MS05-9 **Sodium-dependent phosphate transporter and cardiovascular disease**

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Phosphate (P) is a essential nutrient as a component of ATP, nucleic acids, phospholipids as well as bone. The balance of P pool is maintained by absorption from the intestine, excretion from the kidney and exchange with bone. Among them, the excretion of P from the kidney is the most important step performed by sodium-dependent P transporter (NaPi), and can be tightly regulated by parathyroid hormone, vitamin D, and fibroblast growth factor 23 (FGF23)/Klotho pathway. Recent reports indicate that dysregulation of NaPi can cause hypo- or hyperphosphatemia, and cause various diseases such as arteriosclerosis as well as metabolic bone diseases. Especially, defect of FGF23/Klotho pathway can increase renal P absorption, and also show hyperphosphatemia and premature aging-like phenotype such as osteoporosis and cardiovascular disease (CVD). The phenotype can be ameliorated by P restriction diet. In addition, hyperphosphatemia can be significantly associated with incidence of cardiovascular disease in general population as well as chronic kidney disease patients, because hyperphosphatemia can induce vascular calcification. We recently found that hyperphosphatemia can mediate endothelial dysfunction via P influx by NaPi. In this symposium, we would like to introduce roles of NaPi in P homeostasis and CVD.