et al. that this problem does not concern pumps in general. We also agree that the new adaptor for the H-Tron V-100, which is equipped with two small holes and can be closed with a red plug, should eliminate these problems as long as the pump is used according to the manufacturer's specific instructions. We also fully support the recommendation suggested by Prendergast et al. to use the plug for the adaptor holes only when watertightness is needed (in the bath or shower) instead of removing it only when pressure problems are expected. When such instructions are followed, the H-Tron V-100 is at least an equal alternative to other pumps in the market.

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Lipoprotein(a), Apolipoprotein(a) Polymorphism, and Insulin Treatment in Type II Diabetic Patients

The relation between lipoprotein(a) [Lp(a)] and insulin treatment in diabetes is still unclear. Schernthaner et al. (1) did not find significant correlation between daily insulin dosages and Lp(a) levels; Wolffenbuttel et al. (2) did not observe any changes in Lp(a) levels with insulin therapy as compared with previous treatment with oral agents. Recently, Gruden et al. (3) observed higher Lp(a) concentrations in insulin-treated than in oral agent–treated patients.

In order to investigate the relationship between Lp(a) and hypoglycemic treatment in type II diabetes, we evaluated not only Lp(a) concentration but also apolipoprotein(a) [apo(a)] polymorphism.

In the diabetic center of our clinic, we consecutively recruited 149 type II diabetic subjects without clinical or instrumental evidence of hepatopathy, nephropathy, coronary heart disease (CHD), periferal vascular disease, or proliferative diabetic retinopathy. None of the patients showed microalbuminuria. Out of 149 patients, 107 were treated with hypoglycemic oral drugs and 42 with insulin. These two groups were comparable (Table 1) with respect to sex, body mass index, systolic and diastolic blood pressure, and percentage of hypertension, smoking, and family history of CHD. Of course, patients on insulin treatment had a higher mean age and duration of diabetes because before the secondary failure, they were treated (for 11.5 ± 6.4 years) with oral drugs. Moreover, the two groups had similar glycometabolic control.

Fasting plasma samples for Lp(a) were frozen at −80°C until use. We used an ELISA-test [Macra-Lp(a), SDI, DE] for Lp(a) dosage and a Western Blot method for the identification of apo(a) isoforms, as previously described (4). Twenty-one apo(a) isoforms with molecular masses varying from 400 to 775 kDa were found. A conventional cut off was fixed at 640–655 kDa to split low and high molecular mass. Out of 149 subjects, we detected 0.7% "null" phenotypes [no electrophoretic bands and an Lp(a) concentration of 0 mg/dl] and 38.2% double band phenotypes.

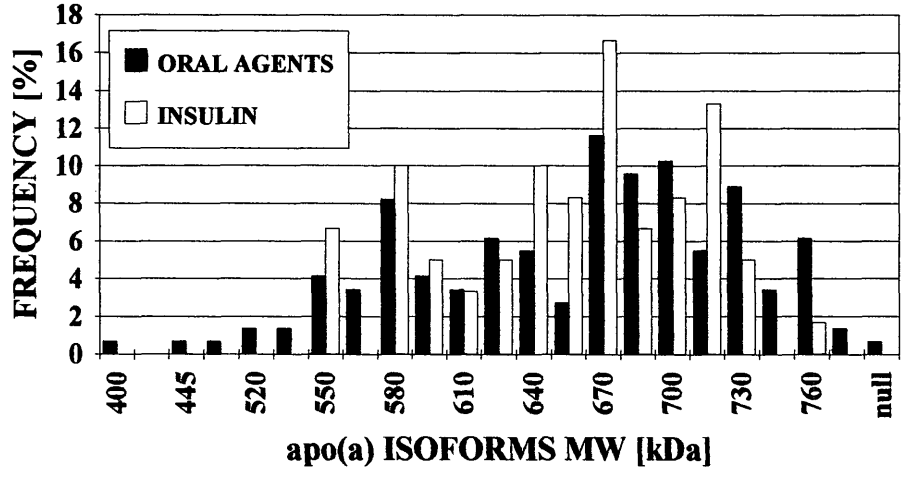

Figure 1—*Graphic representation of apo(a) isoform distribution in type II diabetic patients treated by oral agents (n = 107) and insulin (n = 42). Cutoff was fixed at 640–655 kDa.*

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Table 1—*Clinical and glyco-metabolic parameters, Lp(a) concentration, and apo(a) isoform distribution in type II diabetic patients in therapy with oral agents or insulin*

	Oral agents	Insulin	*P*
n	107	42	
M/F	44/63	18/24	0.85‡
Age (years)	55.6 ± 7.1	62.2 ± 7.0	0.0001*
BMI (kg/m^2)	26.1 ± 3.9	25.1 ± 3.1	0.11*
sBP (mmHg)	135.3 ± 17.5	138.8 ± 21.1	0.35*
dBP (mmHg)	82.0 ± 7.5	82.5 ± 7.9	0.76*
Diabetes duration (years)	9.8 ± 6.5	14.2 ± 7.9	0.001*
Hypertension	55 (51%)	22 (52%)	0.91‡
Smoking	37 (34%)	16 (38%)	0.69‡
Family history of CHD	37 (34%)	14 (33%)	0.88‡
Fasting glucose (mmol/l)	8.34 ± 1.81	8.59 ± 1.58	0.41*
HbA_{1c} (%)	6.8 ± 1.4	7.2 ± 1.3	0.11*
Fructosamine (μmol/dl)	301.4 ± 42.1	304.4 ± 36.4	0.66*
Tryglycerides (mmol/l)	1.70 ± 0.72	1.58 ± 0.64	0.39†
Total cholesterol (mmol/l)	5.49 ± 1.07	5.43 ± 0.97	0.74*
HDL cholesterol (mmol/l)	1.29 ± 0.24	1.30 ± 0.29	0.84*
LDL cholesterol (mmol/l)	3.46 ± 1.01	3.51 ± 0.76	0.71*
Lp(a) (mg/dl)	17.6 ± 19.0	16.2 ± 18.0	0.66*
Low molecular mass apo(a) isoforms (%)	39	40	0.97‡
High molecular mass apo(a) isoforms (%)	61	60	

Data are means ± SD or *n* (%). *Student's *t* test. †Mann-Whitney test. ‡χ^2 test. BMI, body mass index; sBP/dBP, systolic/diastolic blood pressure. To divide apo(a) isoforms with low and high molecular mass, a conventional cutoff was fixed at 640–655 kDa.

By the Spearman test, the classic inverse correlation between Lp(a) levels and apo(a) isoforms' molecular masses was confirmed in both patients treated by oral agents ($r_s = -0.752$, $P = 0.0001$) and patients treated by insulin ($r_s = -0.745$, $P = 0.013$).

We did not observe any significant difference in Lp(a) concentration between type II diabetic patients treated with oral agents and those treated with insulin (17.6 vs. 16.2 mg/dl). Moreover, no significant correlation was found between Lp(a) levels and daily insulin units (26.7 ± 10.2 U/day; Spearman rank test: $r_s = 0.195$, $P = 0.214$).

These results were confirmed by the analysis of apo(a) polymorphism: In both diabetic groups we observed a similar distribution of apo(a) isoforms, with a prevalence of high molecular masses (61 vs. 60%) (Table 1, Fig. 1).

In conclusion, Lp(a) does not seem significantly modulated by the exogenous insulin in type II diabetes, as also confirmed by the lack of association between Lp(a) levels and daily insulin requirements. The higher Lp(a) levels found by Gruden et al. (3) in patients on insulin treatment may be due to a different distribution of apo(a) phenotypes.

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