Addendum Regarding "The Unappreciated Role of Extrarenal and Gut Sensors in Modulating Renal Potassium Handling: Implications for Diagnosis of Dyskalemias and Interpreting Clinical Trials" (*Kidney Int Rep.* 2016;1:43–56)

Meyer Lifschitz and Murray Epstein

Kidney Int Rep (2016) **1,** 210; http://dx.doi.org/10.1016/j.ekir.2016.07.006

Shortly after the in press publication of the review "The Unappreciated Role of Extrarenal and Gut Sensors in Modulating Renal Potassium Handling: Implications for Diagnosis of Dyskalemias and Interpreting Clinical Trials"[1], Lise Bankir and associates published a paper[2] suggesting that glucagon could serve as the mediator for the rapid renal response to an ingestion of potassium. This hypothesis is relevant to and complements our discussion of both (a) the putative location for the GI potassium sensor and, (b) the potential signal(s) that might modulate the increase in renal potassium excretion.

REFERENCES

1. Epstein M, Lifschitz MD. The unappreciated role of extrarenal and gut sensors in modulating renal potassium handling: implications for diagnosis of dyskalemias and interpreting clinical trials. *Kidney Int Rep*. 2016;1:43–56.
2. Bankir L, Bouby N, Blondeau B, et al. Glucagon actions on the kidney revisited: possible role in potassium homeostasis. *Am J Physiol Renal Physiol*. http://dx.doi.org/10.1152/ajprenal.00560.2015, accessed May 18, 2016.