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Reply from the Authors

Thank you for calling our attention to the importance of anion exchange of chloride for short-chain fatty acid anions (SSFAA) as a potential mechanism of sevelamer-induced metabolic acidosis. To the best of our knowledge, no data have been published regarding the relative binding affinity of sevelamer hydrochloride (a quaternary amine anion exchange resin) for the various anions present in the intestinal lumen (phosphates, sulfates, bicarbonate, carbonate, citrate, bile acids, acetate, and other SSFAA). However, it seems likely that binding of SSFAA by sevelamer in the large intestine may well be a major factor in the genesis of sevelamer-induced metabolic acidosis, as you suggest. Final equilibration in the gut with binding of SSFAA, bicarbonate, and bile acids to the sevelamer resin, resulting in displacement of phosphate, may also help to explain the well known discrepancy between the in vitro and in vivo phosphate binding efficacy of sevelamer. In dialysis patients with refractory hyperphosphatemia, it has been recommended that sevelamer and calcium acetate or calcium carbonate be used concomitantly [1]. However, it is theoretically possible that simultaneous use of calcium-based phosphate binders containing acetate or carbonate could further reduce the phosphate-binding efficacy of sevelamer. These considerations suggest that an optimal strategy may be to alternate use of sevelamer and calcium-based phosphate binders with different meals during the day. Moreover, if sodium bicarbonate supplements are needed to maintain serum bicarbonate above 22 mEq/L, as recommended by K/DOQI guidelines [2], the oral bicarbonate supplement should probably not be ingested at the same time as sevelamer to avoid competition between bicarbonate and phosphate for binding to the resin.

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Kidney function itself, and not cystatin C, is correlated with height and weight

To the Editor: In a recent issue of this journal [1], Knight et al presented data from a large population study on factors influencing serum cystatin C concentrations in adults. After adjustment for uncorrected creatinine clearance (in mL/min), multivariate linear regression analysis yielded a significant positive correlation of cystatin C with age, male gender, weight, height, cigarette smoking, and C-reactive protein (CRP) levels.

Traditionally, glomerular filtration rate (GFR) is normalized to 1.73m² body surface area (BSA) [2], which is of particular importance in growing subjects. Therefore, almost all papers correlating cystatin C with a gold-standard GFR were indexed to BSA. When looked at, anthropomorphometric data did not influence the correlation between GFR normalized for BSA and serum cystatin C [3].

It is therefore not surprising that Knight et al [1] identified height and weight—the chief determinants of BSA—as confounding variables for both cystatin C and creatinine when studying correlation with uncorrected GFR. It would be interesting to see which effect normalization of creatinine clearance to BSA has on the other correlations reported. Their large cohort is certainly particularly well suited to identify factors weakly influencing serum cystatin C concentration that have been missed so far.

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