This year, World Kidney Day (WKD) was held on March 8 in more than 50 countries. An initiative of the International Society of Nephrology (ISN) and the International Federation of Kidney Foundations (IFKF), the event included participation from the AHA's Council on the Kidney in Cardiovascular Disease (CKCD).

WKD raises awareness of the importance of our kidneys, the impact of kidney disease, the kidney-heart connection and related research. Since it is abundantly clear that people with chronic kidney disease are at substantially increased risk of cardiovascular disease — including high blood pressure, diabetes, heart attacks and strokes — WKD encourages screening of patients for early detection. These efforts reflect a 2006 advisory authored by the CKCD, “Detection of Chronic Kidney Disease in Patients With or at Increased Risk of Cardiovascular Disease.”

At this year’s WKD, representatives from our council joined advocates from the American Society of Nephrology, the National Kidney Foundation and the American Society of Pediatric Nephrology to lobby Congress. As a result, several Senate and House members, as well as their staffs, now know where their kidneys are and underwent screening for chronic kidney disease.

The council meetings will be preceded by a workshop on new developments in the renin-angiotensin system: the (pro)renin receptor, renin inhibition and ACE2. This one-day workshop will highlight two new components of the renin-angiotensin system (RAS), the (pro)renin receptor and ACE2, and address a new class of RAS blockers, the renin inhibitors. The (pro)renin receptor binds circulating kidney-derived renin, thereby potentially undermining angiotensin production at tissue sites. It also binds prorenin, the inactive precursor of renin. When bound to its receptor, prorenin displays enzymatic activity. Thus, for the first time, a physiological role for prorenin has been established. This is important in view of earlier observations that high prorenin levels in diabetic subjects are an indication of microvascular complications. Unexpectedly, renin and prorenin binding to their receptor not only facilitated angiotensin generation, but also led to activation of second messenger pathways, thereby implying that renin and prorenin act as agonists independently of angiotensin generation. Now that renin inhibitors will soon be clinically available, it is of the utmost importance to learn whether these drugs interfere with the (pro)renin receptor, and to what degree this offers an advantage over the existing RAS blockers. Simultaneously, the ACE homologue ACE2 has recently been identified as angiotensin-degrading enzyme capable of generating angiotensin-(1–7). This angiotensin metabolite is believed to counteract many of the adverse effects of angiotensin II through stimulation of its receptor Mas, and, given its increase during ACE inhibition and AT1 receptor blockade, may contribute to the beneficial effects of these drugs. Intriguingly, ACE2 also functions as a receptor for the virus causing severe acute respiratory syndrome (SARS), and it acts as a protective factor in various experimental models of acute lung failure. The finale of the workshop will be a special presentation entitled “From Prorenin to Renin Inhibition.”

The remainder of the meetings will focus on cardiovascular disease, stroke, hypertension and kidney function. Late-breaking abstracts will be accepted from June 4–July 3, 2007. Registration for the meeting opened May 2 and runs through Aug. 24.