contact, public education, and research, the ADA can better meet the needs of this underserved population.

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Is There a Defect in the Hepatic Extraction of Insulin in Patients with NIDDY?

Previously our group has described the syndrome of non-insulin-dependent diabetes of the young (NIDDY) in a migrant Indian population of South Africa.[1–7] Our patients showed delayed and attenuated insulinemic responses to oral glucose and an absent first phase of insulin release in response to intravenous glucose.[1,5,6] In addition, we have shown that obesity does not modulate insulin secretion in Indian patients with NIDDY when obese and nonobese subgroups are matched for age, sex, body mass, and glucose intolerance.[3] It is interesting to note that in the study by Mohan et al.,[8] the insulin responses of the obese patients appear to be significantly greater than those of the nonobese patients, suggesting that perhaps obesity modulates insulin secretion in indigenous Indian patients with NIDDY. However, cognizance should be taken of the fact that their nonobese group had significantly higher fasting plasma glucose levels than their obese groups (217 ± 21 versus 117 ± 19 mg/dl, respectively), and it is well known that hyperglycemia at this level attenuates the insulinemic responses to oral glucose.[9]

We have attempted to determine whether or not a defect in the hepatic extraction of insulin contributed to the hypoinsulinemic responses in these patients. The study patients had onset of diabetes <30 yr of age and belonged to families in which non-insulin-dependent diabetes was transmitted through at least three generations.[5,6] Ten patients and 10 age-, sex-, and weight-matched controls underwent after preparation[1,3] a 100-g oral glucose tolerance test. The patients had no evidence of hepatic or renal dysfunction as determined by clinical examination, serum creatinine, β_2 microglobulin Cr^{51}-EDTA glomerular filtration rates, 24-h urinary protein excretion,[2,4] and conventional biochemical liver function tests. The patients and controls were matched with respect to age, sex, and BMI (27 ± 4.9, 28.6 ± 3.6, $P < 0.5$; 22.5 ± 1.1, 21.6 ± 0.7 yrs. $kg \cdot m^{-2}$, $P < 0.5$). Fasting plasma glucose levels were significantly higher in the patients than in controls (12.0 ± 1.6, 4.3 ± 0.9 mmol/L, $P < 0.002$). In addition, fasting serum insulin and C-peptide levels were significantly higher in the patients with NIDDY than in control subjects (18.2 ± 2.4, 8.5 ± 0.7 μU/ml, $P < 0.02$; and 1.8 ± 0.1, 1.3 ± 0.1 ng/ml, $P < 0.02$, respectively). The insulin and C-peptide responses are depicted in Figure 1 and it is clearly evident that the patients with NIDDY had delayed and attenuated insulin and C-peptide responses. Incremental insulin and C-peptide areas were significantly lower in patients than in control subjects (100.4 ± 31.6 314.5 ± 28.5 μU/ml, $P < 0.002$; and 5.1 ± 0.9, 23.9 ± 3.5 ng/ml, $P < 0.002$, respectively). Although Mohan et al.[8] have reported that several of their patients had higher insulin concentrations in comparison with C-peptide, no explanation was given for these divergent findings. The molar ratios of C-peptide to insulin have been used by previous workers as an index of the hepatic extraction of insulin.[10–12] This ratio is dependent on many factors, as noted by Polonsky[13] in a recent review. The fact that our patients had normal renal and hepatic function prompted us to compute the molar ratios to give us at best a semiquantitative index of hepatic extraction of insulin. There were no significant differences in the molar ratios between the patients and controls throughout the 3-h period of testing (Table 1). Thus, although our study differed from that of Mohan et al., since we used a higher glucose load (100 g) and sampled for a longer period (3 h), the C-peptide responses were similar in both studies, i.e., patients had significantly lower C-peptide responses than did controls.

TABLE 1
C-peptide/insulin ratios in response to a 100 gram oral glucose load

Time (min)	NIDDY (mol)	Controls (mol)
0 m	6.0 ± 0.8	7.2 ± 0.6
30 m	5.2 ± 0.6	3.4 ± 0.4
60 m	3.8 ± 0.7	3.9 ± 0.4
90 m	4.1 ± 0.8	4.3 ± 0.5
120 m	4.4 ± 0.8	4.7 ± 0.6
180 m	5.3 ± 0.8	4.8 ± 0.7

All ratios are $P > 0.5$.

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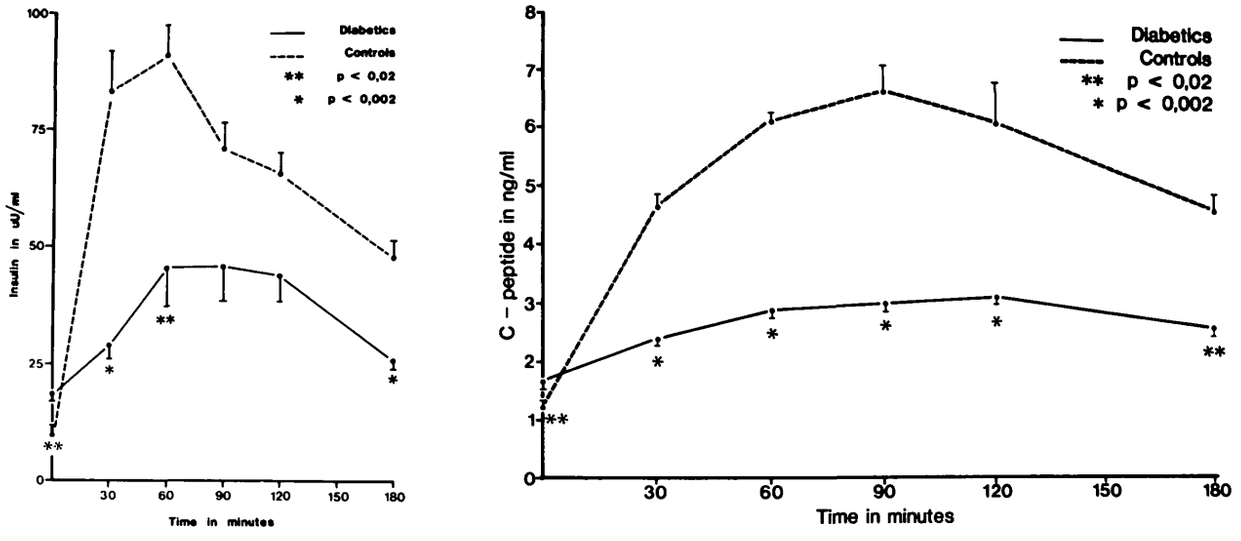

FIG. 1. The insulin and C-peptide response to a 100 gm oral glucose load.

In conclusion, in the discrete syndrome of NIDDY, which is uniformly accepted to be a subset of NIDDM, with the strongest genetic component,[14] insulin deficiency appears to be a major pathogenetic mechanism.

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Insulin Pump Delivery During Parenteral Hyperalimentation

A recent significant advance in patient care is that of total parenteral nutrition (TPN). This strategy is often used in ICU settings for complicated surgical and medical problems associated with catabolic features.

The purposes of TPN are to achieve positive caloric balance and protein sparing. Some people remain euglycemic during this process. Others, who may or may not have diabetes,

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