An empanelled member of the NHLBI Joint National Committee (JNC8), Dr. Townsend is the Principal Investigator of the Pulse Wave Velocity in CKD ancillary project in the Chronic Renal Insufficiency Cohort Study (CRIC), and the Principal Investigator of the Penn Clinical Center in the CRIC Study.

Dr. Townsend's principal research interests focus on the role of hypertension and in particular mechanisms of kidney damage that are related to pulse wave travel and pulse wave reflection in the circulation; and in the role of metabolism, specifically the linkages between insulin resistance and kidney disease progression in people with chronic kidney disease. These two areas (arterial stiffness and metabolism) link the role of hemodynamics (as reflected in both blood pressure and vascular stiffness) to a variety of outcomes in CKD including heart failure, cognitive function changes, retinopathy, CKD progression, and vascular calcification.

ELAINE M. URBINA, MD, MS is the Director of Preventive Cardiology and Professor of Pediatrics (Cardiology) at Cincinnati Children's Hospital Medical Center.

As Director of Preventive Cardiology at Cincinnati Children's Hospital Medical Center, Dr. Urbina's clinical activities and industry sponsored grants focus on prevention (obesity, hypertension and dyslipidemias) while her research grants (AHA, NIH) and masters in epidemiology training concentrate on new non-invasive methods of assessing atherosclerotic CV target organ damage in youth related to CV risk factors especially those that cluster with obesity.

Dr. Urbina has over 20 years of experience in non-invasive imaging of CV structure and function in large epidemiologic studies such as the Bogalusa Heart Study. She is a PI of a National Institutes of Health (NHLBI R01) following the cardiac and vascular effects of obesity and type 2 diabetes on adolescents and a member of the International Childhood CV Cohorts Consortium that will be following Bogalusa, Muscatine, Young Finns and other cohorts that collect CV risk factor data in children over 40 years ago as the participants are now entering middle age. Dr. Urbina also supplies training and/or directs the Vascular Imaging Core for many multi-center pediatric studies including TODAY2 (type 2 diabetes), SEARCH 3 (type 1 diabetes), CKiD (chronic kidney disease) and other grants funded by NIH's Pediatric Heart Network.

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RESISTANCE EXERCISE-INDUCED INCREASES IN CAROTID ARTERY STIFFNESS DO NOT AFFECT CEREBRAL BLOOD FLOW PULSATILITY

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Introduction: Cross-sectional studies have identified arterial stiffness as an important correlate of cerebral blood flow pulsatility. Whether an acute increase in arterial stiffness affects cerebral pulsatility remains unknown. Central artery stiffness acutely increases following a bout of resistance exercise (RE).

Objectives: To investigate the effects of acute RE on carotid artery stiffness and cerebrovascular pulsatility.

Methods: Eighteen healthy men (age 22 ± 1 yr; BMI 23.7 ± 0.5 $kg \cdot m^{-2}$) underwent acute RE and a time control condition (seated rest) on two separate days in a randomized order. Middle cerebral artery (MCA) blood flow velocity pulsatility index (PI) was measured with transcranial Doppler. Common carotid artery (CCA) beta stiffness index (β), elastic modulus (Ep) and PI were assessed using Doppler ultrasound. Both wave intensity analysis (WIA) and wave separation analysis (WSA) were used to derive measures of forward (WIA W_1 and WSA Pf) and backward (WIA Negative Area and WSA Pb) travelling pressure waves to gain insight into origins of pressure pulsatility.

Results: CCA β, Ep and pulse pressure significantly increased post-RE ($p < 0.05$). There were significant increases post-RE in measures of forward wave pressure (W_1 and Pf, $p<0.05$) with no changes in measures of backward/ reflected wave pressure (lnNA and Pb, $p>0.05$). There were no changes in CCA PI or MCA PI following acute RE ($p>0.05$).

Conclusions: Acute RE increases CCA stiffness and pressure pulsatility without affecting CCA or MCA flow pulsatility. Increases in pressure pulsatility are due to increases in forward wave pressure and not pressure from wave reflections.

Table 1. Vascular and hemodynamic response to acute resistance exercise.

Variable	Condition	Baseline	10-min Post	20-min Post	30-min Post	Interaction
Brachial PP, mmHg	Control RE	48±1 49±2	48±2 66±3[a,b]	46±2 61±2[a,b]	46±2 59±2[a,b]	<0.001
CCA PP, mmHg	Control RE	42±2 43±2	40±2 58±4[a,b]	40±2 53±3[a,b]	40±2 54±3[a,b]	<0.001
CCA W_1, $mmHg \cdot m \cdot sec^{-3}$	Control RE	9.4±0.8 9.3±1.0	10.6±1.3 16.6±2.0[a,b]	9.7±1.2 13.5±1.5[a,b]	9.1±0.8 11.7±1.3[a]	0.015
CCA lnNA, $mmHg \cdot m \cdot sec^{-2}$	Control RE	3.3±0.5 3.9±0.4	3.6±0.3 4.9±0.5	3.5±0.2 4.2±0.4	3.3±0.3 3.2±0.5	0.160
CCA β, [-]	Control RE	3.9±0.4 3.9±0.4	3.6±0.3 5.3±0.4[a,b]	3.8±0.2 4.8±0.4[a,b]	3.5±0.2 5.0±0.3[a,b]	0.025
CCA Ep, kPa	Control RE	49±5 47±4	44±3 65±5[a,b]	45±3 56±4[a,b]	42±2 59±4[a,b]	0.032
CCA Pf, mmHg	Control RE	40±2 41±3	38±3 58±4[a,b]	44±5 47±3[a,b]	39±2 49±4[a,b]	<0.001
CCA Pb, mmHg	Control RE	16±1 15±1	15±1 16±1	15±1 14±1	16±1 16±1	0.649
CCA PI, [-]	Control RE	2.1±0.1 2.0±0.1	2.1±0.1 2.2±0.1	2.1±0.1 2.0±0.1	2.1±0.1 2.0±0.1	0.094
MCA PI, [-]	Control RE	0.9±0.1 0.9±0.1	0.9±0.1 0.9±0.1	0.9±0.1 0.8±0.1	0.8±0.1 0.8±0.1	0.325

[a] Significantly different from within condition/RE baseline ($p<0.05$)
[b] Significantly different between condition at the same time point ($p<0.05$)
aU, arbitrary units; PP, pulse pressure; CCA, common carotid artery; MCA, middle cerebral artery; Pf, forward wave pressure; Pb, backward wave pressure; NA, negative area; Ep, elastic modulus; β, beta-stiffness index, PI, pulsatility index. All data are mean ± SEM.

CIRCULATING AGE/RAGE BIOMARKERS AND AORTIC STRUCTURE AND FUNCTION IN OLDER ADULTS

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Introduction: Advanced glycation endproducts (AGEs) and the AGE receptor (RAGE) have been implicated as mediators of arterial stiffness in aging and diabetes. However, associations with subclinical measures of vascular health are not well characterized.

Objective: We determined whether circulating levels of the major AGE carboxymethyl-lysine (CML) and soluble receptors sRAGE and esRAGE are associated with aortic structure and function in older adults at high cardiac risk.

Methods: Aortic arch pulse wave velocity (PWV) and periaortic fat, distensibility, and wall thickness of the ascending aorta (AA) and descending aorta (DA) were measured by MRI in 231 adults (mean age, 69±8 years; 54% female; 78% white) with hypertension (94%), coronary disease (24%), or diabetes (38%). Linear regression models were stratified by diabetes status and adjusted for age, gender, race/ethnicity, coronary disease, hypertension, and BMI.

Results: Diabetics had lower sRAGE (1396±75 vs. 1724±58 pg/ml, p=0.007), but similar AGE-CML (834±24 vs. 878±18, p=0.15) and esRAGE (0.75±0.04 vs. 0.83±0.03, p=0.16) compared to non-diabetics. Lower esRAGE was modestly associated with higher DA fat (β=-326±187, p=0.08) and DA wall thickness (β=-0.278±0.188, p=0.14) in diabetics. Among non-diabetics, modest associations were found between sRAGE and DA distensibility (β=-3.4±2.2x10^{-7}, p=0.13) and between AGE-CML and AA distensibility (β=-1.1±0.07x10^{-7}, p=0.13). There were no significant associations between AGE/RAGE and PWV in either group.

Conclusions: Contrary to our hypothesis, circulating AGE/RAGE biomarkers are not associated with aortic structure or function in high-risk older adults. This suggests that the relationship of AGE/RAGE with adverse cardiovascular events may be mediated by pathways other than increased arterial stiffness.

A MONOCLONAL ANTIBODY TO AN ENDOGENOUS STEROIDAL NA/K-ATPASE LIGAND, MARINOBUFAGENIN, REVERSES EXPRESSION OF PROFIBROTIC GENES IN AGED RATS

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Objectives: Cardiovascular fibrosis is a hallmark of aging. We had previously demonstrated that an endogenous Na/K-ATPase inhibitor, marinobufagenin (MBG) plays a central role in cardiac fibrosis occurring in the context of experimental uremic cardiomyopathy (Hypertension,2007;49:215-24). Here, we hypothesized that MBG is implicated in aging-associated fibrosis, and that immunoneutralization of MBG in old rats will reverse pro-fibrotic signaling.

Methods: To test our hypothesis we measured blood pressure (BP) and plasma MBG in young (3-mo old) and aged (24-mo old) Sprague-Dawley rats, and in aged rats determined the effect of immunoneutralization of MBG on the expression of pro-fibrotic genes in left ventricular (LV) myocardium. One week following a single administration to aged rats of an anti-MBG monoclonal antibody (n=6) or vehicle (n=6), the expression of genes and levels of proteins implicated in pro-fibrotic signaling (qPCR) were assessed in LV myocardium.

Results: BP did not change, and plasma MBG levels were elevated two-fold ($P<0.05$) in old vs. young rats, and was accompanied by upregulation of genes implicated in TGFβ-signaling: TGFβ1 - 3-fold; CTGF1 – 6-fold; SMAD3 – 2-fold; collagen-1 – 2.6-fold. Expression of these genes was significantly suppressed following immunoneutralization of MBG in aged rats, although their expression remained higher than in young controls: TGFβ1 – 1.8-fold, CTGF1 – 3-fold, SMAD3 – 1.4-fold, collagen-1 – 1.7-fold. The expression of a nuclear transcription factor Fli-1, a negative regulator of collagen-1 synthesis, was reduced by 3-fold in old vs. young rats, and anti-MBG antibody restored levels of Fli-1 in old rats to the level in young controls.

Conclusions: The age-associated increase in MBG participates in pro-fibrotic signaling linked to advancing age, and cross-talk between TGFβ-dependent and Fli-1-dependent pro-fibrotic pathways underlies this MBG effect.

RACIAL DIFFERENCES IN PRESSURE RESPONSES FOLLOWING PEAK EXERCISE: INSIGHT FROM PRESSURE WAVE SEPARATION ANALYSIS

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Introduction: African Americans (AA) experience a much higher incidence of hypertension and as a result greater levels of disease morbidity and mortality when compared with Caucasians (CA). Post exercise hypotension (PEH) provides insight of blood pressure (BP) control and PEH may differ between AA and CA.

Objectives: To compare the BP response following, peak aerobic exercise (VO_2peak), between AA and CA.

Methods: Young generally healthy sedentary (~24 yr) AA (n=31, BMI=28.3 kg/m^2) and CA (n=29, BMI=24.7 kg/m^2) individuals had brachial (bSBP), and aortic (aSBP) measurements obtained in the supine position at rest, 15 and 30 min following a peak bout of aerobic exercise. Applanation tonometry was used to obtain aSBP pressure waveforms. Wave separation analyses were used to produce forward and reflected wave pressures height (FPH, RPH).

Results: (See table) CA men decreased both aSBP and bSBP following exercise, but AA men did not ($p<.05$). FPH and RPH decreased following peak exercise in both groups, but the decrease in FPH was greater in CA men, which approached significance ($p=0.052$).

Conclusions: PEH is apparent in CA, but not in AA. Interestingly, the difference in PEH can be partially attributed to greater changes in FPH in CA, which may implicate changes in ventricular function or aortic reservoir function.

	CA			AA		
	Pre	15-min post	30-min post	Pre	15-min post	30-min post
Aortic SBP (mm Hg) †	103 ± 10	100 ± 11	98 ± 9‡	102 ± 9	103 ± 12	103 ± 12
Brachial SBP (mm Hg) ** †	122 ± 4	118 ± 13‡	116 ± 12‡	118 ± 12	120 ± 16	118 ± 13
FPH (mm Hg) **	30 ± 7	26 ± 6‡	26 ± 6‡	27 ± 6	26 ± 8	25 ± 6‡
RPH (mmHg) **	13 ± 2	9 ± 3‡	9 ± 16‡	13 ± 3	10 ± 4‡	10 ± 3‡

All Data is mean ± SD
** time effect, $p<0.05$
† interaction effect, $p<0.05$
‡within group different from pre, $p<0.05$

DIFFERENTIAL ASSOCIATION OF THE FORWARD AND REFLECTED PULSE WAVES WITH AORTIC DIAMETER IN A COMMUNITY-DWELLING POPULATION OF NORMOTENSIVE AND UNTREATED HYPERTENSIVE MEN AND WOMEN

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Background and Objective: Prior studies have shown an inverse association between pulse pressure (PP) and aortic diameter (AoD); this was attributed to greater characteristic impedance with smaller AoD. However, PP is composed of an incident pressure (Pi) (the forward wave), and augmented pressured (Pa) (the reflected wave), with the latter not being affected by characteristic impedance. We hypothesized that in contrast to central PP (cPP) and Pi, Pa would be directly associated with AoD.

Methods: We studied a cohort of normotensive and untreated essential hypertensive Taiwanese participants (675 men, 601 women, mean age 52 years). Carotid applanation tonometry was performed to determine Pa and Pi. M-mode echocardiogram was performed to determine AoD. Linear regression models were performed to test the association of PP, Pa and Pi with AoD in men and women.

Results: Initial models confirmed the inverse association between cPP and AoD in men (β=-0.14, P=0.003), and women (β= -0.19, P=0.050), adjusting for age, mean blood pressure, and body surface area. In subsequent models, when cPP was expressed as Pi and Pa, Pi was inversely associated with AoD (β= -0.09, P=0.019), while Pa was directly associated with AoD (β= 0.09, P=0.0326) among men. No significant association between Pa and AoD was observed in women.

Conclusion: The inverse association observed between PP and AoD is driven by the forward wave; however, the reflected wave seems to impose a dilatory force in men. Additional prospective studies are needed to assess the longitudinal impact of reflected wave on aortic dilatation with aging.

EFFECTS OF GENDER AND RACE ON INTERLEUKIN-6 PRODUCTION IN RESPONSE TO CRP IN HUVECS

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Background: Interleukin-6 (IL-6) is a pro-inflammatory cytokine that can be excreted by endothelial cells. Elevated C-reactive protein (CRP) level is a risk factor for the development of endothelial dysfunction that can lead to hypertension. The study was to determine whether race or gender affected the IL-6 response to CRP in HUVECs.

Methods: Eight HUVEC lines from African American (AA) and Caucasian (CA) donors with gender split evenly were cultured and incubated with CRP for 24-hrs. Doses of CRP were 0, 25, 50 and 100 μg/mL. IL-6 was measured in the media that was collected after the incubation and was normalized to protein concentration (pg/mg).

Results: The female group had higher IL-6 production than the male group for each CRP condition and there was a significant difference between genders at the 50 μg/mL CRP condition (P=0.03). Though the IL-6 production tended to be higher in the AA group under 25, 50, and 100 μg/mL CRP conditions, the differences were not significant.

CRP level (μg/mL)	IL-6 production (pg/mg)	
	Female	Male
0	66.96 ± 30.88	82.73 ± 28.75
25	144.86 ± 50.15	92.03 ± 40.77
50	177.97 ± 20.73	110.98 ± 25.56
100	179.15 ± 59.41	151.94 ± 103.41

Conclusion: It suggests that females with high CRP levels may have a greater risk of developing endothelial dysfunction due to a greater IL-6 response. Whether the gender difference is related to differences in CRP receptor function or downstream signaling mechanisms is unknown.

ENDOTHELIAL MICROPARTICLES SHOW A BLUNTED EXERCISE RESPONSE IN AFRICAN AMERICANS WITH HIGH LEVELS OF INFLAMMATION

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Objective: African Americans (AA) present a population marked by high levels of endothelial dysfunction and inflammation. In this study we sought to assess whether Endothelial Microparticles (EMPs), a measure of endothelial dysfunction (ED), adapt to aerobic exercise (AEXT) the same way in AA with high levels of inflammation when compared to AA with normal-average levels of inflammation.

Methods: 23 AA with CRP levels <3 mg/L and 14 AA with CRP ≥3 mg/L underwent 6 months of mild-intensity AEXT training. Participants were sedentary (aerobic exercise ≤ two times per week), non-diabetic (fasting blood glucose ≤ 125 mg/dL), non-smoking (≥ 2 years), had a clinical blood pressure (BP) < 160/100 mmHg, were non-hyperlipidemic (total cholesterol ≤ 240mg/dL), had no signs of cardiovascular, renal, or pulmonary disease, and were not on any lipid lowering medication or medications affecting cardiovascular or renal hemodynamics. Markers of early stage ED (CD62E+ EMPs) and late stage ED (CD31+/CD42b- EMPs) were measured in platelet free plasma via flow cytometry.

Results: CD62E+ EMPs improved in both the normal-average and high inflammatory groups respectively (39.2±6.4 vs. 24.0±5.5 events/μL, p≤0.05 and 48.5±9.2 vs. 19.7±4.0 events/μL, p≤0.05). CD31+/CD42b- EMPs only improved in the normal-average inflammatory group 3.7±0.5 vs. 2.2±0.3 events/μL, p≤0.05).

Conclusions: Mild-intensity AEXT training was successful in improving markers of early stage endothelial dysfunction in both the high and normal-average inflammatory groups. Higher levels of exercise intensity or perhaps additional interventions may be needed in AA with high levels of inflammation to decrease markers of late stage ED.

THE EFFECT OF AEROBIC EXERCISE TRAINING ON ENDOTHELIAL HEALTH STATUS IN PRE- AND POST-MENOPAUSAL AFRICAN AMERICAN WOMEN

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Objectives: When compared to other racial groups, African American women (AAW) reach menopause at an earlier age and are at an increased risk for cardiovascular disease (CVD) and endothelial health complications. A known intervention for the treatment of both CVD and menopausal changes is aerobic exercise training (AEXT). The purpose of this study was to compare the effects of AEXT on endothelial function between pre- and post-menopausal AAW.

Methods: Healthy, pre- and post-menopausal AAW completed 6 months of mild-intensity AEXT. Plasma samples were analyzed for early and late phase endothelial dysfunction by labeling endothelial microparticles with CD62E+ and CD31+/CD42b respectively and quantified using flow cytometry

Results: CD62E+ EMPs (n=12) were not different between pre- and post-menopausal groups before (44±8.5 vs. 42.5±9 events/μL) or after (26.64±8.4 vs. 19.79±3.2 events/μL) AEXT. Both pre- (44±8.5 vs. 26.6±8.4 events/μL, $p \leq 0.034$) and post-menopausal (42.5±9 vs. 19.8±3.2 events/μL, $p \leq 0.008$) groups had lower levels of CD62E+ EMPs after AEXT.

CD31+/CD42b- EMPs (n=11) did not differ between pre- and post-menopausal AAW before (4.52±0.9 vs. 2.9±0.4 events/μL) or after (2.1±0.4 vs. 2.31±0.5e vents/μL) AEXT. CD31+/CD42b- levels significantly decreased in pre-menopausal AAW (4.52±0.9 vs. 2.1±0.4 events/μL, $p \leq 0.015$) after AEXT.

Conclusions: Our findings suggest that AEXT has the potential to improve early stage endothelial dysfunction regardless of menopausal status. However, post-menopausal women may need additional interventions or a greater exercise stimulus to improve late stage endothelial dysfunction.

RACIAL DIFFERENCES IN BLOOD FLOW DURING ACUTE DYNAMIC EXERCISE

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Objectives: African Americans (AA) have altered vascular function, even with a normal brachial blood pressure and beginning as early as 21 years of age. However, it is unknown if this impacts the blood flow response to exercise. We investigated baseline arterial and hemodynamic variables and blood flow during exercise in AA and Caucasian (CA) men.

Methods: Seventeen young (mean age=25 years), healthy AA (n= 7) and CA (n=10) men underwent measures of vascular function and stiffness at rest. Brachial blood flow was measured at rest and during the last minute of 5 minutes of dynamic rhythmic handgrip exercise (1 second contraction followed by 2 second relaxation) at both 10% and 20% of maximal voluntary contraction (MVC).

Results: AA had lower brachial blood flow at all time points (rest AA=19.17, CA=21.23; 10% MVC AA=22.60, CA=31.76; 20% AA=28.51, CA=39.95), however, only the 10% MVC reached significance (p=0.035), although the 20% condition approached significance (p=0.076). There were no other significant differences in any of the other measures (Table 1).

Conclusions: AA have reduced brachial blood flow at rest and during exercise compared to CA. This potentially could be due to decreased nitric oxide bioavailability, which could be a factor in the endothelial dysfunction previously seen in AA.

Table 1: Descriptive Statistics

	Total (n=17)	**AA (n=7)**	**CA (n=10)**
Age (yr)	25.2 ± 1.1	24.4 ± 1.9	25.8± 1.5
BMI (kg/m^2)	23.6 ± 0.98	24.0 ± 1.6	23.3 ± 1.2
Height (cm)	179.0 ± 1.6	176.6 ± 2.3	180.7 ± 2.2
Weight (kg)	84.6 ± 3.6	85.3 ± 6.5	84.1 ± 4.4
IMT (mm)	.41 ± 0.02	0.45 ± 0.11	.38 ± 0.01
B-stiffness	5.9 ± 0.51	5.4 ± 0.54	6.2 ± 0.79
BrachialSBP (mmHg)	125 ± 2	125 ± 4	126 ± 3
BrachialDBP (mmHg)	68 ± 2	70 ± 3	66 ± 3
carotidSBP (mmHg)	116 ± 2	114 ± 3	117 ± 3
carotidDBP (mmHg)	68 ± 2	70 ± 3	66 ± 3
aorticSBP (mmHg)	105 ± 2	104 ± 3	105 ± 3
aorticDBP (mmHg)	69 ± 2	71 ± 4	68 ± 3
AIx@75	-4.3 ± 1.9	-5.7 ± 2.3	-3.4 ± 3.0
HR (bpm)	62 ± 3	61 ± 3	63 ± 4
FMD%	11.1 ± 1.31	9.44 ± 1.91	12.2 ± 1.76

THE HEMODYNAMIC EFFECTS ON BRACHIAL AND CENTRAL BLOOD PRESSURE WITH THE USE OF 5-HOUR ENERGY® DRINK ON HEALTHY ADULTS

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Introduction

Energy drinks have become one of the fastest growing beverage products in the US. Case reports of myocardial infarction, cardiac arrest, stroke and seizures have been linked to their usage and risen concerns.

Central blood pressure has been shown to be a better predictor of cardiac disease as opposed to peripheral readings.

Few studies have compared the effects of caffeine on central blood pressure; however, this is the first one assessing the effects of energy drinks on central blood pressure.

Methods

We are currently conducting a single center study that will involve a total of fifty healthy subjects between the ages of 20-65 where each subject will be their own control. Measurements of brachial blood pressure and central pulse wave pressure with an AtCor device on a 24hr caffeine free day and separate day with the usage of 5-Hour Energy® Drink will be compared (central pressure 3 hours post consumption of energy drink).

Results

Preliminary data from initial 10 subjects reveal the use of 5-Hour Energy® Drink widens pulse pressure in average by 7mmHg (range -6 to plus 18 mmHg). We noted a decrease in heart rate by 7bpm, with an increase the systolic aortic pressure of 5mmHg (range -18 to plus 22 mmHg) and decreases in the diastolic aortic pressure of 2mmHg. Interestingly the mean arterial pressure obtained by brachial and aortic wave pressure remained unchanged.

Conclusions

The acute use of 5-Hour Energy® Drink has consistent hemodynamic changes on both peripheral blood pressure and central pulse wave pressure. Elevated central aortic systolic blood pressure, if confirmed, may underlie adverse effects of energy drinks. Further investigation is needed.

References

1. Townsend RR: Does consumption of high-caffeine energy drinks affect high blood pressure? *J Clin Hypertens* 8(10): 744-745, October 2006.
2. W Stephen Waring, Judith Goudsmit, John Marwick, David J Webb, Simon R J Maxwell: Acute caffeine intake influences central more than peripheral blood pressure in young adults. *Am J Hypertens*. 2003 Nov; 16 (11 Pt 1):919-24.

SEX DIFFERENCES IN POST EXERCISE HYPOTENSION

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INTRODUCTION: Whether sex differences in post exercise hypotension (PEH) exist is controversial, mostly due to small numbers in previous studies. Also, it is unknown whether potential sex differences in PEH are associated with forward or reflected pressure waves.

OBJECTIVES: The purpose of this study was to investigate potential sex differences in PEH with wave separation analysis in young healthy individuals.

METHODS: 60 adults (27 males, 33 females ~ 24 yrs) participated in the study. Aerobic capacity was measured by graded exercise testing. Central pulse wave analysis was measured using tonometry and separated into forward and reflected wave at pre-exercise, 15 minute, and 30 minute post acute maximum aerobic exercise. Repeated measure Analysis of variance (ANOVA) was performed to investigate sex differences in post exercise hypotension status.

RESULTS: (See table) Males, but not females exhibited significant PEH in brachial SBP ($p<.05$) whereas neither sex exhibited PEH in aortic SBP. In addition, forward wave pressure was greater in men and showed significant changes post exercise in men only ($p>.05$). Reflected pressure was decreased with exercise in both sexes ($p<.05$), but there was no sex difference in reflected wave pressure.

CONCLUSIONS: The results suggest that only males developed PEH following exercise, but this was only evident using brachial pressure measurements. This highlights the importance of aortic pressure measurement to understand the impact of exercise and sex on PEH, and suggest that standard brachial BP measures are inadequate to characterize PEH.

	Brachial SBP (mmHg)			**Aortic SBP (mmHg)**			**Forward pressure (mmHg)**		
	Pre-Ex	Post 15	Post 30	Pre-Ex	Post 15	Post 30	Pre-Ex	Post 15	Post 30
Male	130±7	128±14*	123±12*	108±7	109±10	106±10	34±5	29±8	28±7*
Female	112±9	111±10*	111 ± 10	98±9	96±10	96±0	25±5	24±5	23±4

*Significant at $p<0.05$

DO CENTRAL ARTERIAL HEMODYNAMICS CONTRIBUTE TO WALKING PERFORMANCE IN OLDER ADULTS?

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Objectives: The consequences of vascular aging on clinically important measures of physical function are not fully understood. It is known that central arterial stiffness is associated with slow gait speed; however, the impact of central arterial function on fatigue during walking has not been addressed. The purpose of this study is to test the hypothesis that central arterial hemodynamics are associated with variation in walking performance.

Methods: Twenty-two normotensive healthy older adults (65 ± 4 yrs, 11 women) performed a 400-meter walk test. Peak and end gait speed were recorded. The inability to sustain peak gait speed was used to identify older adults (n=13) with reduced walking performance. Radial applanation tonometry was used to estimate central arterial parameters. Analysis of covariance adjusting for age was used to compare central arterial parameters between subgroups.

Results: Central pulse pressure was significantly higher in adults showing decreased walking performance (47 ± 2 vs. 36 ± 3 mmHg, p=0.01). Additionally, augmentation pressure was higher (15 ± 1 vs. 10 ± 1 mmHg, p=0.01) and pulse pressure amplification tended to be lower (1.17 ± 0.03 vs. 1.26 ± 0.03 mmHg, p=0.08) in the subgroup with decreased walking performance. No difference (p>0.05) was found for aortic pulse wave velocity or augmentation index between subgroups.

Conclusions: These results suggest that central arterial hemodynamics may impact upon the ability of older adults to sustain gait speed during a clinically relevant endurance walking test. More work with a larger sample size is needed to verify these preliminary findings.

EFFECTS OF CAFFEINE SUPPLEMENTATION ON THE RELATIONSHIP BETWEEN TOTAL ARTERIAL COMPLIANCE AND HEMODYNAMIC PARAMETERS DURING EXERCISE RECOVERY

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INTRODUCTION: Exercise recovery is an important and possibly vulnerable period. Caffeine (CAF) has been known to enhance performance measures, but its effects on cardiovascular measures have been equivocal both at rest and in recovery. Total arterial compliance (CWK) is an important hemodynamic determinant of aerobic capacity.

OBJECTIVES: To examine the relationship between CWK and hemodynamic parameters during exercise recovery with or without caffeine supplementation.

METHODS: Using a randomized cross-over design, 18 individuals (25 ± 3 yrs; 70.6 ± 13.9 kg/m^2) took either placebo or caffeine (400 mg) and underwent a graded exercise test to exhaustion (VO_2peak). Beat-to beat hemodynamics were assessed using finger plethysmography at 5-, 15-, and 30-min post-exercise.

RESULTS: CAF increased VO2peak versus placebo (46.5 ± 10.0 vs. 45.2 ± 9.6 mL/kg/min, $p<0.05$). Positive correlations were observed for CWK and stroke volume throughout recovery in both trials, with stronger correlations with CAF ($p<0.05$). A positive correlation existed between CWK and cardiac output only at post-5 and 30-min with placebo, whereas stronger relationships were observed at all time points with CAF ($p<0.05$). A negative correlation between CWK and total peripheral resistance was found at post-30-min with placebo ($p<0.05$), with a stronger relationship at all time points with CAF.

CONCLUSIONS: Higher stroke volume, cardiac output, and lower TPR are associated with higher compliance in the recovery period, which is exacerbated with caffeine. These data suggest caffeine may be beneficial in the recovery period.

Table 1: Correlation (*r*) between total arterial compliance and hemodynamic variables in Placebo vs. Caffeine

	Total Arterial Compliance (CWK)					
	Placebo			***Caffeine***		
	Post-5	Post-15	Post-30	Post-5	Post-15	Post-30
SBP	.156	-.289	-.303	.261	.140	.249
DBP	.250	.148	.067	.124	.055	.360
MAP	.125	-.258	-.266	.057	-.011	.200
PP	-.126	-.401	-.399	.246	.143	.078
SV	.694*	.596*	.623*	.841*	.804*	.728*
CO	.667*	.438	.695*	.757*	.798*	.796*
TPR	-.332	-.409	-.724*	-.850*	-.819*	-.805*
HR	-.118	-.340	-.124	-.123	-.186	-.106

NOTE: Values are mean ± SD. SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP; mean arterial pressure; PP, pulse pressure; SV, stroke volume; CO, cardiac output; TPR, total peripheral resistance; HR, heart rate. * $p<0.05$, significant correlation.

COMPARISON OF STRUCTURAL AND FUNCTIONAL VASCULAR INDICES BETWEEN AMBULATORY AND NON-AMBULATORY ADULTS WITH CEREBRAL PALSY

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Objectives: Adults with cerebral palsy (CP) and associated sedentary lifestyle since their childhood may be at increased risk for major adverse cardiovascular events. We hypothesized that cardiovascular health may be compromised in individuals with CP age 18 years and over, and in particular, in those who are non-ambulatory. Our primary objective was to examine structural and functional vascular indices in ambulatory and non-ambulatory adults with CP.

Methods: In this ongoing study, we aim to recruit 50 individuals with CP. Complete assessments of 15 adults with CP were recruited for the study (33±10.7 yrs) (6 ambulatory) (9 non ambulatory). Central (cPWV), upper (uPWV) and lower limb (lPWV) pulse wave velocities were measured using simultaneous applanation tonometry of the common carotid, femoral, radial, and dorsalis pedis arteries. Absolute and relative flow-mediated dilation (FMD) of the brachial artery was measured on the dominant arm of the participant.

Results: cPWV was not significantly different between the two groups ($p>.05$). However, uPWV and lPWV were faster in non-ambulatory compared to ambulatory participants (uPWV: non-ambulatory 8.94m/s, ambulatory 6.38m/s $p=.02$) (lPWV: non-ambulatory 8.70m/s, ambulatory 7.05m/s $p=.03$). Absolute and Relative FMD were not different between groups ($p>.05$).

Conclusions: These preliminary findings indicate that non-ambulatory adults have elevated peripheral but not central arterial stiffness, despite no significant difference in endothelial function. The peripheral arterial stiffness changes may be associated with both upper and lower extremity mobility impairments in the non-ambulatory group. The absence of significant changes in FMD values may indicate preserved endothelial function in the less active non-ambulatory group.

SPIRONOLACTONE REVERSES PULSE WAVE VELOCITY IN PATIENTS WITH RESISTANT HYPERTENSION

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Objectives: Arterial stiffness in humans, measured as pulse wave velocity (PWV), is directly correlated with an endogenous Na/K-ATPase (NKA) ligand marinobufagenin (MBG). Both PWV and MBG vary with a dietary NaCl intake (CJASN;2013,8), and become elevated in hypertensives with chronic renal failure; and MBG binding to NKA in human arteries initiates the synthesis of collagen (JH;2011,29(4):769-776). Since the inhibitory effect of MBG on NKA is competitively reversed by aldosterone antagonists, we hypothesized that spironolactone might restore NKA activity in patients with resistant hypertension (RH), reduce PWV and improve elasticity of blood vessels.

Methods: We measured blood pressure (BP), PWV, plasma MBG, and erythrocyte NKA in seven control subjects and in eight RH patients treated with lisinopril/amlodipine/hydrochlorothiazide (LAH), before and after addition of spironolactone (50 mg/day) to the LAH regimen for six months.

Results: Prior to the addition of spironolactone to the LAH regimen, plasma MBG, BP, and PWV were higher, and NKA activity was reduced in RH patients vs. controls (Table). Addition of spironolactone to conventional triple (LAH) therapy for RH reduced BP and PWV, and reversed erythrocyte NKA inhibition in the absence of an effect on plasma MBG.

Conclusions: Thus, aldosterone antagonists reversed MBG-induced NKA inhibition and reduced PWV and BP in patients with RH. These beneficial effects of aldosterone antagonists may, in part, linked to a concomitant reduction in arterial fibrosis (see next abstract "Aortic fibrosis induced by a steroidal endogenous Na/K-ATPase inhibitor, is reversed by aldosterone antagonist canrenone").

	Controls	RH, baseline	RH, after spironolactone treatment
Age (years)	50 ± 3	55 ± 2	55 ± 2
Plasma MBG (nmol/L)	0.24 ± 0.03	0.42 ± 0.07*	0.53 ± 0.05**
Erythrocyte NKA (μmol P_i/mL/hr)	2.8 ± 0.2	1.9 ± 0.2**	2.3 ± 0.2$^{\#\#}$
PWV (m/sec)	6.3 ± 0.6	8.9 ± 0.3**	8.4 ± 0.2$^{\#\#}$
24-hour SBP (mmHg)	125 ± 2	149 ± 3**	142 ± 2$^{\#\#}$
24-hour DBP (mmHg)	75 ± 2	95 ± 2**	86 ± 2$^{\#\#}$

Mean±SE; *-P<0.05;**-P<0.01 vs. controls, $^{\#\#}$-P<0.01 vs. baseline RH (ANOVA)

AORTIC FIBROSIS INDUCED BY A STEROIDAL ENDOGENOUS NA/K-ATPASE INHIBITOR IS REVERSED BY ALDOSTERONE ANTAGONIST CANRENONE

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Objectives: The engagement of the Na/K-ATPase (NKA) in human arteries by an endogenous NKA ligand marinobufagenin (MBG) stimulates synthesis of collagen via Fli-1-dependent pro-fibrotic signaling (JH;2011,29 (4):769-776). Since the inhibitory effect of MBG on the NKA is competitively reversed by aldosterone antagonists, we hypothesized that canrenone will reverse the pro-fibrotic effects of MBG, i.e. collagen deposition, and improve arterial relaxation.

Methods: Explants of thoracic aortae and renal NKA from Wistar rats were incubated in the presence of vehicle or MBG with or without 10 μmol/L canrenone. Vasoconstriction and vasorelaxation properties of aortic explants were then tested, and collagen content amount was assessed by immunohistochemistry and western blotting.

Results: MBG inhibited renal NKA in a concentration-dependent fashion (IC_{50}=1.9±0.5 μmol/L), and the sensitivity of NKA to MBG was markedly reduced by 10 μmol/L canrenone (113±11 μmol/L, P<0.01). Treatment of aortic explants with MBG induced a two-fold increase in collagen-1, and this was accompanied by a reduction in the sensitivity to the vasorelaxant effect of sodium nitroprusside following endothelin-1-induced constriction (EC50=480±67 nmol/L vs. 23±3 nmol/L; P<0.01 vs. vehicle treatment). Canrenone blocked the effects of MBG to stimulate collagen synthesis, and restored the relaxation of aortic explants and response to sodium nitroprusside (EC50=17±1 nmol/L).

Conclusion: Canrenone reversed MBG-induced vascular collagen deposition and enhance relaxation in rat aortae explants. These data support the conclusion raised in the prior abstract that the ability of spironolactone to reduce arterial stiffness in patients with resistant hypertension and to restore their NKA in the presence of increased plasma MBG is linked to a reduction in arterial collagen. Thus, MBG-induced arterial remodeling and arterial stiffness are novel targets for aldosterone antagonists.

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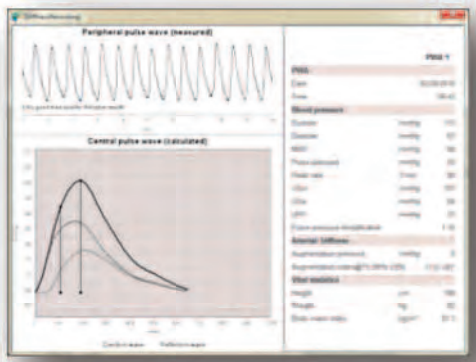

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