20 YEARS OF EXCELLENCE IN INTERNATIONAL MEETINGS
alliedacademies.com

International Conference on
NEPHROLOGY AND UROLOGY

November 21-22, 2018 | Madrid, Spain

Jose Pedro A, J Clin Nephrol Ther 2018, Volume 2

GASTRORENAL AXIS IN THE CONTROL OF BODY SODIUM HOMEOSTASIS

Jose Pedro A
The George Washington University School of Medicine & Health Sciences, USA

Organ to organ communication is important in the maintenance of normal fluid and electrolyte balance and blood pressure (BP). The gastrointestinal tract and the kidney are major organs involved in this process. Neural mechanisms and gut hormones mediate the natriuresis of an oral sodium load. The flow of sodium into the sodium channels of the stomach antrum activates a sequence of events, leading to G-cell-mediated increase in gastrin secretion and its release into the circulation. Of all the gut hormones circulating in the plasma, gastrin is the one that is reabsorbed to the greatest extent by renal tubules. Gastrin, via its receptor, the cholecystokinin type B receptor (CCKBR) in the kidney inhibits renal sodium transport. Germline deletion of gastrin (*Gast*) or *Cckbr* gene in mice causes salt-sensitive hypertension. Selective silencing of *Gast* in the stomach and duodenum in mice impairs their ability to excrete an oral sodium load and increases BP. Thus, the gastro-renal axis, mediated by gastrin, can complement pronatriuretic hormones, such as dopamine, produced by the kidney in response extracellular fluid volume expansion, to increase sodium excretion after an oral sodium load. However, BP is not increased in patients who have had gastric bypass. Indeed, the high BP can be normalized by gastric bypass because of the release of other enterokines. Sleeve gastrectomy actually enhances the increase in plasma gastrin following a mixed meal. By contrast, Roux-en-Y gastric bypass surgery prevents the increase in plasma gastrin following a mixed meal but either type of bypass surgery increases plasma levels of natriuretic enterokines, such as glucagon-like peptide-1 (GLP-1). Gastrin, acting on renal CCKBR, GLP-1, acting on its receptor GLP-1R, also in the kidney, and dopamine produced in the kidney, acting on D1 dopamine receptors interact to negatively regulate renal sodium transport and keep the BP in the normal range.

BIOGRAPHY

Jose Pedro A received his MD degree, magna cum laude, meritissimus, from the University of Santo Tomas Philippines, and placed first in the Philippine National Board Examinations in Medicine and Surgery. He received his PhD degree in Physiology from Georgetown University, Washington, DC, USA. The primary goal of Jose's research is to determine the genetic and pharmacogenetic bases of human essential hypertension and the metabolic syndrome. He has published more than 380 scientific articles in book chapters and journals. Jose has received several academic and research awards, including the 2003 Lewis K. Dahl Memorial Lecture (American Heart Association), 2007 Ernest H. Starling Distinguished Lecture (American Physiological Society) and 2015 Excellence Award for Hypertension Research (American Heart Association). A key finding of Jose's research is the demonstration of the crucial role of gene variants of GRK4 in the pathogenesis and personalized treatment of hypertension.

pjose@mfa.gwu.edu