that in vitro acidosis inhibits chloride reabsorption in TALHs.2 The decrease in chloride reabsorption in the TALHs causes sodium loss and hypercalciuria seen in dRTA. Another recent paper in Kidney International3 reported that responses to furosemide are blunted in dRTA patients, suggesting that sodium reabsorption in the TALHs is impaired in dRTA.

Even in the same range of urine pH, the urinary ammonium concentration of dRTA patients is much lower than in other types of acidosis,4 and the low interstitial pH can also explain the low NH4+ excretion rate. NH4+ reabsorption in the TALHs is suppressed by the mechanisms described above, and the low pH in the interstitial space results in a low NH3/NH4+ ratio and further decreases the concentration of NH3, which diffuses into the urinary space and is trapped as NH4+.


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Antiproteinuric effects of cilnidipine


To the Editor: We read with interest the CARTER trial,1 but we would like to see a number of issues clarified. First, does the statement that an investigator monitored randomization in order of the entry of the subjects in each institute mean that allocation to randomized treatment depended on the order of presentation of patients? Systematic rather than random allocation might explain why the cilnidipine group compared with the amlodipine group included 19 more patients (11.9%). Second, it is remarkable that the authors did not give more information on the quality control of the primary outcome measure, the urinary protein-to-creatinine ratio (UPCR). This multicenter trial ran over 4 years. Were the measurements done in a central laboratory? What was the interassay variability of 10% would approximate to the observed differences. Third, although UPCR was not normally distributed, it was statistically analyzed assuming normality. Did the results remain consistent after transformation to approach a normal distribution? Finally, the most important issue, not reported in the paper, is whether the observed changes in UPCR were due to the nominator (protein), the denominator (creatinine), or both?


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Response to ‘Antiproteinuric effects of cilnidipine’


We are grateful for the opportunity to respond to the comments of Dr Staessen. First, as mentioned in the interventions section of the methods in our manuscript,2 randomization was done in order of the entry of the subjects in each institute. Because only one investigator in some institutes participated in this study, the allocation was done at random in order that a number of the two group subjects equal out for every 10 subjects in each institute. Thus, even in the institute where more than 10 subjects participated, the number of subjects was often uneven. Second, this trial was an independent study of disinterested physicians and done as part of routine care, and the individual payment was done by individual patients. Thus, the measurement was not done in a central laboratory and the quality control of primary outcome measure was not done. We used the average of two consecutive measured values of urinary protein-to-creatinine ratio (UPCR) during a 4-week observation period before the treatment, but measured UPCR once at each follow-up period. Despite this limitation, percentage of changes of UPCR were consecutively (three times) lower since early period (3 months) of the treatment in cilnidipine group as compared with amlodipine group. Thus, it is suggested that the add-on therapy of cilnidipine can suppress urinary protein to a greater extent than that of amlodipine. Third, we reanalyzed data of UPCR as common logarithm. As a result, baseline value was not different between cilnidipine and amlodipine groups (mean ± s.d.: 3.10 ± 0.39 vs 3.10 ± 0.34, NS), but logarithm