different periods in the study. The values in the text are the mean of all PTH, calcium and phosphorus measurements obtained during the whole period of 9 months, before (PTH 590.4 pg/ml) and 9 months after the treatment with cinacalcet (PTH 414.2 pg/ml). In the Table, the PTH, calcium and phosphorus at time 0 (just before starting cinacalcet) and after 9 months of treatment with cinacalcet are shown.

Control of secondary hyperparathyroidism (SHPT) in CKD patients is frequently difficult [1,2] and it has important implications for patient morbidity and mortality [3,4]. Fortunately, in recent years, there have been important advances with the appearance of new drugs which represent a clear benefit in the treatment of this problem. The ability of cinacalcet to reduce PTH secretion, along with reductions in the serum calcium, phosphorus and calcium–phosphorus product, provides an alternative to the traditional treatment paradigm, and should be an addition to our therapeutic strategy in the management of SHPT. In addition to its effects on PTH and mineral metabolism, cinacalcet has demonstrated favourable effects on important clinical outcomes: combined results from four clinical trials (cinacalcet randomization) led to significant reductions in the risk of parathyroidectomy, fractures and cardiovascular hospitalization, along with improvements in self-reported physical function and diminished pain [5]. On the other hand, the pleiotropic effects of vitamin D have recently been highlighted; vitamin D regulates cell proliferation and differentiation, modulates the immune system, and is involved in several endocrine systems. Vitamin D metabolites therapy provides survival benefits and, certainly, paricalcitol has demonstrated good results in terms of PTH, calcium and phosphorus control, as well as a reduced mortality risk when comparing treated and untreated patients [6].

The optimal treatment for SHPT in HD patients is yet to be established. It is possible that the combined use of both drugs (cinacalcet and paricalcitol) might offer some therapeutic advantages: we should further investigate the use of these drugs (either alone or in combination) in different patient populations and see the outcomes in terms of mortality, morbidity and costs, in order to establish the most efficient treatment in each situation.

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**Significance of endotoxaemia in chronic kidney disease**

Sir,

I read with great interest the recent article of Goncalves and colleagues [1] on the association between mild endotoxaemia and fluid overload or inflammatory markers in patients with chronic kidney disease (CKD). They concluded that mild endotoxaemia was linked to fluid overload although there was no association between circulating endotoxin concentrations and systemic inflammation.

Recently, there is an increasing evidence to suggest that a leaky bowel wall may lead to translocation of bacteria and/or endotoxin, which may be an important stimulus for inflammatory cytokine activation, leading to the progression of chronic heart failure [2]. Similarly, endotoxin translocation could be associated with the deterioration of renal function in patients with CKD. This hypothesis provides a plausible mechanism for the downward systemic course that commonly occurs in patients with end-stage renal disease, in which bowel wall oedema, mesenteric hypoperfusion, or both, are likely.

The presence of endotoxin in minute amounts in blood was reported in 80% of healthy individuals [3]. Mild to modest endotoxaemia has been observed after elective abdominal surgery [4], strenuous exercise [5] and in patients with liver cirrhosis [6]. Usually, the degree of endotoxaemia is not related to the circulating cytokine or acute-phase protein concentrations. As written in the discussion of the article, endotoxin concentrations (0.16–1.4 ng/l) in patients with CKD are too low to produce systemic inflammatory response. Moreover, the value of 1.4 pg/ml (1.4 ng/l), which is the minimum detection limit of the LAL assay used in the study, exceeds almost all of the data.

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Reply

Sir,

It was interesting to read the letter to the editor commenting on our manuscript recently published in Nephrology, Dialysis and Transplantation [1]. The authors of the letter kindly recognize the importance of our contribution to the understanding of potential causes of the inflammatory response observed in chronic kidney disease (CKD) patients. They also comment on our findings of the lack of association between endotoxaemia and the levels of inflammation markers, which we will briefly discuss.

Initially, it must be emphasized that our study is a clinical observational investigation that was not controlled for many factors. Therefore, as clearly stated in the discussion of our article, the aim of the study was the establishment of associations, and could not safely enter the field of mechanisms.

In the last part of the letter, the authors correctly observed that most of the values of plasma endotoxin presented in our study appear to be below the detection limit of the method utilized. This was due to an error in the conversion of the units, which will be acknowledged in errata. In the results section, values should have been presented in ng/ml instead of ng/l as presented in the article. The corrected results indicate that most patients in our study present levels compatible to what is considered mild to moderate endotoxaemia. Finally, with respect to our statement, that the lack of correlation between levels of endotoxin and inflammation markers observed in our study could be explained by insufficient amounts of circulating endotoxin, we believe that it is impossible at the moment to present a definitive answer. Although most published data in other populations have not shown a significant correlation between inflammation markers and endotoxin levels [2,3], data on healthy individuals [4] show a significant correlation between endotoxaemia and C-reactive protein after ultra-endurance exercise, in levels (5-15pg/ml) much lower than observed in our CKD patients. Furthermore, raised concentrations (in similar levels when compared with our findings) of endotoxin and cytokines are found in patients with oedematous chronic heart failure, which are normalized after diuretic treatment [5]. In the search for causes of inflammation activation in the progression of CKD, further studies with adequate design will need to address these issues.

Conflict of interest statement. None declared.

Sir,

Moriya et al. [1] demonstrate that weekly averaged blood pressure recorded at home and in the dialysis unit is a better correlate of target organ damage in haemodialysis patients [1]. I would like to point out, contrary to the authors’ suggestion, that I used home BP monitoring for 2 days. I reported self-recorded home BPs three times a day over one week. I also discussed in a review why weekly averaged values at home would assist physicians in managing home BP. Including dialysis unit BPs in the calculations average value at home would assist physicians in managing home BP. For instance, Moriya et al. [1] show a significant correlation between endotoxaemia and C-reactive protein after ultra-endurance exercise, in levels (5-15pg/ml) much lower than observed in our CKD patients. Furthermore, raised concentrations (in similar levels when compared with our findings) of endotoxin and cytokines are found in patients with oedematous chronic heart failure, which are normalized after diuretic treatment [5]. In the search for causes of inflammation activation in the progression of CKD, further studies with adequate design will need to address these issues.

Conflict of interest statement. None declared.

Sir,

I believe that Moriya et al. confirm the utility of home blood pressure monitoring in Japanese haemodialysis patients. The type of home BP monitor they used...