abetic) population, the incidence decreases from 3.38 to 2.68 ($P = 0.013$, χ^2). When data are expressed in terms of the total population, the benefit of changes in management may be obscured by the increasing prevalence of the disease.

Although the decrease in amputation incidence was only 20%, the actual incidence in 2004 was well within the range reported by other European centers. Since the magnitude of any such decrease is dependent on the baseline value, we suggest that rather than aim for a percentage reduction in incidence, future health care targets should specify an absolute value. Evidence from the published literature suggests that this should be of the order of 2 to 3 per 10^3 of those with diabetes or even lower.

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Decreased Insulin Secretion but Not Insulin Sensitivity in Normal Glucose Tolerant Thai Subjects

Reduced insulin secretion and insulin sensitivity has been demonstrated in normal glucose tolerant (NGT) subjects whose 2-h plasma glucose levels after an oral glucose tolerance test were 5.6–7.7 mmol/l compared with those with 2-h plasma glucose levels <5.6 mmol/l (1–3). These data are from high-risk ethnic subjects. Whether these data are true in Asians is uncertain.

We studied insulin secretion and insulin sensitivity in 51 NGT and 15 impaired glucose tolerant Thai subjects. Subjects were grouped according to 2-h plasma glucose levels after an oral glucose tolerance test into four groups (Table 1). Insulin sensitivity was determined by euglycemic-hyperinsulinemic clamp and expressed as glucose infusion rate. Insulin secretion was determined by homeostasis model assessment (HOMA) of steady-state β-cell function (%B) from a HOMA2 model (available at http://www.dtu.ox.ac.uk/homa) and adjusted with glucose infusion rate ($HOMA\%B_{adjusted}$) to obtain the accurate result of insulin secretion. For statistical analysis, ANOVA was used for group comparison, and between-group differences were compared using Bonferroni post hoc analysis.

As shown in Table 1, age, BMI, and waist circumference were significantly different between groups. Glucose infusion rate was also different between groups, but the difference disappeared after adjustment with age, BMI, and waist circumference. $HOMA\%B_{adjusted}$ of gr.IV was significantly lower than those of gr.I ($P = 0.003$) and gr.II ($P = 0.039$) but was not different from that of gr.III. The difference of $HOMA\%B_{adjusted}$ between groups could still be demonstrated after adjustment with age ($P = 0.005$).

This study demonstrated that insulin secretion adjusted for insulin sensitivity in NGT subjects started to decline progressively from 2-h plasma glucose >5.6 mmol/l. This study agrees with others (1–3). This is the first study in Asians where the declined β-cell function adjusted for insulin sensitivity is demonstrated in NGT subjects. These findings are in accordance with those from studies of other ethnic populations, including Mexican Americans, African Americans, Hispanics, and Caucasians, indicating that the results are not ethnic specific. It can be hypothesized that these high-normal oral glucose tolerance test subjects may have an increased risk of developing diabetes; therefore, lifestyle modification should be implemented early in this group as in impaired glucose tolerant subjects.

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Table 1—*Clinical characteristics of subjects*

	2-h plasma glucose (mmol/l) after OGTT				
	NGT ($n = 51$)			IGT ($n = 15$)	
	gr.I (<5.6)	gr.II (5.6–6.7)	gr.III (6.7–7.8)	gr.IV (7.8–11.1)	*P*
Sex (female/male)	5/10	12/11	8/5	6/9	NS
Age (years)	32.2 ± 1.2	34.1 ± 1.8	36.2 ± 2.9	45.1 ± 1.4*†‡	<0.0001
BMI (kg/m^2)	21.7 ± 0.9	23.1 ± 0.9	25.6 ± 1.0*	27.2 ± 0.8*†	<0.0001
Waist circumference (cm)	74.9 ± 2.7	75.6 ± 2.4	81.4 ± 2.6	91.8 ± 2.8*†	<0.0001
Glucose infusion rate (mg · kg_{FFM}^{-1} · min^{-1})	9.31 ± 0.82	9.43 ± 0.71	7.49 ± 0.51	6.04 ± 0.67*†	0.004
HOMA%B	127.9 ± 6.9	116.0 ± 8.6	129.0 ± 13.7	124.0 ± 12.2	NS
$HOMA\%B_{adjusted}$	1150.9 ± 89.2	1012.2 ± 67.8	951.1 ± 111.1	690.9 ± 73.4*†	0.005

Data are means ± SE. $HOMA\%B_{adjusted}$ = HOMA%B × glucose infusion rate. *$P < 0.05$ compared with gr.I; †$P < 0.05$ compared with gr.II; ‡$P < 0.05$ compared with gr.III. IGT, impaired glucose tolerance; OGTT, oral glucose tolerance test.

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Could Blood Ketone Monitoring Be A Tool for Managing Gestational Diabetes Mellitus?

Nutritional management of gestational diabetes mellitus (GDM) is based on guidelines from diabetology societies (1). Ketonuria is often monitored, but clear management guidelines have not been established. Home-based methods of measuring ketonemia are available. We believe that it is important to evaluate the utility of this tool in GDM.

We measured ketonemia in a control population of pregnant women and a GDM population. Pregnant women were systematically screened for GDM between the 24th and 28th weeks (75-g oral glucose tolerance test [OGTT], World Health Organization guidelines). A total of 56 women (29.98 ± 4.86 years of age, prepregnancy BMI 23.14 ± 4.62 kg/m^2, weight gain 14.49 ± 4.93 kg) with a normal OGTT and 49 women (31.35 ± 5.39 years, prepregnancy BMI 25.96 ± 5.91 kg/m^2, weight gain 9.25 ± 5.52 kg) with GDM were included.

Each subject was monitored in accordance with the appropriate guidelines; in addition, the control subjects performed glycemia and ketonemia self-monitoring three times a day (upon waking and before the midday and evening meals). GDM women were also asked to measure their postprandial glycemia. All subjects measured their fasting ketonuria.

Glycemia measurement was performed using test strips and a meter (Abbott), and capillary blood ketonemia measurement was performed using Optium β-Ketone test strips and the same meter (2). The replicate analysis resulted in CVs of 3.3%. The study protocol was approved by an ethics committee.

The two groups did not differ in terms of age, but BMI and weight gain were higher in the GDM than in the control group ($P < 0.01$). The mean ketonemia was lower in the control than in the GDM group (0.01 ± 0.10 vs. 0.04 ± 0.009 mmol/l, $P < 0.001$). Fasting ketonemia did not differ between the control and GDM groups (0.01 ± 0.11 vs. 0.01 ± 0.06 mmol/l, respectively). Ketonemia values measured before the midday and the evening meal were lower for control than for GDM patients (midday 0.01 ± 0.08 vs. 0.05 ± 0.11 mmol/l, $P = 0.002$; evening 0.02 ± 0.09 vs. 0.05 ± 0.10 mmol/l, $P = 0.005$).

A ketonemic episode was defined as the unbroken period during which each day is a part of a sliding 7-day interval containing >25% of height value. Of the control subjects, 6 (12%) experienced at least one ketonemic episode (average length 10.5 days) versus 23 (47%) in the GDM group (average length 13.8 days) (a total of 37 episodes).

For women with GDM, we are not currently in a position to conclude whether their ketonemia levels have clinical significance in terms of the pregnancy outcome or the health of the child. Ketonemia values differ from those recorded in control subjects, and this difference is not irrelevant. A study needs to be performed to be certain that higher ketonemia has a detrimental prognostic significance for fetal development.

Reports from the literature have focused exclusively on ketonuria. A negative correlation between ketonuria and intellectual quotient in children born to diabetic mothers has been reported (3,4). A relationship between high fasting ketonemia during the last trimester and delayed educational development has been suggested (5).

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Blood Pressure Measurement in Diabetes Clinic

Are we paying enough attention?

The American Diabetes Association statement (1), "Care of Children and Adolescents With Type 1 Diabetes," outlines recommendations for management of hypertension in children with type 1 diabetes. Hypertension in children can be missed if appropriate norms are not used, and, as the authors state, "clinicians who care for children with diabetes often pay little or no attention to blood pressure." Here, we report results of a retrospective chart review of serial clinic