*Letters to the editor***Hypomagnesaemia in IDDM patients with microalbuminuria and clinical proteinuria**

Dear Sir,

Further to your recent correspondence about the frequency of hypomagnesaemia in diabetic patients and its clinical significance [1, 2], we have recently measured total serum magnesium concentrations in IDDM patients with microalbuminuria and clinical proteinuria. The latter group have a markedly increased frequency of cardiovascular disease and mortality, and are hypertensive and insulin resistant compared to IDDM patients without proteinuria.

These are features which may be associated with hypomagnesaemia [3]. Moreover, since microalbuminuria predicts the development of clinical proteinuria, it is also possible that hypomagnesaemia is more prevalent in these patients than in those without proteinuria.

We studied 16 IDDM patients with normal albumin excretion ($< 20 \mu\text{g}/\text{min}$), 16 patients with microalbuminuria ($> 20 < 200 \mu\text{g}/\text{min}$) and 16 with clinical proteinuria ($> 200 \mu\text{g}/\text{min}$). Patients were matched for age, duration of diabetes and glycated haemoglobin percentage (Table 1). Forty non-diabetic subjects of similar age were used as the reference group.

The mean serum magnesium concentration was significantly lower in each of the diabetic groups with differing renal function, than in the normal subjects. However, there was no significant difference in the serum magnesium levels amongst the diabetic groups (Table 1). None of the patients in any of the diabetic groups had a serum magnesium level lower than the mean minus 2SD for the non-diabetic subjects (0.61 mmol/l).

Although it is possible that measurements of serum ionised or intracellular free magnesium concentrations [4], rather than total serum magnesium, may reveal a more subtle difference between the patients with normoalbuminuria, microalbuminuria

and clinical proteinuria, the present data suggest that gross hypomagnesaemia is not a feature of microalbuminuria or clinical proteinuria and the decrease in the mean levels is probably not an important risk factor for the development of cardiovascular disease in these patients.

Yours sincerely,

J. C. Pickup, G. D. Chusney, M. A. Crook, G. C. Viberti

References

1. de Valk HW, Haalboom JRE, Struyvenberg A (1992) Hypomagnesaemia and type 2 (non-insulin-dependent) diabetes mellitus. *Diabetologia* 35: 904 (Letter)
2. Schnack C, Schernthaner G (1992) Hypomagnesaemia and type 2 (non-insulin-dependent) diabetes mellitus. *Diabetologia* 35: 904–905 (Letter)
3. Rude RK (1992) Magnesium deficiency and diabetes mellitus. Causes and effects. *Postgrad Med* 92: 217–223
4. Resnick JM, Altura BT, Gupta RK, Laragh JH, Alderman MH, Altura BM (1993) Intracellular and extracellular magnesium depletion in type 2 (non-insulin-dependent) diabetes mellitus. *Diabetologia* 36: 767–770

Selection of control subjects in genetic studies of diabetes mellitus

Dear Sir,

Pozzilli and colleagues [1] draw our attention once again to the problems of assembling groups for the study of genetic associations in Type 1 (insulin-dependent) diabetes. The importance of ethnic matching is irrefutable but we would suggest that difficulties are not limited to the control group – significant problems may arise in the collection of affected subjects.

There is considerable evidence that both Type 1 and Type 2 (non-insulin-dependent) diabetes are heterogenous categories

Corresponding author: Dr. J. C. Pickup, Division of Chemical Pathology, United Medical and Dental Schools, Guy's Hospital, London SE1 9RT, UK

Table 1. Clinical features and serum magnesium concentrations in IDDM patients subjects with normo- or microalbuminuria and clinical proteinuria, compared to normal subjects

	Age (years)	Sex (M/F)	Diabetes duration (years)	GHb (%)	Serum Mg^{2+} (mmol/l)
Non-diabetic patients ($n = 40$)	41.9 ± 10.4	19/21	–	–	1.01 ± 0.20
Normoalbuminuria ($n = 16$)	38.0 ± 9.0	11/5	20.5 ± 5.0	9.3 ± 1.3	0.79 ± 0.08^a
Microalbuminuria ($n = 16$)	38.7 ± 8.0	14/2	23.9 ± 7.0	9.4 ± 1.0	0.82 ± 0.09^a
Clinical proteinuria ($n = 16$)	37.9 ± 8.4	7/11	24.4 ± 5.9	9.3 ± 1.6	0.83 ± 0.11^a

^a $p < 0.001$ vs non-diabetic subjects. GHb, glycated haemoglobin. Results are mean $\pm$ SD