Dear Sir,

Complement receptor (CR1) [1] has been found to play an important role in the treatment of immune complex in vivo [2]. Miyakawa et al. [3] have reported a high incidence of CR1 deficiency in systemic lupus erythematosus patients. Both congenital or genetic factors and acquired factors have been reported as causes of systemic lupus erythematosus [4]. We investigated primary glomerular disease by the immune adherence hemagglutination method and found CR1 deficiency in 2 cases with mesangiocapillary glomerulonephritis (MCGN).

Case 1 was a 26-year-old female with nephrotic syndrome. At first renal biopsy, she was diagnosed as having mild mesangial proliferative glomerulonephritis which was once improved by steroid therapy, but, about 8 years later, nephrotic syndrome recurred. At second renal biopsy, she was diagnosed as having MCGN. She responded to steroid therapy at any stage of nephrotic syndrome, and her urinary protein level decreased to below 1 g/day, but did not return to negative. According to the results of other examinations, antinuclear antibody was found to be always weakly positive, but no abnormalities were noted in other investigations, including renal function tests. CH50 was 54 U and C3, 84 mg/dl.

Case 2 was a 17-year-old female who had nephrotic syndrome during the observation course. At first renal biopsy, MCGN was found focally. At second renal biopsy, performed 4 years later, she was diagnosed as having MCGN. She revealed persistent hypocomplementemia (C3 11 mg/dl; C4 10.6 mg/dl; CH50 12 μ), and C3 NeF was noted.

As shown in figure 1, family background studies carried out on these 2 patients demonstrated CR1 deficiency in either the father or the mother, and their CR1 deficiency was regarded as
genetic. Interestingly, the C3 value in the parent who had CRI deficiency corresponded to 2 or more times the normal level. Possible

Positive □ O Negative ■ #
nd = not done
Fig. 1. Family studies on CRI and C3 measurements (% normal).
Genetic CRI deficiency was inferred in these cases, and it was interesting to note that C3 NeF and antinuclear antibody (as autoantibody) were detected.
CRI deficiency appeared to have a certain significance or relationship to the disease occurring in these 2 patients. It is necessary to examine in detail the incidence of CRI deficiency in MCGN.
References