Fig. 2. Correlation between glycosylated LDL (%) and plasma glucose (mmol/l) at time of sampling. Circled points (○) represent values for diabetic patients and open points (•) represent non-diabetic control values. The Pearson correlation coefficient for all patients is $r = 0.72$

References

1. Lyons TJ, Baynes JW, Patrick JS, Colwell JA, Lopes-Virella MF (1986) Glycosylation of low-density lipoprotein in patients with Type 1 (insulin-dependent) diabetes: correlations with other parameters of glycaemic control. *Diabetologia* 29: 685–689
2. Jack CM, McAlister VP, Sheridan B, Stout RW, Kennedy L (1986) Non-enzymatic glycosylation of low-density lipoprotein (LDL). *Diabetologia* 29: 552(a)
3. Havel RJ, Eder JA, Bragdon JH (1955) The distribution and chemical composition of ultracentrifugally separated lipoproteins in human serum. *J Clin Invest* 34: 1345–1353
4. Johnson RN, Metcalf PA, Baker JR (1982) Fructosamine: a new approach to the estimation of serum glycosylprotein. An index of diabetic control. *Clin Chem Acta* 127: 87–95
5. Brown MS, Goldstein JL (1983) Lipoprotein metabolism in the macrophage. *Ann Rev Biochem* 52: 223–261
6. Lyons TJ, Klein RL, Baynes JW, Lopes-Virella MF (1986) Stimulation of cholesteryl ester synthesis in human monocyte-derived macrophages by LDL isolated from diabetic patients. *Clin Res* 34: 208A

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Elevated serum uric acid

Dear Sir,

We were very interested in the report of Modan and colleagues, which demonstrated a positive association of serum uric acid with plasma insulin response to an OGTT [1]. The authors concluded that elevated serum uric acid is a feature of hyperinsulinaemia/insulin resistance.

What remains unknown, however, is whether it is the hyperinsulinaemia per se or the insulin-resistant state that is responsible for the elevation in serum uric acid. In this regard, we recently studied 5 normal women by the hyperinsulinaemic-euglycaemic clamp technique for 12 h [2]. During the clamp mean ($\pm$ SE) serum insulin was 11.25 ± 1.80 nmol/l, while serum glucose was maintained at 6.41 ± 0.28 mmol/l. During the insulin infusion serum uric acid levels progressively fell from a median (and range) value of 286 (149–381) μ mol/l at 0 h to 226 (125–327) μ mol/l at 12 h ($p < 0.05$ by two-tailed Wilcoxon signed rank test).

Our data suggest that hyperinsulinaemia decreases serum uric acid, and that it is the insulin-resistant state which is responsible for the elevated serum uric acid levels in the study by Modan and colleagues. This explanation would also be consistent with that study's observation that newly-diagnosed diabetic patients tended to have elevated serum uric acid levels despite a reduction in sum insulin levels [1].

Yours sincerely,

J. E. Nestler, J. N. Clore and W. G. Blackard

References

1. Modan M, Halkin H, Karasik A, Lusky A (1987) Elevated serum uric acid – a facet of hyperinsulinaemia. *Diabetologia* 30: 713–718
2. Nestler JE, Clore JN, Failla ML, Blackard WG (1987) Effects of extreme hyperinsulinaemia on serum levels of trace metals, trace metal binding proteins, and electrolytes in normal females. *Acta Endocrinol (Copenh)* 114: 235–242

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Familial diabetes mellitus

Dear Sir,

In a recent issue of *Diabetologia*, Bodansky and Kelly [1] describe a diabetes syndrome characterised by apparent autosomal dominant inheritance, variable clinical severity, and insulinopenia. In two newly diagnosed patients, islet cell autoantibodies were absent. As well, evidence of an autoimmune aetiopathogenesis was not supported by HLA typing as only two of the seven patients that were HLA typed carried a high risk allele for Type 1 (insulin-dependent) diabetes, DR4. No patient carried DR3.

We were struck by the similarity of this familial diabetes syndrome to the atypical diabetes syndrome that we have recently described in Black Americans [2]. Our patients were initially diagnosed as having Type 1 diabetes, while months to years later their dependency on insulin waned or disappeared. Our patients similarly lacked islet cell autoantibodies and high-risk Type 1 diabetes alleles, and insulin secretion as measured by C-peptide response to a mixed meal (Sustacal) was intermediate between control subjects and patients with Type 1 diabetes. Atypical diabetes in Black Americans was