

Biomedical Discussion Group

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 1 α -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:

1. Goetz R, Ohnishi M, Kir S, Kurosu H, Wang L, Pastor J, Ma J, Gai W, Kuro-o M, Razzaque MS, Mohammadi M. Conversion of a paracrine fibroblast growth factor into an endocrine fibroblast growth factor. *J Biol Chem*. 2012; 287(34):29134-46.
2. Razzaque MS. The FGF23-Klotho axis: endocrine regulation of phosphate homeostasis. *Nature Rev Endocrinol*. 2009; 5(11):611-9.
3. Goetz R, Nakada Y, Hu MC, Kurosu H, Wang L, Nakatani T, Shi M, Eliseenkova AV, Razzaque MS, Moe OW, Kuro-o M, Mohammadi M. Isolated C-terminal tail of FGF23 alleviates hypophosphatemia by inhibiting FGF23-FGFR-Klotho complex formation. *Proc Natl Acad Sci U S A*. 2010; 107(1):407-12.
4. Razzaque MS. Phosphate toxicity: new insights into an old problem. *Clin Sci*. 2011; 120(3):91-7.

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



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