Dr. Gotloib raises three points in his letter. These are (i) being cited incorrectly, (ii) no mention of lipid peroxidation and (iii) the presence of carbonyl stress in patients treated with icodextrin. First, the citation was based on what I considered to be a logical explanation of Dr. Gotloib’s findings, that is the possibility of intracellular accumulation of icodextrin degradation products. It now appears that he meant other organic osmole. If so, I regret my statement on this issue and apologize. Second, I am not convinced by the presented data on lipid peroxidation. These are all based on MDA concentrations in effluent, which increase during the 4-h dwell in parallel to total protein. This suggests a peritoneal transport of MDA from the circulation, and is quite different from the MDA already present in fresh 4.25% glucose dialysis fluid. The issue could have been solved easily, had MDA concentrations in plasma been provided in the paper.

The third point is on the suggestion that icodextrin would cause oxidative stress in humans. The paper quoted by Dr. Gotloib shows that a fresh icodextrin solution has a low capacity to generate advanced glycosylation end-products and that the influx of reactive carbonyl compounds from the circulation was not different from their findings with an amino acid-based solution.

Based on the above reasoning, I can see no convincing evidence that icodextrin would cause oxidative stress by lipid peroxidation or carbonyl stress.

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Reply

We appreciate the careful reading of our recent publication [1] by Glassock et al., and we are happy that they agree on the need for a system to identify individuals with chronic kidney disease (CKD). Nonetheless, the authors state in the first paragraph of their letter that screening for albuminuria to delay disease progression is not cost-effective in the absence of knowledge of hypertension and diabetes status. A reference is made to the study by Hoerger et al. [2]. This statement seems to be in contrast with studies of our group that do indicate cost-effectiveness of a full-population screening [3,4]. Of note, the study by Hoerger et al., as well as our studies, indicates that the critical factor for cost-effectiveness of screening for microalbuminuria is the costs involved with the screening procedure. The difference between both studies is that, in the study by Hoerger et al., costs are taken into account for each screening participant for the test itself ($15.32) and also the much higher costs for a visit to a general practitioner (GP).