Hypertension Update
John E. Hall, Editor-in-Chief, on behalf of the Editors

The editors of Hypertension are grateful to the members the Council for High Blood Pressure Research for their strong support of the journal. Hypertension continues to thrive and to expand its readership and scientific impact. Hypertension now has a total circulation of over 20,000 with over 4.5 million online accesses for 2008 and over 1.4 million unique visitors to its Web site. Hypertension also has eight foreign translations. Thus, progress is being made to our goal of expanding journal readership.

All of the available indicators suggest that the scientific impact of Hypertension is also growing. For 2008, the Journal Citation Reports (JCR) scientific impact factor of Hypertension was the highest in the journal’s history at 7.368 and the highest of any journal devoted to basic or clinical hypertension research.

There has been a steady increase in manuscript submissions during the past seven years. Fortunately, the American Heart Association has provided increased page allowances so that a reasonable acceptance rate of 18–19 percent can be

MESSAGE FROM THE CHAIR
R. Clinton Webb, PhD, FAHA

Since our last newsletter, the main activities of your Leadership Committee have been coordinating abstract submissions and sessions for the fall conference. This will be the 63rd Annual Fall Conference of the Councils on High Blood Pressure and the Kidney in Cardiovascular Disease, to be held Sept. 23–26 at the InterContinental Chicago O’Hare Hotel. The Program Committee, headed by Rhian Touyz, is identifying session topics and structuring the workshop. Over 350 abstracts will be presented at the meeting. Information about the meeting and the preliminary program of the conference and the workshop are available online.

The workshop preceding the fall conference, to be held on Sept. 23, will focus on “Systems Medicine Strategies in Hypertension: From Molecules to Patients.” It has been organized by Drs. Rhian Touyz, Ernesto Schiffrin, Anna Dominiczak and Thomas Coffman. A joint plenary session will end the workshop where Dr. Dan Levy will give a state-of-the-art lecture entitled “Genes, Molecules, Systems Biology and Epidemiology in Hypertension — Bringing it All Together Through Framingham.” This lecture will be presented at 5:30 p.m., followed by Poster Session I. The first oral session is planned for Thursday.

The following topics will be covered in the workshop:

- Studying hypertension at the gene level — innovative strategies, new models and novel genes
- Studying hypertension at the molecular level — receptors, signal transduction, oxidative stress and imaging
- Vascular pathobiology in hypertension
- Clinical assessment of blood pressure — new trends
- Chairs
- Brain and kidney in hypertension

During the meeting, the following conference awards will be presented:

- Annual High Blood Pressure Research Conference New Investigator Award, sponsored by the Council for High Blood Pressure Research
- Kidney Council New Investigator Travel Award
- New Investigator Travel Award, sponsored by the Trainee Advocacy Committee of the Council on High Blood Pressure Research
- The Harry Goldblatt New Investigator Award
- New Investigator Award for European Fellows, supported by an educational grant from AstraZeneca

In addition to the fall conference, the plans for our next hypertension summer school continue to develop. The goal of this program is to attract and motivate postdoctoral fellows, students and clinical trainees into careers in hypertension and cardiovascular disease. The chair of the organizing committee for the school is Ray Townsend. Dates and location for the 2010 Hypertension Summer School are as follows:

Portland State University, Portland, Oreg.

Saturday, July 31 — Wednesday, Aug. 4, 2010

One of my major activities as Council Chair is to represent at meetings of the AHA Science Advisory & Coordinating Committee (SACC). This committee meets quarterly and reviews the business of the Councils of the AHA. The most recent meeting of this committee was on June 24 in Dallas. The action item from this committee that will have the greatest impact on members of our Council is the decision to reconsider the conflict of interest policy of the American Heart Association. There will be major changes in this policy and all members are encouraged to stay current with them through the Web site.

Remember that Scientific Sessions will be held in Orlando, Fla. at the Orange County Convention Center from Nov. 14–18, 2009.

Finally, I would like to say that this is an exciting time for the members of our Council. The next meeting of the Leadership Committee is Sept. 23 and we welcome suggestions for agenda items. We are here to serve you.
maintained. Approximately 58 percent of manuscripts submitted in 2008 were related to clinical or population science and 42 percent were basic science manuscripts.

We welcome your suggestions for new avenues to broaden the readership, to reach healthcare professionals and scientists who may not have traditionally viewed Hypertension as a “must read” journal, and to increase the journal’s overall scientific and clinical impact. Several new initiatives are underway to further expand readership and improve quality: 1) we have increased distribution of the journal’s contents by making “Editor’s Picks” (see Web site at http://hyper.ahajournals.org/) and other articles freely available to members of several international scientific societies; 2) we have raised the profile of the journal among AHA members by multiple initiatives, such as a partnering with the Council to co-sponsor a “Sunday Morning Session” at AHA Scientific Sessions; 3) last year we introduced two new features — “Controversies in Hypertension” and “Basic Mechanisms of Disease”; 4) we have introduced “Best Papers in Hypertension” Awards with the intent of recognizing the top basic, clinical and population science papers published in the journal; 5) we have provided more information for healthcare professionals to translate advances in basic and clinical research into more effective treatment of hypertension, especially by publishing guidelines and articles that are mainly “practical” in their application for effective treatment of hypertension and by publishing a continuing medical education (CME) feature.

The editors hope that Council members will consider submitting their best original papers to Hypertension. We promise a fair and speedy decision. The time from manuscript submission until first decision averages a short 2.6 weeks, acceptance to online publication is about 4 weeks, and the time required for print publication is about 7.4 weeks.

The editors are committed to ensuring that Hypertension continues to be the outstanding journal you deserve. We extend our sincere thanks for your support and outstanding scientific contributions. Please continue to send us your suggestions for improvement of the journal.

Editorial

Our Council membership has continued to grow and to support the many exciting things happening in our Council. One of the exciting upcoming events is our Council’s Fall Meeting in Chicago, Ill. (Sept. 23–26, 2009). Highlights of the meeting are outlined in the message from the Chair and can be found on the meeting Web site.

The two-and-a-half-day meeting will be preceded by a one-day workshop on “Systems Medicine Strategies in Hypertension: From Molecules to Patients” as discussed above. This meeting is one of the most important medical meetings on hypertension in the world and draws an international audience of over 700 attendees. It provides an educational program for physicians and research investigators to enhance their knowledge, advance their skills, and apprise them of the latest developments in research pertaining to hypertension and its relationship to stroke, cardiac disease, kidney function/renal diseases, obesity and genetics.

Our Council also participates in planning the National AHA Scientific Sessions in Orlando, Fla. (Nov. 14–18, 2009). Many sessions are sponsored or co-sponsored by our Council including the basic science sessions: “Mitochondria and Vascular Disease: Biology and Translational Priorities;” “ACE2: A Novel Regulator of Cardiovascular and Respiratory Physiology and Pathophysiology;” and “Genetics of Hypertension: How Far Have We Come and What Does the Future Hold?” Many of the translational science sessions should also be of interest to members of our Council including “Genetics of Peripheral Arterial Disease;” “Type 1 Diabetes and CVD: Bench to Bedside;” “Research and Clinical Applications of Measurements of Endothelial Function;” and “Epigenetics and Cardiovascular System.” Please plan to attend and take advantage of these outstanding meetings.

Plans are also underway for the 7th Hypertension Summer School July 31–Aug. 4, 2010. This will be at Portland State University in Portland, Oreg. Additional details will be included in the next newsletter. As always, remember that our Council Web site is filled with useful links to all things hypertension and is a tremendous resource for members and nonmembers.

Hypertension Research

To highlight some of my favorite articles in our Council journal, Hypertension, I have given short summaries of several articles from each issue this quarter. (Hypertension impact factor 7.368!)

May 2009 (Vol. 53 [5])


This clinical study investigated the effects of adding an aldosterone receptor (MRA) antagonist to an angiotensin converting enzyme (ACE) inhibitor in patients with chronic kidney disease (CKD). The data demonstrate that potassium handling is markedly blunted in CKD patients even in the absence of hyperkalemia and provides clinically useful information to establish a model for future study of medication-related effect on potassium homeostasis. This article is highlighted in a scholarly editorial commentary by Domenic Sica.


Regular exercise for five weeks increased the stability of atherosclerotic plaques independent of changes in blood pressure and markers of inflammation. This was associated with decreases in angiotensin type 1 but not type 2 receptor mRNA. This interesting study in ApoE null mice clearly demonstrates hemodynamic independent effects of angiotensin II in atherogenesis and the benefits of swimming exercise as an adjuvant therapy for atherosclerosis.

Epidemiological studies strongly suggest a link between exposure to various air pollutants and cardiovascular disease. This study in Detroit, Mich. found increased exposure to particulate matter was associated with increased systolic blood pressure, particularly in individuals <55 years of age. This lays the groundwork for future studies of the mechanisms producing this blood pressure effect and puts a new twist on interpreting studies that low-income communities are both more likely to be exposed to air pollutants and to have elevated blood pressure.

June 2009 (Vol. 53 [6])


The epithelial sodium channel (ENaC) is expressed in vascular tissue, and data from this study suggest it is a negative modulator of eNOS activity through inhibition of AKT. Several isoforms of the ENaC family were found to be present in both the endothelium and the tunic media while functional studies demonstrated a functional contribution to agonist-induced constriction. Upregulation of this channel by aldosterone may contribute to the deleterious effects of mineralocorticoid excess in the vasculature.


This study used Sprague Dawley rats to study potential mechanisms for renal damage caused by elevated renal perfusion pressure (RPP). The studies found increased RPP stimulated oxidative stress (seen as increased H2O2) and increased production of NO, independent of increases in renal hydrostatic pressure. The H2O2 production was stimulated by increased flow in the medullary thick-ascending limbs of Henle, while NO synthesis was stimulated by increases in vasa recta blood flow. Together the parallel pressure-induced increases of H2O2 and NO would be predicted to affect pressure-natriuresis so that increased RPP causes sodium and fluid loss through NO effects but at the expense of increased oxidative stress. These data suggest antioxidants might better protect the kidney during high perfusion than agents that increase NO levels.


This update on in vivo actions of the renin precursor, pro-renin, suggest that elevated prorenin in transgenic mice is not sufficient to cause cardiac fibrosis or renal glomerular sclerosis but does elevate arterial pressure. However, the lack of effect of active site-mutated prorenin and the efficacy of angiotensin-converting enzyme inhibition to lower blood pressure are consistent with prorenin acting through the generation of angiotensin II to raise blood pressure.

July 2009 (Vol. 53 [7])


This study used data from 8829 adult survey participants to assess trends in and predictors of blood pressure control. The analysis found that uncontrolled blood pressure is still common in chronic kidney disease patients in the US although there is some improvement in control through the use of multi-drug regimens, especially those including ACE inhibitors or ARBs. The incidence of uncontrolled blood pressure was especially high in nonwhites, older persons and women with CKD but was not influenced by socioeconomic factors. Finally, albuminuria was strongly associated with increased prevalence of uncontrolled blood pressure suggesting that there is still much room for improvement in effective treatment of this population.


Both low physical fitness and increased visceral adiposity are known risk factors for elevated blood pressure. This study evaluated the individual risks of each condition and found that both are independently associated with increased blood pressure. However, in individuals of similar fitness, elevated visceral adiposity independently associated with higher arterial pressure, especially in individuals in the highest tertile of cardiorespiratory fitness. Therefore increased visceral adiposity appears to mitigate the benefits of exercise and these findings call into question the protection of being both fit and fat.


Intermittent hypoxia appears to be one of the factors of sleep apnea leading to increased cardiovascular morbidity. This study in mice found that 10 days of intermittent hypoxia increased left ventricular remodeling with associated increases in inflammatory cytokines, oxidative stress, chymase activity and angiotensin II expression independent of changes in blood pressure. Inhibition of chymase activity with NK3201 suppressed all of these changes suggesting chymase activation during intermittent hypoxia produces local elevations in cardiac angiotensin II. Therefore chymase inhibition in patients with sleep apnea may more selectively target cardiac pathophysiology than other blockers of the RAS system.