Endocrine regulation of phosphate homeostasis

Friday September 29, 2017
1:00 – 2:00 pm, East Campus 4, Boardroom (EC4-2101a)
Coffee and Cookies will be available - RSVP required

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Abstract:
Normal phosphate homeostasis is tightly controlled by numerous endocrine factors that coordinately exert effects on the intestine, kidney, and bone to maintain physiological balance. The importance of the fibroblast growth factor (FGF)-23–klotho axis in regulating phosphate homeostasis has been proposed from recent research observations. Human and experimental studies suggest that 1) FGF23 is an important in vivo regulator of phosphate homeostasis, 2) FGF23 acts as a counter regulatory hormone to modulate the renal 1α-hydroxylase and sodium–phosphate cotransporter activities, 3) most of the FGF23 functions are conducted through the activation of FGF receptors, and 4) such receptor activation needs klotho, as a cofactor to generate downstream signaling events. In this presentation, I will summarize how the FGF23–klotho axis might coordinately regulate normal phosphate homeostasis, and explain the cause and consequences of phosphate toxicity.

Further reading:

Biosketch:
My research is mostly devoted to determine molecular interactions of vitamin D, PTH (parathyroid hormone) & FGF23 (fibroblast growth factor 23) in physiological regulation of phosphate balance, & how dysregulation of these factors can lead to hyperphosphataemia with extensive tissue damage caused by phosphate toxicity. My clinical expertise is Pathology focusing on Renal diseases.

Keywords: bone health, endocrine, phosphate toxicity, fibrogenesis, molecular and cell biology, renal, collagen, heath shock proteins, fibroblast growth factor, physiology, pathology

Event is FREE – please RSVP via EventBrite. Pay parking available in lot Q (map)
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