S46-4 PGE2 circulation in renal cortex and blood pressure regulation

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Unveiling the genetic backgrounds for congenital hypertension revealed the common pathogenesis of enhanced sodium (Na) transport in distal tubule of the patients. However, the physiological mechanisms leading to the elevation of blood pressure (BP) after enhanced Na transport are still to be elucidated. Recently, novel PGE2 transporter expressed only in the kidney (OAT-PG) has been identified, and following studies including ours studies on experimental animals have demonstrated the presence of local circulation of PGE_2, which influences glomerular filtration, renal renin production/release, and vascular tonus of afferent and efferent arteries, from distal tubule to proximal tubule. These studies also demonstrated that the basal PGE_2 concentration would be determined by OAT-PG expression, which transports PGE_2 into proximal tubular cells for degradation, and transient alteration of its concentration might occur by the reactive PGE_2 production in distal tubule cells to sodium transport. We also suggested that basal PGE_2 concentration determined by the level of OAT-PG expression would be closely associated with BP level. Based on these previous reports and our recent findings, we presently try to propose putative mechanisms of the BP regulation influenced by the sodium transport in distal tubule.