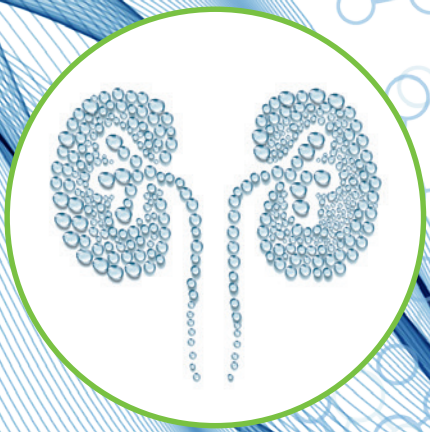
DANONE NUTRICIA RESEARCH

64TH AMERICAN COLLEGE OF SPORTS MEDICINE ANNUAL MEETING

# HYDRATION & EXERCISE : IMPLICATIONS FOR KIDNEY HEALTH



May 31st • 5.30pm-7.00pm
Room 103
Colorado Convention Center • Denver

# AGENDA

## Session chaired by :

**Stavros A. Kavouras, Ph.D., F.A.C.S.M., F.E.C.S.S.**
University of Arkansas, USA

**Exercise, cardiac output at high intensity & renal injury**
**Evan C. Johnson, Ph.D.**
University of Wyoming, Laramie, WY

**Mechanism(s) for dehydration-mediated kidney disease : the Mesoamerican Nephropathy example**
**Richard J. Johnson, M.D.**
University of Colorado, Denver, CO

**Kidney injury and repair Biomarkers in Marathon Runners**
**Chirag R. Parikh, M.D., Ph.D., F.A.C.P.**
Yale University, New Heaven, CT



CHAIRMAN OF THE SESSION,

## Stavros A. Kavouras Ph.D., F.A.C.S.M., F.E.C.S.S.

Department of Health, Human Performance and Recreation
University of Arkansas, USA

**Dr Stavros A. Kavouras** is an Associate Professor and Director of the Hydration Science Laboratory at the University of Arkansas & Adjunct Professor in Medicine at the University of Arkansas Medical Sciences, Division of Endocrinology. His laboratory is studying the mechanisms by which water intake affects health and performance. Dr Kavouras is the author of more than 100 peer review articles and he has given lectures in 28 countries. He is a section Editor of the European Journal of Nutrition and program coordinator for the Exercise Science program. Dr Kavouras is a Fellow of the American College of Sports Medicine & the European College of Sports Science as well as elected member of the American Physiological Society, the American Society of Nutrition, and the Obesity Society.

# Evan C. Johnson, Ph.D.

University of Wyoming, Laramie, WY

**Dr Evan Johnson** is currently an Assistant Professor at the University of Wyoming. His research as part of the Human Integrated Physiology Laboratory aims to examine the physiological mechanisms behind and the overall health benefits related to the hydration process, physical activity, and heat exposure. He has moved to his current position following the completion of post-doctoral appointment at the University of Arkansas working with Dr Stavros Kavouras on projects related to accurate measurement of fluid intake as well as the relationships between water intake and health outcomes. Previously, he completed his doctoral and master's degrees at the University of Connecticut, mentored by Professor Lawrence Armstrong. Over his career Dr Johnson has led eight research studies and assisted with more than 20 total projects, resulting in 27 publications, and seven as lead author. He has presented research findings at national and international meetings. Between his master's and doctoral degrees, Dr Johnson worked as a researcher at the Naval Health Research Center in San Diego, California specializing in optimizing safety during Navy SEALs training under the direction of Jay Heaney in the Department of Warfighter Performance.



# EXERCISE, CARDIAC OUTPUT AT HIGH INTENSITY & RENAL INJURY

The kidneys are integral to maintenance of homeostasis. In response to numerous hormonal and physical signals, filtration of the blood plasma regulates, allowing for adequate water retention, waste removal, electrolyte balance, and acid-base stability. Within the hospital setting kidney dysfunction is most often the result of chronic insults, commonly associated with the increased pressure within the renal nephron as seen during hypertension, or high volume glucose reabsorption in the case of diabetes. However, in otherwise healthy populations, acute kidney injury is a specific reduction in kidney function that occurs following to short-term reductions in renal blood flow, direct kidney damage, or blockage of the urinary tract. Exercise and the changes in cardiac output that are concurrent, alter renal plasma flow and glomerular filtration rate. Generally, these changes are advantageous for exercise performance and have little to no impact on kidney function. However, in certain circumstances acute kidney injury can result. This lecture will serve as a primer to introduce the physiology of kidney function before, during, and following exercise of different modalities and its relationship to cardiac output.

# Richard J. Johnson, M.D.

University of Colorado, Denver, CO

**Dr Richard Johnson** is a Professor and Chief of the Division of Renal Diseases and Hypertension at the University of Colorado, in Denver, USA. Dr Johnson is well known as a clinician, teacher and researcher, and is one of the founding editors of Comprehensive Clinical Nephrology, a popular textbook. Dr Johnson is especially known for his research on the pathogenesis of kidney diseases, including glomerulonephritis, diabetic nephropathy, and chronic kidney disease Dr Johnson's research has examined the role of subtle renal injury in salt sensitive hypertension and uric acid and fructose in the pathogenesis of hypertension, metabolic syndrome, and diabetic kidney disease. More recently Dr Johnson has had an interest in the etiology of the epidemics of chronic kidney disease that have emerged in Central America, India and Sri Lanka, and he has been a proponent for a role for heat stress and dehydration as contributory factors. Dr Johnson is a prolific and highly cited author with over 500 publications and a number of books in the field of nephrology. Dr Johnson has served on the International Society of Nephrology (ISN) Executive Committee and has been an ISN Councilor for the United State. In 2017 he received the David Hume Award from the National Kidney Foundation in the USA.



# MECHANISM(S) FOR DEHYDRATION ASSOCIATED KIDNEY DISEASE : THE MESOAMERICAN NEPHROPATHY EXAMPLE

Acute dehydration is well known to be associated with a "pre-renal" type of dysfunction, in which urea is retained out of proportion to serum creatinine due to enhanced reabsorption of water, urea, and electrolytes. Typically, this type of dehydration induced renal dysfunction is not thought to be associated with tubular injury per se, and it is considered completely reversible. However, there is now increasing evidence that the processes associated with urinary concentration come at a cost of some oxidative stress and injury, and that this may lead to the development of chronic kidney disease over time. Two major mechanisms are linked primarily with water depletion-those being the effects of hyperosmolarity to induce vasopressin, which by acting via the vasopressin 2 receptors mediates both glomerular and tubular injury; and second is the induction of the polyol-endogenous fructose pathway that can lead to proximal tubular injury. Other mechanisms causing renal damage may also result from chronic heat stress and dehydration, including the effects of subclinical rhabdomyolysis, salt loss and extracellular volume depletion, hyperuricemia and uricosuria/crystal formation, and heat stroke. Dehydration may also potentiate the effect of toxins and nephrotoxic agents on the kidney. Indeed, understanding the mechanisms by which dehydration can contribute to chronic kidney disease reveals a new and previously unrecognized mechanism by which inadequate hydration may accelerate kidney disease of any etiology. Thus, increased hydration should be viewed as a possible therapeutic intervention in all subjects with chronic kidney disease.

# Chirag R. Parikh, M.D., Ph.D., F.A.C.P.

Yale University, New Heaven, CT

**Chirag R. Parikh** is Professor of Medicine in the Section of Nephrology, Professor of Investigative Medicine, and Director, Program of Applied Translational Research, at Yale University School of Medicine. He is also a Professor in the Clinical Epidemiology Research Center at the VA Connecticut Health Care System. Dr Parikh received his MD at Seth G.S. Medical College and KEM Hospital in Bombay, India and his PhD in Clinical Investigation from the University of Colorado. His principal research interests focus on the translation and validation of novel biomarkers for the diagnosis and prognosis of acute kidney injury and chronic kidney disease. In 2005, Dr Parikh founded TRIBE-AKI (Translational Research Investigating Biomarker Endpoints in acute kidney injury) - a consortium of researchers across North America who have enrolled over 3 000 patients in several prospective cohort studies. TRIBE-AKI has yielded groundbreaking, nationally recognized achievements in biomarker discovery and validation, and its success led to the establishment of the Program of Applied Translational Research (patr.yale.edu) in 2012 at Yale University. This Program is comprised of a multidisciplinary team of talented physicians and scientists committed to patient-oriented research and improved outcomes in clinical medicine and disease management. As Director of PATR, Dr Parikh oversees several NIH-sponsored, multicenter studies testing biomarkers in the areas of cardiac surgery, diabetic kidney disease, kidney transplantation, HIV kidney disease, and hepatorenal syndrome.



# KIDNEY INJURY AND REPAIR BIOMARKERS IN MARATHON RUNNERS

Investigation into strenuous activity and kidney function has gained interest given increasing marathon participation. Kidneys are particularly susceptible to these injury due to ischemia and hyperthermia. We describe a prospective observational study of runners participating in the 2015 Hartford Marathon. Acute kidney injury (AKI) as defined by AKI Network (AKIN) criteria. Stage 1 AKI was defined as 1.5- to 2-fold or 0.3-mg/dL increase in serum creatinine level within 48 hours of day 0 and stage 2 was defined as a more than 2- to 3-fold increase in creatinine level. Microscopy score was defined by the number of granular casts and renal tubular epithelial cells. Serial samples were collected 24 hours premarathon (day 0), immediately postmarathon (day 1), and 24 hours postmarathon (day 2). Six novel injury urine biomarkers (IL-6, IL-8, IL-18, kidney injury molecule 1, neutrophil gelatinase-associated lipocalin, and tumor necrosis factor α) and 2 repair urine biomarkers (YKL-40 and monocyte chemoattractant protein-1) were measured.

82 % of runners developed an increase in creatinine level equivalent to AKIN-defined AKI stages 1 and 2. 73 % had microscopy diagnoses of tubular injury. Serum creatinine, urine albumin, and injury and repair biomarker levels peaked on day 1 and were significantly elevated compared to day 0 and day 2. Serum creatine kinase levels continued to significantly increase from day 0 to day 2. Marathon runners developed AKI and urine sediment diagnostic of tubular injury. An increase in injury and repair biomarker levels suggests structural damage to renal tubules occurring after marathon. Majority of the findings were reversible in 48-72 hours. Long term effects of this transient injury on the kidneys should be investigated further.

# Apply for
# The Hydration for Health
# Young Researcher Award 2017

**The Hydration for Health Initiative** invites young researchers in the field of healthy hydration to submit abstracts that contributes to the scientific field of hydration and health. Submission of abstracts is encouraged in, but not restricted to, the following areas: hydration markers, hydration and health in general and kidney health in particular, hydration and cognition, hydration, behaviour and well-being, fluid and water intake studies.


The young researcher who submits the winning abstract will be invited to present his/her research and results as an oral presentation at the **9th Annual Hydration for Health Scientific Conference** in Evian (France) on July 5th 2017.

**Deadline:**
**6th June 2017**

**To know more about the Hydration for Health Initiative:**

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