as this group appears to be similar to women without diabetes with respect to infant feeding decisions.

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DOI: 10.2337/dc06-0174

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Treatment of Hyperglycemia in a 7-Year-Old Child Diagnosed With Neonatal Diabetes

Permanent neonatal diabetes is persistent, insulin-requiring hyperglycemia occurring before 1 month of life. Recent studies have identified activating mutations in *KCNJ11* encoding the Kir6.2 subunit of the ATP-sensitive K^+ channel as a common cause of neonatal diabetes (1–5). These patients can be successfully managed with oral sulfonylureas rather than insulin (2,4,6,7). We describe our experience with a 7-year-old Mexican-American girl diagnosed with insulin-dependent diabetes at 2 weeks of age when she presented with a respiratory infection. Her blood glucose level was 442 mg/dl, C-peptide 0.33 ng/ml (ref. 0.8–4), insulin 3.1 uU/ml (ref. 0–22), and HbA_{1c} (A1C) 7.7% (ref. <6.4), and she had negative diabetes autoantibodies. Her mother was diagnosed with insulin-dependent diabetes at 6 months of age. The propositus was evaluated at the pediatric diabetes center at Loma Linda University at the age of 6 years, and DNA testing revealed the presence of the permanent neonatal diabetes–associated mutation Arg201His in *KCNJ11* in both her and her mother (2). Glucagon and mixed-meal glucose tolerance testing did not show an increment in C-peptide from the baseline value. Insulin was weaned and discontinued over 2 weeks, with a starting glyburide dose of 1.25 mg b.i.d. ($0.05 \text{ mg} \cdot \text{kg}^{-1} \cdot \text{day}^{-1}$) and incremental increase to the current dose of 3.5 mg glyburide b.i.d. Previously, the patient's A1C on insulin ranged from 7.1 to 11.5%. Off insulin, and over the past 24 months, her quarterly A1C range was 5.1–6.3%, with no record or symptoms suggestive of hypoglycemia. Our results were consistent with other reports in the literature (2,4,6,7). The patient and her family were overwhelmingly pleased with the discontinuation of insulin, which they referred to as "a miracle."

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DOI: 10.2337/dc06-0487

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Circulating Vitamin D Concentrations in Two Neighboring Populations With Markedly Different Incidence of Type 1 Diabetes

Recent studies have suggested that vitamin D deficiency may increase the risk of type 1 diabetes (1). In Finland, the incidence of type 1 diabetes is the highest in the world, while in the neighboring Karelian Republic of Russia, the incidence is approximately one-sixth

that in Finland, despite no difference in HLA-conferred susceptibility (2). Thus, the reason(s) must be linked to environmental factors.

We assessed vitamin D status in the Russian Karelian and Finnish populations to determine whether vitamin D could play a role in the huge difference observed in diabetes incidence. The geographical location in terms of daily sunlight exposure is approximately the same in both populations (~62–66°N). Circulating concentrations of 25-hydroxy (25-OH) vitamin D were analyzed using a commercial enzyme immunoassay kit (Immunodiagnostic Systems Limited, Boldon, U.K.) in cohorts representing the background population (schoolchildren and pregnant women). The schoolchildren series included 100 subjects from Finland and 100 subjects from Russian Karelia matched for age, sex, and month of sampling (mean age ± SD, 10.9 ± 1.7 years, 52% male subjects). Serum samples were collected during the years 1994–2000, and 86% were drawn during March–April. The series of pregnant women included 103 female subjects from Russian Karelia and 172 women from Finland matched for age and date of sampling (mean age 26.7 ± 5.3 years). Samples were collected at the end of the first trimester of pregnancy as a part of the routine prenatal follow-up during the year 2000, and 81% of them were drawn during October–December. The study was approved by the local ethical committees and the Finnish Maternity Cohort steering group. The study was carried out in accordance with the Declaration of Helsinki.

The median serum concentrations of 25-OH vitamin D were approximately the same in both countries among schoolchildren (35.0 in Karelia vs. 39.3 nmol/l in Finland, P = NS Wilcoxon test) and pregnant women (28.4 vs. 28.9 nmol/l, P = NS by conditional logistic regression test). Sex had no effect on vitamin D status among the schoolchildren. According to the previously suggested cutoff limit for vitamin D deficiency (serum 25-OH vitamin D <25 nmol/l) (3,4), the proportion of vitamin D–deficient subjects was not higher in Finland compared with Karelia (16 vs. 27% in schoolchildren, $P = 0.091$; 38 vs. 41% in pregnant women, P = NS, respectively).

The results suggest that circulating vitamin D concentrations do not differ markedly between Finland and Russian Karelia. Accordingly, vitamin D status may not contribute to the marked difference in the incidence of type 1 diabetes between the countries, and there must be other factors protecting the Karelian children from developing type 1 diabetes.

Nevertheless, vitamin D substitution remains an important issue in these countries, even beyond infancy, and regular substitution has long been recommended in both countries (5–7), especially during pregnancy, infancy, and the dark months of the year.

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DOI: 10.2337/dc06-2559

Acknowledgments— This study was supported by the Päivikki and Sakari Sohlberg Foundation, the Tuberculosis Foundation in Tampere, and the EU INCO-Copernicus program (contract number IC15-CT98-0316).

The EPIVIR Study Group is acknowledged for collaboration (8).

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Soluble Tumor Necrosis Factor Receptor 2 Is Independently Associated With Brachial-Ankle Pulse-Wave Velocity in Nonobese Japanese Type 2 Diabetic Patients

Type 2 diabetes is associated with high mortality and morbidity due to atherosclerosis. Biermen (1) estimated that typical risk factors, including blood pressure, cholesterol, and smoking, can account for no more than 30% of excess cardiovascular risk factor in diabetic patients. Thus, other factors seem to play a role in the progression of atherosclerosis in diabetes.

Aortic stiffness measured by pulse-wave velocity (PWV) is shown to be highly predictive of cardiovascular mortality in type 2 diabetic patients (2). While age and blood pressure are shown to be associated with PWV, age and blood pressure alone do not completely account for the abnormalities of aortic stiffness in type 2 diabetic patients.

Tumor necrosis factor (TNF) system activity seems to be associated with the progression of atherosclerosis in type 2 diabetes. Shai et al. (3) demonstrated that soluble TNF receptor 2 (sTNF-R2) is