for prolonged periods [4]. It is therefore very difficult to determine when calcium oxalate crystals are the cause or the result of acute oliguric renal failure. To ascertain a link between calcium oxalate crystals and acute tubular necrosis, we undertook an audit of the renal biopsies performed at Gloucestershire Royal Hospital between February 1997 and August 2007. There were a total of 855 renal biopsies done in that period. We relied on retrospective review of the pathology reports to identify renal biopsies with both ATN and crystals. In total, there were 20 cases of ATN. In 10 of the cases, the cause of ATN was not identified, in 2 cases the cause was volume depletion and in 3 cases the cause was the use of non-steroidal anti-inflammatory drugs (NSAIDs). Three further cases of ATN were associated with liver disease, one with heart failure and one with sepsis. There were 11 renal biopsies with crystals deposition. In six of them, the crystals were not identifiable. There was one case of cholesterol crystals, one case of urate crystals and one case of calcium phosphate crystals due to the use of fleet for bowel preparation prior colonoscopy and two cases of calcium oxalate crystals. Both cases of calcium oxalate crystals also had unexplained ATN and the patients were taking orlistat at the time of presentation. One of these patients died; the other had partial recovery of his renal function after a short period on dialysis. His renal function remained stable despite continuing taking orlistat. Our audit does not directly support an association between the use of orlistat, oxalate crystalluria and ATN. However, the retrospective use of biopsy reports is a limitation in drawing conclusions. Patients with pre-existing renal disease, undiagnosed mild forms of primary hyperoxaluria or secondary hyperoxaluria, who also take orlistat, may be predisposed to developing acute oxalate crystalluria if their renal function deteriorates for other reasons. The identification of high-risk patients treated with orlistat and the regular monitoring of their renal function might reduce the theoretical risk of renal failure due to acute oxalate nephropathy.

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Editorial Note: Dr Courtney et al declined the invitation to reply to this letter.

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Negative outcome studies in end-stage renal disease: how dark are the storm clouds?

The Salt Factor

Sir,

It is incredible that in 2008, an editorial review by such learned gentlemen as Covic and Goldsmith fails to discuss the ‘salt factor’ in their gloomy attitude towards the prognosis of ESRD patients [1]. It is even more astonishing that this omission occurred after KDOQI guidelines revised their advice on salt intake and recommended a salt-restricted intake for all CKD patients [2]. Clearly, they are not aware of the outstanding reductions in CVD and mortality reported by Nancy Cook in a 10-year follow-up of prehypertensive, but otherwise normal, population maintained on a low-salt diet [3]. Finally, they do not cite the two centres that have reported the best long-term survival data on dialysis in the world; both have employed obsessive salt restriction as a main part of their programme [4,5]. Even given the long gestation period of the review and its revision, one must question whether the review process was adequate for this editorial comment to see the light of day. One must conclude that, given their extensive conflict of interest declarations, there can be little commercial benefit from recommending a low-salt diet.

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Reply

Sir,

We thank Dr Stanley Shaldon for his interest in our article [1]. He is of course quite correct to point out that we