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Effect of Structured Group Education on Glycemic Control and Hypoglycemia in Insulin-Treated Patients

We investigated the effect of a structured group diabetes education program (1) on insulin-treated patients who had received individual counseling (at the time of diagnosis and insulin initiation). A total of 1,369 type 1 and insulin-treated type 2 diabetic patients with mean disease duration of 11.7 and 13.7 years, respectively, were allocated to receive or not receive structured group education, which was delivered in 10 45-min sessions over 3–5 days.

Baseline GHb levels of type 1 diabetic patients were 9.3% for the education group and 9.13% for the control group and decreased at 6 months by −0.82 and −0.22%, respectively ($P = 0.0005$). The number of injections per day and self-monitoring increased, while the total insulin dose remained unchanged. The incidence of severe hypoglycemia was reduced by 0.18 events/6 months in the education group, whereas it increased by 0.03 events/6 months in the control group ($P = 0.003$).

In type 2 diabetic patients, baseline GHb was 9.1% in the education and 8.7% in the control group, with a significant difference in the changes after 6 months in favor of the education group: −0.48 vs. 0.17%, respectively ($P = 0.0005$). A trend of reduced incidence of hypoglycemic episodes was observed.

Education for people with diabetes is a prerequisite for an understanding and acceptance of their condition and its successful self-management (2). A recent study found that group education was as effective as individual tuition in achieving improvements in glycemic control (3). In our study, structured group education after initial individual counseling at diagnosis and insulin initiation resulted in improved glycemic control and reduction of hypoglycemia. This observation is in line with results from previous publications (4,5). These improvements could be attributed to better self-care (increased frequency of self-monitoring in both type 1 and insulin-treated type 2 diabetic patients) and the increase in the number of insulin injections (in type 1 diabetic patients). It should be noted that there was not a significant difference in the mean insulin doses at the 6-month follow-up visit compared with baseline in both type 1 and type 2 diabetic patients; therefore, it was not the amount of injected insulin but the educational intervention that contributed to the improved glycemic control.

It can be suggested that a group education program following individual counseling could be an effective tool in diabetes management.

DRAGOMIR J. KOEV, MD, DMSC[1]
TZVETALINA I. TANKOVA, MD, PHD[1]
PLAMEN G. KOZLOVSKI, MD[2]

From the [1]Diabetic Clinic, Medical University, Sofia, Bulgaria; and [2]Novo Nordisk A/S, Bagsværd, Denmark.

Address correspondence to Plamen Kozlovski, MD, Novo Nordisk A/S, Novo Allé, 2880 Bagsvaerd, Denmark. E-mail: plko@novonordisk.com.

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Relationship of β2-Adrenergic Receptor Polymorphism With Obesity in Type 2 Diabetes

Adrenergic receptor genes are candidate genes for obesity and type 2 diabetes because they regulate lipid mobilization. An association has been reported between the frequently occurring Gly27Gln polymorphism of the β2-adrenergic receptor (B2ADR) gene with obesity (1,2) and triglycerides (3) in nondiabetic subjects.

We studied 542 unrelated type 2 diabetic subjects to assess the possible relationship of the Glu27Gln polymorphism of the B2ADR gene with obesity, as well as with plasma lipids. The Medical Ethics Committee of the University Medical Center Utrecht approved the study, and all participants signed an informed consent. The Glu27Gln polymorphism was examined by a modification of a PCR/restriction fragment–length polymorphism–based method (3) in duplicate and included sequence-verified controls.

After exclusion of PCR failures, genotypes were obtained for 502 type 2 diabetic patients. Subjects were subdivided according to three genotype groups: 109 subjects were Gln27Gln, 225 were Glu27Gln, and 168 were Glu27Glu. ANOVA of age at diagnosis of type 2 diabetes (61 ± 10, 60 ± 9, and 61 ± 9 years for the Gln27Gln, Glu27Gln, and Glu27Glu genotype groups, respectively), BMI (27.2 ± 3.8, 27.9 ± 4.4, and 28.2 ± 4.0 kg/m^2), HbA_{1c} (7.2 ± 1.4,

7.4 ± 1.3, and 7.2 ± 1.2%; normal values <6.1%), plasma cholesterol (5.3 ± 1.2, 5.3 ± 1.0, and 5.3 ± 1.0 mmol/l), plasma HDL cholesterol (1.2 ± 0.3, 1.1 ± 0.3, and 1.2 ± 0.3 mmol/l), and plasma triglycerides (1.8 ± 1.1, 1.9 ± 0.9, and 1.9 ± 0.1 mmol/l) showed no statistically significant differences (all $P > 0.26$). After exclusion of the insulin-treated subjects, the differences were also not statistically significant. We also found no significant differences for men or women separately.

We conclude that, at least in our cohort of subjects with type 2 diabetes, the Glu27Gln polymorphism of the β2-adrenergic receptor has no important effect on BMI, age at diagnosis of diabetes, metabolic control, or plasma lipids.

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JONATHAN H. VAN TILBURG, PHD[1]
CISCA WIJMENGA, PHD[1]
HARM VAN BAKEL, MSC[1]
LEIDA ROZEMAN, MSC[1]
PETER L. PEARSON, PHD[1]
TIMON W. VAN HAEFTEN, MD[2]

From the [1]Department of Medical Genetics, University Medical Center, Utrecht, the Netherlands; and the [2]Department of Internal Medicine, University Medical Center, Utrecht, the Netherlands.

Address correspondence to Timon W. van Haeften, MD, Department of Internal Medicine, G 02.228, P.O. Box 85500, NL 3508 GA Utrecht, The Netherlands. E-mail: t.w.vanhaeften@azu.nl

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Decreased Glucagon-Like Peptide 1 Fasting Levels in Type 2 Diabetes

The significance of over- or underproduction of glucagon-like peptide 1 (GLP-1) (7–36) amide in diabetes remains controversial (1,2). Although it has been proposed that at least one factor contributing to the pathogenesis of type 2 diabetes is desensitization of the GLP-1 receptor on β-cells (2), exogenous administration of GLP-1 in patients with type 2 diabetes fully normalizes fasting hyperglycemia and reduces postprandial glycemic increments (3). Also, the GLP-1 response to oral glucose is not altered in postmenopausal women with impaired glucose tolerance (2). The purpose of the present study was to establish any difference in the fasting state concentration of GLP-1 in type 2 diabetes as compared with the normal state.

We examined 26 subjects (12 men and 14 women) with type 2 diabetes who were aged 67.6 ± 10.09 years (means ± SD) and had a BMI of 29.2 ± 3.5 kg/m^2 and a diabetes duration of 137.0 ± 23.7 months. As a control group, we included 15 normal subjects (9 women and 6 men) who were aged 65.8 ± 8.01 years and had a BMI of 28.1 ± 3.7 kg/m^2. Patients with type 2 diabetes were treated by diet and were on metformin and/or sulfonylurea therapy with HbA_{1c} 6.81 ± 1.37% and fasting glucose 87.30 ± 5.20 mg/dl. Written consent was obtained from all participants. Blood samples were obtained at rest between 8:00 and 9:00 A.M. after an overnight fast and 24 h alcohol abstention. Insulin and GLP-1 were measured using an enzyme-linked immunosorbent assay (AxSYM; Abbott Lab, North Abbott, IL) and competitive radioimmunoassay (Peninsula Lab, Belmont, CA), respectively.

Interestingly, GLP-1, displaying a normal distribution, exhibited a statistically significant decrease ($P < 0.001$) in type 2 diabetic patients (23.8 ± 3.17 pg/ml) compared with control subjects (76.4 ± 4.47), whereas the plasma insulin concentration was higher in type 2 diabetic subjects (20.9 ± 13.4 ÌU/ml) than in control subjects (9.8 ± 1.3), with the difference being statistically significant ($P < 0.03$).

Using linear regression analysis, we found that GLP-1 was more negatively correlated with insulin in the type 2 diabetic patients than in the normal subjects ($r = -0.64$, $P < 0.001$ vs. $r = -0.43$, $P = 0.040$). In addition, GLP-1 was also negatively correlated with HbA_{1c} in type 2 diabetic subjects at a statistical level ($r = -0.43$, $P = 0.037$). Interestingly, the results were independent of diabetes duration.

The physiological importance of the decreased fasting level of GLP-1 in type 2 diabetic patients requires further clarification. If it is a reflection of increased sensitivity of β-cell responsitivity to secretagogues, then this study further confirms previous findings on the existence of a negative feedback loop between GLP-1 and insulin secretion and provides further evidence for the importance of the entero-insular axis in the regulation of insulin secretion. Moreover, the dissociation of GLP-1 levels with the duration of the disease suggests an early impairment of the entero-insular axis.

IOANNIS N. LEGAKIS, MD
COSTAS TZIORAS, MD
COSTAS PHENEKOS, MD

From the Endocrinology Unit, Red Cross Hospital, Athens, Greece.

Address correspondence to Dr. Ioannis N. Legakis, 27 Navarinou St., Cholargos 155 62 Athens, Greece. E-mail: legakis@ath.forthnet.gr.

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