looked. However, we do agree with Weir et al. (1) that dietary means and physical activity play a central role as the first line of therapy. Finally, the correction of known cardiovascular risk factors, including typical lipid abnormalities in NIDDM, may be as important as the correction of hyperglycemia when we are considering the long-term prognosis of NIDDM patients.

MATTI I.J. UUSITUPA, MD
LEO K. NISKANEN, MD

From the Departments of Clinical Nutrition (M.I.J.U.) and Medicine (L.K.N.), University of Kuopio, Finland.

Address correspondence to M. Uusitupa, MD, Department of Clinical Nutrition, University of Kuopio, PO Box 1627, FIN-70211 Kuopio, Finland.

References

1. Weir GC, Nathan DM, Singer DE: Standards of care for diabetes (Technical Review). *Diabetes Care* 17:1514–1522, 1994
2. University Group Diabetes Program: A study of the effects of hypoglycemic agents on vascular complications in patients with adult-onset diabetes. II. Mortality results. *Diabetes* 19 (Suppl. 2):789–830, 1970
3. University Group Diabetes Program: Effects of hypoglycemic agents on vascular complications in patients with adult-onset diabetes. VIII. Evaluation of insulin therapy: final report. *Diabetes* 31 (Suppl. 5):1–81, 1982
4. U.K. Prospective Diabetes Study Group: UK prospective diabetes study (UKPDS) VIII: study design, progress and performance. *Diabetologia* 34:877–890, 1991
5. Uusitupa MIJ, Niskanen LK, Siitonen O, Voutilainen E, Pyörälä K: Ten-year cardiovascular mortality in relation to risk factors and abnormalities in lipoprotein composition in type 2 (non-insulin-dependent) diabetic and non-diabetic subjects. *Diabetologia* 36:1175–1184, 1993
6. Kuusisto J, Mykkänen L, Pyörälä K, Laakso M: NIDDM and its metabolic control predict coronary heart disease in elderly subjects. *Diabetes* 43:960–967, 1994
7. Uusitupa M, Pyörälä K, Laakso M: Metabolic control and macrovascular disease. In *International Textbook of Diabetes Mellitus*. Alberti KGMM, DeFronzo RA, Keen H, Zimmet P, Eds. Chichester, U.K., Wiley, p. 1471–1485, 1992
8. Brownlee M: Glycation products and the pathogenesis of diabetic complications. *Diabetes Care* 15:1835–1843, 1992
9. Niskanen L, Siitonen O, Suhonen M, Uusitupa MI: Medial artery calcification predicts cardiovascular mortality in patients with NIDDM. *Diabetes Care* 17:1252–1256, 1994

Prevalence of IDDM In Adults in the Community

In a paper recently published in *Diabetes Care* (1), Harris and Robbins addressed the issue of prevalence of adult-onset insulin-dependent diabetes mellitus (IDDM) in the U.S. population. They found that 0.3% of the population 30–74 years old and 7.4% of all diabetic patients diagnosed at that age were individuals with IDDM. The study was based on the data from the Second National Health and Nutrition Examination Survey (2). The authors' conclusion was that adult-onset IDDM was uncommon. Given the limitations of the survey, they could not detect slowly progressive IDDM with gradual loss of β-cell function. It was suggested that a very large population base would be required to identify sufficient numbers of adult-onset IDDM cases. They also stated that for accurate diagnosis of IDDM, fasting or stimulated C-peptide should be determined.

In a recent issue of *Diabetes Care* (3), we published our experience with IDDM in adults in a community-based study (registered population = 9,573 adults ≥30 years of age) in Israel. In a study to determine the clinical characteristics of insulin-treated non-insulin-dependent diabetes and IDDM patients, we examined all known insulin-treated diabetic patients from three community clinics, regardless of age at onset of diabetes. Fasting plasma C-peptide was measured in all patients as a marker of β-cell function. A total of 588 diabetic patients were found; of the 100 insulin-treated patients, only 25% were insulinopenic (C-peptide <0.132 nmol/l). Thus, the prevalence of IDDM in the surveyed population of age 30 years or older was 0.25%, similar to that found by Harris and Robbins.

Our results differ in that in almost 64% of our IDDM patients, diabetes was diagnosed at the age of 20 or older. Of all the IDDM patients, 33% were first treated by either diet or oral agents, indicating that at least at the beginning of the disease their pancreases were able to produce insulin. From this subgroup of patients, 7 of the 14 patients diagnosed at ≥21 years of age started insulin therapy at least 1 year after the diagnosis; 4 of these patients started insulin 15.7 years after diagnosis of diabetes.

We conclude that, at least in Israel, slowly progressive adult-onset IDDM is not uncommon. It is possible that studies based upon age of diagnosis, relative body weight, and insulin treatment alone could underestimate the prevalence of IDDM among the adult population.

MAXIMO MAISLOS, MD
SIMON WEITZMAN, MD

From the Diabetes Research Laboratory (M. M.) and the Epidemiology and Health Services Evaluation Unit (S. W.), Soroka Medical Center of Kupat Holim, Ben Gurion University, Faculty of Health Sciences, Beer Sheva, Israel.

Address correspondence to Maximo Maislos, MD, Department of Medicine "B," Soroka Medical Center, PO Box 151, 84101 Beer Sheva, Israel.

References
1. Harris MI, Robbins DC: Prevalence of adult-onset IDDM in the U.S. population. *Diabetes Care* 17:1337–1340, 1994
2. Harris MI, Hadden WC, Knowler WC, Bennett PH: Prevalence of diabetes and impaired glucose tolerance and plasma glucose levels in U.S. population aged 20–74 yr. *Diabetes* 36:523–534, 1987
3. Maislos M, Bodner-Frishman B, Weitzman S: Prevalence and clinical characteristics of type I and type II insulin-treated diabetes in the community. *Diabetes Care* 17:1230–1231, 1994

Is It Time to Modify the Glucose Tolerance Test for the Diagnosis of Gestational Diabetes?

The diagnosis of gestational diabetes mellitus (GDM) identifies two people at increased risk. The glucose levels in pregnancy that define the risk to the mother of developing non-insulin dependent diabetes at a future date are well established. However, the method of testing and the maternal glucose levels that will be most sympathetic to the fetal outcome are still to be defined.

In the November 1994 issue of *Diabetes Care*, Pettitt et al. (1) raised several important points regarding the diagnosis of GDM.

First, a 2-h glucose level ≥7.8 mmol/l after a 75-g glucose load appeared to be a better predictor of certain fetal outcomes than the National Diabetes Data Group criteria. A change to the World Health Organization (WHO) criteria during pregnancy would also facilitate comparisons with subsequent nonpregnant glucose tolerance test (GTT) results.

Second, doubts about the reliability and hence the usefulness of the fasting glucose level for the diagnosis of GDM were raised. These concerns have also been expressed by the WHO (2).

Third, nearly one-third of patients who were scheduled for a two-stage diagnostic procedure did not return for the second stage. Little importance has been given so far to the "no show" rate, which must markedly alter the incidence and the clinical reliability of testing for GDM.

These observations suggest that more consideration should be given to a testing procedure that is simple to administer from the medical side, encourages a high rate of maternal compliance, and is predictive of adverse fetal outcomes.

Since the beginning of 1993, a modification of the 75-g GTT (MGTT) has been evaluated in the Illawarra area of Australia. Women at two prenatal clinics have, over alternating 6-month periods, been offered either a GTT with both fasting and 2-h samples or an MGTT in which only the 2-h sample is taken. Both tests are administered in the morning after an overnight fast. The advantage of the MGTT is that the patient can be given the glucose solution at a preceding prenatal clinic (or doctor's office) visit and only needs to present for one blood test (3). GDM is diagnosed if the fasting plasma glucose is ≥5.5 mmol/l and/or the 2-h plasma glucose is ≥8.0 mmol/l (4).

However, even with this one-stage procedure there have been different rates of initial compliance. Ninety-four percent (707 out of 752) have completed the simpler MGTT, while only 90.1% (1,237 out of 1,367) have completed the GTT ($\chi^2 = 8.01$, df = 1, $P < 0.01$).

Apart from a 0.2 mmol/l difference in the 2-h glucose level for the diagnosis of GDM, the major difference between the MGTT and the one-stage test used by Pettitt et al. (1) is whether the woman needs to fast before the test. The argument against the need for fasting is convincing, and not fasting would further simplify the procedure.

Perhaps now is the time to revise the GTT used in pregnancy so that the glucose criteria are more sympathetic to the fetal outcome and the testing procedure is simplified to encourage the highest possible rate of maternal compliance.

ROBERT G. MOSES, FRACP

From the Diabetes Education Centre, Illawarra Area Health Service, Wollongong, New South Wales, Australia.

Address correspondence to Associate Professor R.G. Moses, 4/393 Crown St., Wollongong, 2500 NSW, Australia.

References
1. Pettitt DJ, Bennett PH, Hanson RL, Venkat Narayan KM, Knowler WC: Comparison of World Health Organization and National Diabetes Data Group procedures to detect abnormalities of glucose tolerance during pregnancy. *Diabetes Care* 17:1264–1268, 1994
2. World Health Organization: *Prevention of Diabetes Mellitus*. Geneva, World Health Org., 1994 (Tech. Rep. Ser., no. 844)
3. Moses RG: Screening for gestational diabetes mellitus (Letter). *Med J Aust* 157:500, 1992
4. Martin FIR: The diagnosis of gestational diabetes. *Med J Aust* 155:112, 1991

Insulin Sensitivity in Patients with NIDDM and the A-to-G Mutation at Nucleotide 3,243 of the Mitochondrial tRNA$^{Leu(UUR)}$ Gene

Recent studies have shown linkage between diabetes and mutations in mitochondrial DNA, the most common of which is a G-to-A transition in the tRNA$^{Leu(UUR)}$ gene at nucleotide 3,243