The complete correlations between heredity and cordblood autoantibodies are presented in Table 1. Seventeen per cent (14/81) of the remaining children had cordblood autoantibodies and 2 of these 14 had fathers with Type I diabetes. The fraction of children with positive islet autoantibodies was greater among children with diabetic fathers; 29 % (2/7), than among children without heredity for the disease; 16 % (12/74), the difference being non-significant ($p = 0.60$). Paternal heredity for Type I diabetes is still, however, a possible risk factor for early islet cell autoantibodies.

It is worth noting that 16 % of the children later developing Type I diabetes had cordblood autoantibodies against islet cells even in the absence of heredity for the disease among parents.

Yours sincerely,
B. Lindberg, S-A. Ivarsson, Å. Lernmark

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Effect of insulin on apolipoprotein(a) synthesis

Dear Sir,
Neele et al. have reported that apolipoprotein(a) and apolipoprotein B-100 secretion by cynomolgus monkey hepatocytes is modestly inhibited after incubation with insulin in concentrations ranging from 10^4 to 10^6 pmol/l [1]. Such insulin concentrations are approximately 15 to 1500-fold higher than those measured after a glucose challenge in humans. Nonetheless, they state that an insulin concentration of 10^4 pmol/l is low and relevant, considering a half life of insulin about 4 h under the experimental conditions used. They do not, however, show that this assumption is correct. In comparison, others have shown effects on cholesterol uptake by HepG2 cells and rat hepatocytes when insulin is added in a concentration as low as 600 pmol/l [2]. Furthermore, they use an enzyme linked immunoassay that measures the apo(a) moiety of lipoprotein(a) [3] and not free apo(a). Since it is most likely that the assembly of lipoprotein(a) is an extracellular event [4], it is possible that the observed decrease in so-called apo(a) is confounded by a decrease in apolipoprotein B-100. Finally, there is no evidence that insulin decreases plasma lipoprotein(a) in healthy humans, despite the lowering of apolipoprotein B-100 synthesis [5]. For these reasons, it is our opinion that the findings of the in vitro study by Neele et al. cannot be extrapolated to the in vivo situation in humans.

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Yours sincerely,
S. C. Riemens, R. P. F. Dullaart

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Insulin suppresses apolipoprotein(a) synthesis in cultured cynomolgus monkey hepatocytes

Dear Sir,
In their comment on our paper which appeared in Diabetologia 42: 41–44 Riemens and Dullaart conclude that the findings of our in vitro study cannot be extrapolated to the in vivo situation in humans. We fully agree with this statement and have not attempted to make such an extrapolation in our paper. We just suggested that our findings that insulin suppresses apolipoprotein(a) synthesis in primary cultures of cynomolgus monkey hepatocytes could provide an explanation for the increased plasma concentrations of lipoprotein(a) found in patients with insulin dependent diabetes mellitus and its return to normal after treatment with insulin (as stated clearly in the Summary and in the Discussion). The next step is to prove this in turn-over studies in appropriate experimental animals and in humans.

In studies of the metabolic effects of insulin in cell culture experiments, using hepatocytes from different species, the insulin concentrations applied are usually in the range of 1 to 150 nmol/l. In this context the concentration at which we found

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