fall over a 70–80 min interval to 4.7 mmol/l or above (average fall rate $0.19 \pm 0.05 \text{ mmol}^{-1} \cdot \text{min}^{-1}$, lower levels being prevented by prompt glucose infusion).

No normal subjects released growth hormone during the study. In contrast, six of the nine diabetic patients (three Type 1 and three Type 2; Figs. 1 and 2) released growth hormone during or shortly after the fall in glucose concentration. These results suggest that rapidly declining hyperglycaemia may stimulate the release of growth hormone in Type 1 and non-obese Type 2 diabetic patients. This response is not seen in normal subjects. The release of growth hormone is likely to be induced by the rapid fall in glucose. Reaching a threshold level of plasma glucose probably acts as a trigger for growth hormone release, reflecting an upward resetting of the glucostat in diabetic subjects [6], although this cannot be substantiated by these data.

In view of the debated role of growth hormone in the pathogenesis of diabetes mellitus and its chronic complications, the abnormal release of growth hormone during a decrease in plasma glucose level in diabetes may have clinical significance. Intermittent elevations of growth hormone may also contribute to the problems of diabetic control.

Yours sincerely,

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No Linkage Between HLA and Maturity Onset Hyperglycaemia in the Young

Dear Sir,

Platz et al. [1] have recently failed to find evidence for linkage between HLA and maturity onset hyperglycaemia in the young (MOHY) in a large kindred. They mention our previous report [2] which suggested possible linkage with HLA in a similar family we had studied.

We have recently typed for HLA and analysed for association and linkage in ten large MOHY families. Like Platz et al., we have found no evidence for linkage (unpublished observations). Thus, we agree with them that MOHY is probably neither associated nor linked to HLA.

Yours sincerely,
J. Barbosa

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Short-term Changes in Levels of Circulating T₃ and Reverse T₃ in Type 1 (Insulin-Dependent) Diabetic Patients

Dear Sir,

Several studies have shown that Type 1 diabetic patients with severe metabolic upset have low levels of serum T₃ and high levels of reverse T₃ [1, 2]. Recently, Schlienger et al. [3] found low T₃ and high reverse T₃ levels in Type 1 diabetic patients the morning after admission for periodic evaluation and/or adjustment of treatment. They found a correlation between T₃ and glycosylated haemoglobin A_{1c}, suggesting that long-term diabetic control may determine the plasma T₃ levels. No correlation was shown between glycosylated haemoglobin A_{1c} and reverse T₃.

In an earlier study, we emphasised the influence of short-term metabolic control on levels of T₃ and reverse T₃ [4]. We examined the earliest possible phase of development of metabolic control with IV insulin infusion and found a significant decrease in T₃ within 1 h of withdrawal of insulin. After 12 h of insulin withdrawal, a mean 29% decrease in T₃ level was seen (Table 1). The concentration of reverse T₃ was increased significantly after 8 h of insulin withdrawal; an average increase of 37% in reverse T₃ was seen after 12 h of insulin withdrawal (Table 1).

Therefore not only long-term metabolic control, but also short-term changes, seem to be of importance in determining the serum levels of T₃ and reverse T₃ in Type 1 diabetic patients.

Yours sincerely,
S. Madsbad

Table 1. Serum levels of T₃ and reverse T₃ in Type 1 diabetic patients before and after 12 h of insulin withdrawal

	T ₃ (nmol/l)	Reverse T ₃ (nmol/l)	Blood glucose (mmol/l)
Before insulin withdrawal	1.28 ± 0.39	0.30 ± 0.10	7.2 ± 0.5
After insulin withdrawal for 12 h	0.91 ± 0.27	0.41 ± 0.15	17.2 ± 1.3
<i>p</i>	< 0.01	< 0.01	< 0.01

Results expressed as mean ± SEM. Normal range for serum T₃: 1.27–2.50 nmol/l and for serum reverse T₃: 0.17–0.57 nmol/l