Dear Sir,

We have previously reported that cysts in rat autosomal recessive polycystic kidney (ARPK) originate in the collecting duct [1]. In the present study, the renal tissues of 8-month-old rats with ARPK and normal rats were examined by electron microscopy after PEI staining, to clarify the pathogenesis of ARPK. PEI staining was performed by the immersion method described by Schurer et al. [2].

Anionic sites in the basement membrane of the collecting duct were sparsely observed, centering on the epithelial side in the normal rats (fig. 1 a). On the other hand, in the ARPK rats, anionic sites were markedly increased and present in the entire layer of the tubular basement membrane (TBM) of the cysts (fig. 1b).

Electron microscopy after staining with ruthenium red in rats with renal cystic changes induced by the administration of 2-amino-4,5-diphenylthiazole HC1 (DPT-treated rats) showed a decrease in anionic sites in the cyst TBM [3]. Another study by the fluorescent antibody technique using anti-heparan sulfate proteoglycan in patients with autosomal dominant polycystic kidney and DPT-treated rats showed decreased staining in the cyst TMB.
[4]. These findings suggest the involvement of impaired synthesis of heparan sulfate proteoglycan in cyst formation. However, Ojeda et al. [5] induced cysts due to dilatation of the collecting duct by administration of methylprednisolone and observed thickening of the cyst epithelium and increased anionic sites. In this study, anionic sites in the cyst TMB of ARPK rats were markedly increased as was reported by Ojeda et al. This suggests that abnormal turnover of heparan sulfate proteoglycan in the cyst epithelium is involved in the development of rat ARPK. We speculate that cysts in rat ARPK originate in the collecting tube and are caused by changes in compliance induced by structural abnormalities in the basement membrane.

References