Dear Sir,

Although patients with diabetes mellitus frequently show high levels of serum IgA, the etiology of such a phenomenon is still unclear. Triolo et al. [1] discussed the possible role of IgA-containing immune complexes in the pathogenesis of late complications of diabetes including nephropathy. They also reported that IgA1- and IgA2-immune complexes in sera are equally distributed in type 2 diabetes mellitus according to a solid-phase anti-C3(Fab')2 enzyme immunoassay [2]. We recently examined the levels of IgA1 and IgA2 in the sera of 18 type 2 diabetic patients with or without nephropathy and 6 age-matched healthy adults using single radial immunodiffusion (SRID). Patients and healthy adults who had infections within 2 months before the present study were excluded. Total IgA, IgM and IgG in serum samples were measured by ordinary laser nephelometry. Radial immunodiffusion (RID)-specific instruments were purchased from The Binding Site Inc., San Diego, Calif., USA, for determining the levels of serum IgA1 and IgA2. The results of serum IgA and IgA1 in type 2 diabetic patients were significantly higher than those in healthy adults (p < 0.01). The levels of serum IgA or IgA1 in patients with diabetic nephropathy were higher than those in diabetic patients without nephropathy. The levels of serum IgM in type 2 diabetic patients were significantly lower than those in healthy adults (p < 0.05). However, there was no significant difference in the levels of IgG and IgA2 between type 2 diabetic patients with or without nephropathy and healthy adults (table 1).

The explanation of this phenomenon is not very clear at present. Triolo et al. [1] re-
Mean ± lSD. *p < 0.01,**p < 0.05

ported a close association among hyper-IgA, secretory IgA, IgA/secretory component-containing immune complexes and microangiopathy such as nephropathy in diabetic patients. Although IgA-producing plasma cells in bone marrow consist of 88% IgA1 and 12% IgA2, those in intestinal mucosal membrane consist of 65% IgA1 and 35% IgA2 as described by Conley and Delacroix [3]. It is generally considered that IgA1 derived from the mucosal membrane easily develops into a polymerized form. Furthermore, 80-87% of IgA in the sera of diabetic patients are polymeric as described by Triolo et al. [4]. It appears that IgA1 may play an important role in the increase of total IgA in the sera of type 2 diabetic patients with or without nephropathy although its origin is still obscure. Another possibility might involve the catabolic characteristics of serum IgA1 in patients with diabetes mellitus.

References