KSN 2017 Abstract

Ammonia (NH3) transport – the textbooks were wrong

David Weiner

University of Florida College of Medicine, USA

Acid-base homeostasis is central to almost all aspects of normal health. The kidneys contribute to acid-base homeostasis through net acid excretion, of which the quantitatively greatest component, both under basal conditions and in response to acid-base disturbances, is ammonia excretion. Ammonia is generated in the kidney and then is selectively transported into either the urine or the renal vein. Urinary ammonia excretion is associated with new bicarbonate generation, whereas ammonia transported into the renal vein either increases blood urea nitrogen levels, a result of its metabolism in the liver forming urea, or it increases arterial ammonia levels, potentially contributing to generation of hepatic encephalopathy. The traditional view of renal ammonia metabolism involves passive, lipid phase-based NH3 diffusion coupled to NH4+ trapping, because the latter could not across lipid bilayers, both components of this paradigm are now known to be incorrect. This talk will discuss evidence that renal ammonia excretion is beneficial in patients with chronic kidney disease, will review the current state of knowledge regarding ammonia transport in the kidney, and it will examine evidence that the Rhesus glycoprotein family has a central and essential role in this process.