The authors used the correct control preparation which was treated identically, with the exception that glucose was omitted. In our lab, we recently prepared AGEs by incubating endotoxin-free BSA 300 mg/ml at 37°C for 6 weeks with D-glucose (90 g/l) in a 0.4 M phosphate buffer at pH 7.6. Interestingly, we saw an increase in CML and CEL in our control preparation, which was treated identically but without the addition of glucose, when compared with non-incubated BSA (Figure 1). It would have been interesting to use non-incubated endotoxin-free BSA as a control as well.

We conclude that Chang et al. propose a novel mechanism that could be involved in disturbances of sodium balance in diabetic nephropathy, i.e., an AGE-induced increase in expression of the epithelial sodium channel mRNA and protein, with enhanced sodium uptake in renal CCD cells. To substantiate this interesting hypothesis, it would be important to exclude endotoxin mediated effects.

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Note: Dr Chang et al. have been invited to reply to this letter, but we did not receive an answer.

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Treatment with corticosteroids does not seem to benefit nephrogenic systemic fibrosis

Sir,

Nephrogenic systemic fibrosis (NSF) is a formerly unknown disease, which has become more and more recognized. It affects patients with kidney failure and, to a great extent, mimics systemic sclerosis [1]. An association between NSF and magnetic resonance image with gadolinium-based contrast agents has been suggested [2]. Herein, we report a case of NSF, in which corticosteroids were administered at the very beginning of the disease.

A 62-year-old male haemodialysis patient abruptly developed symmetrical painful, oedematous swelling of the skin on his fingers and palms. In addition, the swollen tissue around the palmar flexor tendons resembled tendovaginitis. The range of motion of the fingers was severely limited and painful. The patient received NSAIDs, but the symptoms persisted. Therefore, and because the disease was believed to be an inflammatory condition, the patient was treated with 50 mg prednisolone daily. However, swelling and pain did not improve. In the following weeks, the dosage of prednisolone was slowly tapered and finally discontinued after 2 months. As time passed, the oedematous swelling gradually resolved, but in parallel, the tissue became more and more fibrotic. After 1 year, the patient had contractions of his fingers, toes, elbows and knees (Figure 1). Given the course of the disease, we retrospectively recognized this condition to be NSF.

Notably, 7 days before the start of symptoms, the patient had an MR angiography. The diagnosis was finally verified with a skin biopsy. The histological examination revealed large amounts of fibroblastic tissue, containing numerous CD34-positive fibroblasts [3]. NSF is a debilitating and sometimes fatal disease, affecting the skin, muscle, and internal organs [1,2]. These days, no consistently successful treatment exists. Because of their anti- oedematous and anti-fibrotic properties, corticosteroids may theoretically be helpful in the treatment of NSF. Given the ineffectiveness of prednisolone in our patient with regard to swelling, pain and progression of the disease, we believe that corticosteroids neither ameliorate the symptoms nor are they of benefit in the evolution of NSF.

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Fig. 1. Contractions of the fingers in nephrogenic systemic fibrosis.