References

1. Pickup JC, Crook MA (1998) Is Type II diabetes mellitus a disease of the innate immune system? Diabetologia 41: 1241–1248
2. Ross R (1999) Atherosclerosis – an inflammatory disease. N Engl J Med 340: 115–126
3. Yudkin JS, Panahloo A, Stehouwer C, Emeis JJ, Bulmer K, Mohamed-Ali V, Denver AE (2000) The influence of improved glycaemic control with insulin and sulphonylureas on acute phase and endothelial markers in Type II diabetic subjects. Diabetologia 43: 1099–1106
4. Albertini J-P, Valensi P, Lormeau B, Aurousseau M-H, Ferriere F, Attali J-R, Gattegno L (1998) Elevated concentrations of soluble E-selectin and vascular cell adhesion molecule-1 in NIDDM. Effect of intensive insulin treatment. Diabetes Care 21: 1008–1013
5. Baumann H, Gauldie J (1994) The acute phase response. Immunol Today 15: 74–80
6. Hamon Y, Luciani M-L, Becq F, Verrier B, Rubartelli A, Chimini G (1997) Interleukin-lβ secretion is impaired by inhibitors of the atp binding cassette transporter ABC1. Blood 90: 2911–2915
7. Nishikawa T, Edelstein D, Du XL et al. (2000) Normalizing mitochondrial superoxide production blocks three pathways of hyperglycaemic damage. Nature 404: 787–790
8. Ebeling P, Teppo A-M, Koistinen HA et al. (1999) Troglitazone reduces hyperglycaemia and selectively acute phase serum proteins in patients with Type II diabetes. Diabetologia 42: 1433–1438

Comments to: Tarnow L, Kjeld T, Knudsen E et al. (2000) Lack of synergism between long-term poor glycaemic control and three gene polymorphisms of the renin angiotensin system on risk of developing diabetic nephropathy in Type I diabetic patients. Diabetologia 43: 794–799

To the Editor: Chronic hyperglycaemia is the most important risk factor in the development of diabetic complications including diabetic nephropathy. Since not all patients with long-term, poor metabolic control develop nephropathy, interaction between poor metabolic control and putative genetic risk factors for nephropathy is clearly a major contributor to its aetiopathogenesis. Many of the studies on this interaction could be criticised for using short-term observation of glycated haemoglobin, which might not reflect glycaemic control over several years, let alone over decades.

The recent study of Tarnow et al. [1] showed a significant correlation ($r = 0.74$, $p < 0.001$) between a random and long-term (13.5 years) measurement of glycated haemoglobin within the usual therapeutic range (7–11 %) in a large number of adult diabetic patients. This observation, in accordance with an earlier study of Barnas et al. [2] suggests that a single random measurement of glycated haemoglobin offers a valid estimate of glycaemic control during the preceding decade.

No such studies have been carried out in children. We compared a random glycated haemoglobin value with the average of 15 (range 5 to 23) previous measurements (HPLC, Bio-Rad Diamat) made over a period of 5 years in 190 diabetic children and adolescents with a mean duration of diabetes of 9.2 ± 5.6 (SD) years. In this cohort, a significant correlation ($r = 0.75$, $p < 0.001$) was found (Fig. 1). Our observation shows that despite the possibly larger fluctuation in glycaemic control in children compared with adults [3], a significant correlation exists between a recent random measurement and a long-term measurement of HbA_{1c}. Single random glycated haemoglobin values could therefore be used as surrogate markers for long-term metabolic control in the paediatric age group in retrospective studies similar to that of Tarnow et al.

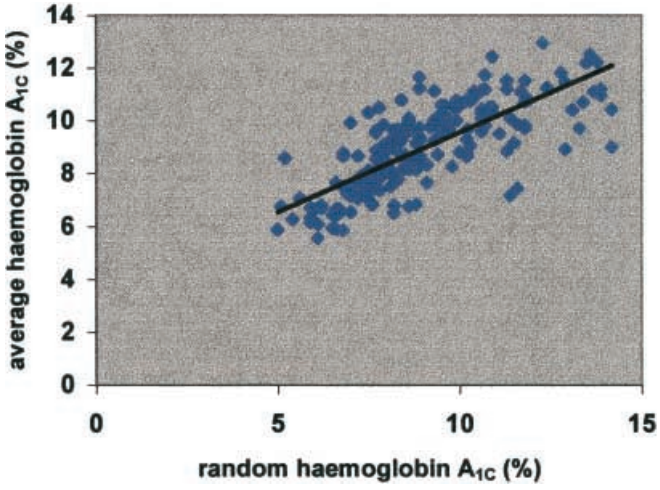

Abb. 1. See text

G. Soltész and B. Rózsai

Corresponding author: G. Soltész, Pécs University, Faculty of Medicine, Department of Paediatrics, H-7623 Pécs, József A. u. 7., Hungary

References

1. Tarnow L, Kjeld T, Knudsen E et al. (2000) Lack of synergism between long-term poor glycaemic control and three gene polymorphisms of the renin angiotensin system an risk of developing diabetic nephropathy in Type I diabetic patients. Diabetologia 43: 794–799
2. Barnas U, Schmidt A, Illievich A et al. (1997) Evaluation of risk factors for the development of nephropathy in patients with IDDM: insertion/deletion angiotensin converting enzyme gene polymorphism, hypertension and metabolic control. Diabetologia 40: 327–331
3. Mortensen HB; Vestermark S, Kastrup KW (1982) Metabolic control in children with insulin dependent diabetes assessed by haemoglobin A_{1C}. Acta Paediatr Scand 71: 217–222