Dear Sir,

A Letter to the Editor recently submitted by DeSanto et al. [1] regarding protein-mediated increases in renal hemodynamics reports an important phenomenon which deserves further comment. Work in our laboratory corroborates the findings of DeSanto et al. [1]. Using a pancreatectomy model (surgical excision) in anesthetized dogs, we collected data suggesting the importance of the pancreas, and in particular glucagon, in modulating amino acid-induced increases in canine renal blood flow (RBF) and glomerular filtration rate (GFR) [2]. Vascular infusion of amino acids in control dogs elevated RBF and GFR in parallel while concurrently increasing the arterial plasma level of glucagon. Yet, during identical infusion in pancreatectomized dogs, amino acids failed to reproduce the elevation in RBF and GFR and the increase in plasma glucagon that they evoked in control dogs. Replacement of plasma glucagon during amino acid infusion in pancreatectomized dogs, with an incremental plasma level that was not different from the level produced by amino acids alone in controls, was able to elevate RBF and GFR to levels that were on the average 71 and 78%, respectively, of the total RBF and GFR responses elicited by amino acid infusion in control dogs. Yet, when a physiological dose of glucagon was infused alone in pancreatectomized dogs, the hormone failed to reproduce the elevation in RBF and GFR evoked by either amino acids alone in control dogs or during combination amino acid/glucagon infusion in pancreatectomized dogs [2]. Thus, an interaction between amino acids and specific hormones (at least pancreatic glucagon) does appear to play an important role in the pathway by which protein and amino acids elevate renal postprandial hemodynamics. The nature of this interaction during the postprandial state remains an active area of investigation.

References
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