



Diabetes Rates May Triple in Bangladesh by 2030

The number of people with diabetes will triple in Bangladesh and double worldwide by 2030, according to new estimates from World Health Organization (WHO) and several European universities. That's because researchers say

deaths due to infectious diseases as well as maternal and infant deaths in developing countries are expected to continue to drop in the next 30 years. Meanwhile, as diabetes rates climb in these areas, deaths due to related diseases, will increase and account for a larger proportion of deaths have been shown in the box.

Rank	2000		2030	
	Country	People with diabetes (millions)	Country	People with diabetes (millions)
1	India	31.7	India	79.4
2	China	20.8	China	42.3
3	U.S.	17.7	U.S.	30.3
4	Indonesia	8.4	Indonesia	21.3
5	Japan	6.8	Pakistan	13.9
6	Pakistan	5.2	Brazil	11.3
7	Russian Federation	4.6	Bangladesh	11.1
8	Brazil	4.6	Japan	8.9
9	Italy	4.3	Philippines	7.8
10	Bangladesh	3.2	Egypt	6.7

Diabetes Epidemic Growing Ahead of Schedule

Researchers say the new estimate is based on the fact that up to 50% of people with diabetes do not know they have disease, and their projection is based on people with both diagnosed and undiagnosed diabetes.

Researchers say it's unlikely that their projections overestimate the future of the diabetes epidemic because they are based on the prevalence of obesity remaining stable. But obesity rates have been rising substantially in recent years. Obesity is the leading modifiable risk factor for diabetes.

Even if the prevalence of obesity remains stable until 2030, which seems unlikely, it is anticipated that the number of people with diabetes will more than double as a consequence of population aging and urbanization.

SOURCE: Wild, S. Diabetes Care, May 2004; vol 27: pp 1047-1053.

Diabetescope

Possible Link Between Diabetes and Alzheimer's Disease

It is known that people with diabetes have a greater risk of developing Alzheimer's disease, but the exact cause is not known. Researchers in Germany and Boston, suggests that insulin resistance neurons can cause biochemical changes seen in Alzheimer's disease. Scientists realized that insulin receptors are present on all tissues and affect function of these tissues. Furthermore, various research findings have suggested that disruption of the insulin signaling system may occur in Alzheimer's disease. One large European study found people with diabetes to be at least twice as likely to develop Alzheimer's disease. The risk was higher in people taking insulin. To study effects of insulin resistance in brain, researchers used genetically altered mice called Neuronal Insulin Receptor Knockout

(NIRKO) mice, which missing insulin receptors in neurons. Previously, using these NIRKO mice, it has been shown that neuron-specific insulin resistance could contribute to type 2 diabetes, loss of normal appetite, obesity, and infertility. Using behavioral and memory testing, high-tech imaging, variety of biochemical tests, metabolic processes within the brains of NIRKO mice is studied. Compared with normal, NIRKO mice had markedly reduced activity of insulin signaling proteins in the brain. It was found to lead to overactivity of enzyme GSK3 beta, which led to hyperphosphorylation of a protein called tau. Hyperphosphorylation of tau is a hallmark of brain lesions seen in Alzheimer's disease and suggested as early marker of this disease. NIRKO mice showed no changes in proliferation or survival of neurons, memory, or basal brain glucose metabolism, suggesting that insulin resistance may interact with other risk factors. Further research is needed to clarify how insulin resistance interacts with other genetic & biochemical abnormalities in the development of Alzheimer's and other neurodegenerative diseases.

17 Feb 2004, Proceedings of the National Academy of Sciences.

Young People Prone to Type 2 Diabetes Exhibit Alterations in Mitochondria

Researchers at Yale have found that decreased activity in muscle mitochondria may be a major factor in the development of type 2 diabetes in young, lean offspring of parents with the disease.

They demonstrated a potential mechanism for the accumulation of fat in muscle cells of young, lean, insulin-resistant children of parents with type 2 diabetes by comparing them with insulin-sensitive control subjects.

There is a strong relationship between lipid content in the muscle and insulin resistance in skeletal muscle. Insulin resistance is the best predictor for whether someone will eventually develop type 2 diabetes. Using proton magnetic resonance spectroscopy (MRS), researchers found that insulin resistance in muscle of the diabetic offspring was accompanied by an increase in muscle cell lipid content.

It has to be distinguished whether the increase in muscle cell triglycerides was the result of increased delivery of fatty acids to muscle cells from the fat stored in adipocytes, or the result of a decreased rate of fat oxidation by the mitochondria in the muscle cells. Using phosphorus MRS it is found that a 30% reduction in the rate of mitochondrial energy production in the muscle of insulin resistant subjects compared to control group.

These data support the hypothesis that insulin resistance in young, lean, healthy insulin resistant offspring of patients with type 2 diabetes may be due to an inherited defect that causes decreased mitochondrial activity and predisposes them to accumulate fat inside their muscle cells & develop insulin resistance.

Now it has to be determined whether the decrease is due to a reduced number of mitochondria and/or reduced mitochondrial function and whether these abnormalities can be reversed with exercise training.

These results support the hypothesis that nuclear encoded genes that regulate mitochondrial biogenesis may be an important genetic cause of type 2 diabetes and that mitochondrial biogenesis represents a novel therapeutic target for treatment and possible prevention of type 2 diabetes.

Source: New England Journal of Medicine, February 12, 2004; vol. 350, issue 7 pp. 664-71.

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Diabetescpoe

Giving babies cereals too early - diabetes risk

Introducing cereal too early or too late in infancy might increase the odds of diabetes in children already at risk for the disease, a study suggests. Another study found a similar risk for introducing gluten-containing foods too soon. Both studies suggest that starting solid food at the wrong time could overwhelm at-risk infants' immature immune systems and trigger changes that might lead to diabetes.

The preliminary findings are far from proof, and the researchers themselves said the results should not prompt any changes in babies' feeding habits. The studies -- one from the University of Colorado, the other from Germany -- are published in *Journal of the American Medical Association*.

Both involved youngsters already at risk for juvenile diabetes. Both studies also compared the timing of the introduction of solid food in infancy with the development of antibodies that sometimes lead to juvenile diabetes.

Doctors recommend starting solid food -- usually cereal -- between 4 to 6 months. It was found a fourfold increased risk of developing pre-diabetes antibodies in babies started on any type of cereal before the recommended period, and a fivefold higher risk for those fed afterward. Introducing solid food too soon induces the production of antibodies that destroy insulin-producing cells. Starting solid food after 7 months also might overload infants' still-developing immune systems. The study involved 1,183 children followed for about 4 years. Only 34 showed persistent evidence of the pertinent antibodies, and only 16 actually developed diabetes.

The other study, from the Diabetes Research Institute in Munich, involved 1,610 children for about 6 years. It found an increased risk in introducing solids earlier than 4 months of age -- but only with foods containing gluten, a protein found in wheat and other grains.

October 2003, Journal of the American Medical Association

Metformin/TZDs to Insulin Improves Survival 46%

Insulin-sensitizing therapy was associated with better survival and outcomes than insulin-providing therapy for patients with type 2 diabetes. The addition of insulin-sensitizing therapy improved survival almost 46%.

A study follow-up ranged from 0.5 to 8.5 years in cardiovascular department at LDS Hospital in Utah. Information about discharge prescriptions were available on 8,004 patients. They were divided into four groups according to their regimens: exogenous insulin (7,404 patients), sulfonylurea (3,442 patients), metformin (1,601 patients) and thiazolidinedione (320 patients). Some patients received more than one agent.

Patient parameters were similar in all groups. Ages ranged from 63 to 67 years, and about 52% were men. The rates of CHD, peripheral vascular disease, coronary artery disease and congestive heart failure were similar, ranging from 41% to 45%.

The results of fasting blood glucose and A1C tests were similar across all four groups. Where the results differed was death rates. While death rates improve clearly with use of metformin or thiazolidinedione, and somewhat with the use of sulfonylurea. This is strikingly not the case with the use of exogenous insulin, which is associated with a substantial increase in all-cause mortality.

Insulin-receiving patients clearly have lower survival. The use of sulfonylurea is associated with somewhat improved survival with the hazard ratio (HR) of 0.8, adding that metformin and thiazolidinedione were superior in improving survival (HR, 0.75 and 0.55, respectively).

Adding one of those agents to an insulin-receiving strategy improved survival rates by 50%. Compared with insulin alone, the addition of sulfonylurea had no effect on mortality, however, when metformin or thiazolidinedione were added to insulin, this resulted in highly significant - 46% - reduction in death.

Oral anti-diabetic agent was associated with improved hazard ratio compared with non-use, while exogenous insulin was associated with a doubling of all-cause mortality even when adjusted for co-morbidities.

Source: Abstract # 810-5. Presented at the Annual Scientific Session of the American College of Cardiology. March 7-10. New Orleans.

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Gestational diabetes mellitus

Definition

Gestational diabetes mellitus (GDM) is defined as any degree of glucose intolerance with onset or first recognition during pregnancy. The definition applies whether insulin or only diet modification is used for treatment and whether or not the condition persists after pregnancy. It does not exclude the possibility that unrecognized glucose intolerance may have antedated or begun concomitantly with the pregnancy.

Approximately 7% of all pregnancies are complicated by GDM. The prevalence may range from 1 to 14% of all pregnancies, depending on the population studied and the diagnostic tests employed.

Detection and diagnosis

Risk assessment

Risk assessment for GDM should be undertaken at the first prenatal visit.

Women with clinical characteristics consistent with a high risk of GDM should undergo glucose testing as soon as feasible. If they are found not to have GDM at that initial screening, they should be retested between 24 and 28 weeks of gestation. Women of average risk should have testing undertaken at 24-28 weeks of gestation. Low-risk status requires no glucose testing, but this category is limited to those women meeting all of the following characteristics:

(OGTT) was recommended. In the GCT, plasma glucose is measured 1 hour after ingestion of a 50 g pure glucose load in 150 mL of fluid and may be performed without regard to the time of day or time of last meal. Opinions differ as to the optimal cutoff value for the 50 g GCT. A value of 7.2 mmol/L (130 mg/dL) will identify 90% of women with GDM, but 20%-25% of all women screened will need to continue to the 100 g OGTT. Raising the cutoff value to 7.8 mmol/L (140 mg/dL) will identify only 80% of women with GDM but decrease to 14%-18% the number of women who will have GCT results that necessitate further testing. Study showed that different cutoff values for the 50 g GCT can be assigned to subgroups of the tested population based on a clinical risk factor scoring system. Low-risk individuals were not tested, while for intermediate-risk patients the 7.8 mmol/L (140 mg/dL) threshold was maintained. For high-risk patients the threshold was lowered to 7.1 mmol/L (128 mg/dL), achieving an 82.6% detection rate with only 16% false positives in this group. This strategy allowed 34.6% of the study population to avoid the glucose challenge test altogether without compromising detection rates.

SCREENING WITH 75 G OGTT

Although the 75 g OGTT is usually used as a one-step diagnostic test, some investigators have reported the use of the 75 g test as a screening test with one-hour values 7.8 mmol/L and 8.0 mmol/L identifying women that need to proceed to a diagnostic 100 g or 75 g tolerance test.

RANDOM BLOOD GLUCOSE OR FASTING BLOOD GLUCOSE

Random blood glucose or fasting blood glucose measurements have been suggested as screening options that are more economical and well tolerated than glucose challenge tests. There is a lack of conclusive data documenting the reproducibility, sensitivity, and specificity of these tests.

Diagnostic Criteria For Gestational Diabetes Mellitus

1. FASTING OR RANDOM BLOOD SUGARS

A fasting plasma glucose level >7.0 mmol/L (126 mg/dL) or a random plasma glucose >11.1 mmol/L (200 mg/dL) meets the threshold for the diagnosis of gestational diabetes if confirmed on a subsequent day, and precludes the need for any glucose challenge.

High Risk Factors For GDM

- Previous history of gestational diabetes or glucose intolerance
- A family history of diabetes
- Previous macrosomia ($>4,000$ g)
- Previous unexplained stillbirth
- Previous neonatal hypoglycemia, hypocalcemia, or hyperbilirubinemia
- Advanced maternal age
- Obesity
- Repeated glycosuria in pregnancy
- Polyhydramnios
- Suspected macrosomia

Low Risk Factors For GDM

- Age <25 years
- Weight normal before pregnancy
- Member of an ethnic group with a low prevalence of GDM
- No known diabetes in first-degree relatives
- No history of abnormal glucose tolerance
- No history of poor obstetric outcome

Screening Alternatives

Three methods of biochemical screening for GDM have been described.

SCREENING WITH GLUCOSE CHALLENGE TEST FOLLOWED BY AN ORAL GLUCOSE TOLERANCE TEST

Screening with a 50 g glucose challenge test (GCT) followed by a 100 g oral glucose tolerance test

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2. TESTING WITH THE 75 G OGTT

The 75 g two-hour OGTT is the diagnostic test recommended by the WHO and is practiced in most of the world excluding North America where the 100 g three-hour OGTT is the principal diagnostic test. Several recent North American guidelines have emphasized the need for common diagnostic criteria although still allowing both approaches. The Hyperglycemia and Adverse Pregnancy Outcomes (HAPO) study is a large (n = 25,000) prospective study on GDM worldwide that has also adopted the 75 g approach. The new guidelines together with the HAPO study are likely to shift even more North Americans to the 75 g screening.

There is no consensus regarding the criteria for the 75 g OGTT in pregnancy. The WHO criteria have the advantage of requiring only two blood samples, but since the threshold values are less stringent, approximately 7%-8% of the population tested will be diagnosed with GDM. The more stringent ADA criteria require three blood samples but will decrease the number of women diagnosed with GDM to 2%-3%. **Table 1** summarizes the diagnostic criteria for the 75 g OGTT as proposed by several medical organizations. The data from the HAPO study and other large studies may lead to a standardization of the diagnostic criteria based on thresholds associated with identifiable clinical outcomes.

conversion of the original O'Sullivan values **Table 2**. Schwartz et al., based on a retrospective analysis of 8557 OGTT results, estimated that replacing the NDDG criteria with the Carpenter and Coustan

TABLE 2
100 G OGTT DIAGNOSTIC CRITERIA FOR GESTATIONAL DIABETES MELLITUS

Status	Carpenter-Coustan Conversion <i>Plasma or Serum Glucose Level</i>		NDDG Conversion <i>Plasma Level</i>	
	mg/dL	mmol/L	mg/dL	mmol/L
Fasting	95	5.3	95	95
One hour	180	10.0	180	180
Two hour	155	8.6	155	155
Three hour	140	7.8	140	140

NDDG: National Diabetes Data Group. Two or more values must be met or exceeded. The test should be performed after 8–14 hour fast and following 3 days of unrestricted diet (>150 g carbohydrate per day).

criteria would increase by 54% the number of pregnant women with a diagnosis of GDM and would also increase costs, while only minimally affecting prevalence of infant macrosomia.

4. POSTPARTUM TESTING

TABLE 3
VALUES FOR POSTPARTUM 75 G GLUCOSE TOLERANCE TEST

	Diabetes Mellitus	Impaired Glucose	Impaired Fasting Glycaemia (IFG)
Fasting plasma glucose (FPG)	≥ 7.0 mmol/L (126 mg/dL)	≥ 7.0 mmol/L (126 mg/dL)	≥ 6.1 mmol/L □ (110 mg/dL) and ≥ 7.0 mmol/L (126 mg/dL)
2-hour 75 g value	≥ 11.1 mmol/L (200 mg/dL)	≥ 7.8 mmol/L □ (140 mg/dL) ≥ 11.1 mmol/L (200 mg/dL)	≥ 7.8 mmol/L □ (140 mg/dL)

Adapted from : WHO Consultation: Definition, diagnosis and classification of diabetes mellitus and its complications; 1999.

Women diagnosed with GDM in pregnancy should have a fasting blood sugar and 75 g OGTT to determine

their glycemic status 6-12 weeks postpartum **Table 3**.

Management of Gestational Diabetes Blood Glucose Monitoring

In patients requiring insulin therapy, the ideal frequency of glucose monitoring has not been established. A common practice is to check the glucose level four times daily. A first morning glucose level can rule out fasting hyperglycemia, and additional one- or two-hour postprandial values can ensure adequate control. Postprandial testing is preferable to preprandial testing. In one randomized study comparing postprandial and preprandial blood

glucose monitoring in patients with gestational diabetes who required insulin therapy, those who measured their glucose levels after meals had larger drops in A1c (-3.0 versus -0.6 percent, P < .001), gave birth to infants with lower birth weights (3,469 g [7 lb, 10 oz] versus 3,848 g [8 lb, 7 oz], P = .01), and had

TABLE 1
CRITERIA FOR DIAGNOSIS OF GDM WITH THE 75 G OGTT

Organization	Fasting	1 h PG	2 h PG	Diagnostic Criteria for GDM
WHO	≥7.0 mmol/L (126 mg/dL)	≥Not measured 10.0 mmol/L	≥7.8 mmol/L (140 mg/dL)	One abnormal value
Fourth International Workshop/ ADA	(95 mg/dL)	(180 mg/dL)	8.6 mmol/L (155 mg/dL)	Two or more abnormal values
	≥5.3 mmol/L			

PG: Post glucose, ADA: American Diabetes Association.

3. THE 100 G OGTT

Despite criticism of the 100 g OGTT, in view of its popularity in North America, a 100 g glucose load may be used in diagnosing GDM. Diagnostic criteria are either the Carpenter-Coustan or NDDG

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percent, $P = .04$).

There is neither objective evidence nor a clinical guideline to support a frequency for glucose monitoring in patients with diet-controlled gestational diabetes. In these patients, an acceptable practice is to use the four-times-a-day schedule on two days per week and begin more intensive treatment if two values per week exceed the limits.

Diet

A recent Cochrane review found no difference in the prevalence of birth weights greater than 4,000 g (8 lb, 13 oz) or cesarean deliveries in women with gestational diabetes who were randomly assigned to receive primary dietary therapy or no specific treatment. The review concluded that insufficient evidence exists to recommend dietary therapy in patients with altered glucose metabolism.

The ideal diet for women with gestational diabetes remains to be defined, and current recommendations are based on expert opinion. A diet that adequately meets the needs of pregnancy but restricts carbohydrates to 35 to 40 percent of daily calories. Caloric restriction should be approached with caution, because two studies have reported a relationship between elevated maternal serum ketone levels and reduced psychomotor development and IQ at three to nine years of age in the offspring of mothers with gestational diabetes.

For patients with a body mass index greater than 30 kg/m², lowering daily caloric intake by 30 to 33 percent (to approximately 25 kcal per kg of actual weight per day) would avoid ketonemia. Regular exercise has been shown to improve glycemic control in women with gestational diabetes, but it has not been shown to affect perinatal outcomes.

Insulin

Most, but not all, prospective trials involving insulin therapy in women with gestational diabetes have shown a reduction in the incidence of neonatal macrosomia. Therefore, insulin therapy traditionally has been started when capillary blood glucose levels exceed 105 mg per dL (5.8 mmol per L) in the fasting state and 120 mg per dL (6.7 mmol per L) two hours after meals. These cutoff values are derived from guidelines for managing insulin in pregnant women who have type 1 diabetes. A more aggressive goal of a fasting capillary blood glucose level below 95 mg per dL (5.3 mmol per L) is supported by a prospective study of 471 women with gestational diabetes that showed a decrease in large-for-gestational-age neonates, from 28.6 to 10.3 percent (relative risk, 5.99; 95 percent confidence interval, 1.37 to 8.88), in the women with fasting blood glucose levels of 95 to 105 mg per dL who were treated, respectively, with diet or insulin; the study reported no data on additional birth outcomes. Because of variable and imperfect data on this point, it is acceptable to use

either cutoff value for fasting glucose testing. One prospective nonrandomized study of 445 patients has shown a reduction in operative deliveries and birth trauma in women with gestational diabetes who are treated with insulin. However, the findings of this study remain to be demonstrated in an adequately powered RCT.

There are no specific studies declaring one type of insulin or a certain regimen as superior in affecting any perinatal outcome. A common initial dosage is 0.7 units per kg per day, with one dose consisting of two thirds of the total amount given in the morning and one dose consisting of one third of the total amount given in the evening. One third of each dose is given as regular insulin, and the remaining two thirds as NPH insulin. A recent study of 42 women with gestational diabetes supports the safety of very-short-acting insulin lispro, which can be used with once-daily extended insulin ultralente.³¹ The simplest regimen that will control blood glucose levels is the best.

Physicians should expect to increase the insulin dosage as the pregnancy progresses and insulin resistance increases.

Oral Hypoglycemic Medications

Use of oral hypoglycemic agents to treat gestational diabetes has not been recommended because of concerns about potential teratogenicity and transport of glucose across the placenta (causing prolonged neonatal hypoglycemia). Although first-generation hypoglycemic agents (chlorpropamide, tolbutamide) have been shown to cross the placenta, recent *in vitro* and *in vivo* evidence has determined that glibenclamide (Dibenol) does not enter the fetal circulation.

Comparing the use of glibenclamide (Dibenol) and insulin in women with gestational diabetes demonstrated that glibenclamide (Dibenol) therapy resulted in comparable maternal outcomes (e.g., glycemic control, cesarean deliveries) and neonatal outcomes (e.g., macrosomia, hypoglycemia, intensive care unit admissions). Glibenclamide (Dibenol) therapy was not started before 11 weeks of gestation and was not detected in any of the neonatal cord blood samples. Preliminary evidence from this trial suggests that glibenclamide (Dibenol) may be a safe, effective alternative to insulin in the management of gestational diabetes.

But glibenclamide (Dibenol) should not be prescribed for the treatment of gestational diabetes until additional evidence support its safety and effectiveness. Despite these recommendations, many physicians are using glibenclamide (Dibenol) in this setting because of its ease of use compared with insulin. In a recent prospective cohort study of patients with polycystic ovary syndrome, metformin (Comet) therapy has been shown to

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decrease the subsequent incidence of gestational diabetes, reduce first-trimester miscarriage rates, and result in no apparent increase in congenital anomalies.

Antepartum Fetal Assessment

Data on gestational diabetes and an increased risk of fetal demise are conflicting. Evidence is insufficient to determine the optimal antepartum testing regimen in women with gestational diabetes who have relatively normal glucose levels on diet therapy and no other perinatal risk factors. Acceptable practice patterns for monitoring pregnancies complicated by gestational diabetes range from testing all women beginning at 32 weeks of gestation to no testing until 40 weeks of gestation.

Antenatal testing should be done in patients whose blood glucose levels are not well controlled, who require insulin therapy, or who have concomitant hypertension. The antenatal testing can be initiated at 32 weeks of gestation. In this situation, no method of antenatal testing has proved superior to others.

Timing And Route Of Delivery

In gestational diabetes, shoulder dystocia is the complication most anticipated at the time of delivery. In one study, this complication occurred in 31 percent of neonates weighing more than 4,000 g who were delivered vaginally to unclassified mothers with diabetes. No prospective data support the use of cesarean delivery to avoid birth trauma in women who have gestational diabetes. One remaining limiting factor is the 13 percent error rate (± 2 SD) in estimating fetal weight by ultrasonography.

A decision analysis that evaluated the cost and efficacy of a policy of elective cesarean delivery for an estimated fetal weight of 4,500 g (9 lb, 15 oz) in mothers with diabetes found that 443 cesarean deliveries would need to be performed to prevent one case of brachial plexus injury. A reasonable approach is to offer elective cesarean delivery to the patient with gestational diabetes and an estimated fetal weight of 4,500 g or more, based on the patient's history and pelvimetry, and the patient and physician's discussion about the risks and benefits. There are no indications to pursue delivery before 40 weeks of gestation in patients with good glycemic control unless other maternal or fetal indications are present.

Intrapartum Management

The goal of intrapartum management is to maintain normoglycemia in an effort to prevent neonatal hypoglycemia. Patients with diet-controlled diabetes will not require intrapartum insulin and simply may need to have their glucose level checked on admission for labor and delivery. While patients with

insulin-requiring diabetes are in active labor, capillary blood glucose levels should be monitored hourly. Target values are 80 to 110 mg per dL (4.4 to 6.1 mmol per L).

Postpartum Management

Women with gestational diabetes rarely require insulin in the postpartum period. As insulin resistance quickly resolves, so does the need for insulin. Patients with diet-controlled diabetes do not need to have their glucose levels checked after delivery. In patients who required insulin therapy during pregnancy, it is reasonable to check fasting and two-hour postprandial glucose levels before hospital discharge.

Because women with gestational diabetes are at high risk for developing type 2 diabetes in the future, they should be tested for diabetes six weeks after delivery via fasting blood glucose measurements on two occasions or a two-hour oral 75-g glucose tolerance test. Normal values for a two-hour glucose tolerance test are less than 140 mg per dL. Values between 140 and 200 mg per dL (11.1 mmol per L) represent impaired glucose tolerance, and greater than 200 mg per dL are diagnostic of diabetes. Screening for diabetes should be repeated annually thereafter, especially in patients who had elevated fasting blood glucose levels during pregnancy.

Breastfeeding improves glycemic control and should be encouraged in women who had gestational diabetes.

Contraception should be discussed, because women who have diabetes during one pregnancy are likely to have the same condition in a subsequent pregnancy. There are no limits on the use of hormonal contraception in patients with a history of gestational diabetes. As previously noted, these women also are at increased risk of developing type 2 diabetes in the future.

Patients should be counseled about diet and exercise. By losing weight and exercising, women can significantly decrease their risk of developing diabetes.

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2. SOGC Clinical Practice Guidelines Screening For Gestational Diabetes Mellitus
No. 121, November 2002
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Last Note!

Dairy Foods Help Burn Fat, Speed Weight Loss!

Milk, Cheese, and Yogurt May Enhance Weight Loss Efforts!

Although calories still count, the study showed that obese adults who ate a high-dairy diet lost significantly more weight and fat than those who ate a low-dairy diet containing the same number of calories.

"If you compare a dairy-rich versus a dairy-poor diet you can nearly double the rate of weight and fat loss with the same level of calorie restriction," says researcher Michael Zemel, PhD, professor of nutrition and medicine at the University of Tennessee in Knoxville. Eating three to four servings of dairy products a day is more effective at enhancing weight loss efforts than calcium supplementation alone.

The study appears in the April issue of *Obesity Research* and was supported by the National Dairy Council.

Previous studies have already shown that dieters who eat a calcium-rich diet are more successful at losing weight, but this study indicates that it's more than just the calcium in dairy products that helps shed pounds.

Dairy Products Beat Calcium for Weight Loss

In the study, researchers compared the effects of 3 different calorie-restricted diets on weight loss in 32 obese adults. Each of the participants reduced daily calorie intake by about 500 calories per day for 24 weeks & were divided into three groups:

- High-dairy. Total calcium intake of 1,200-1,300 milligrams per day from three to four servings of dairy foods, specifically milk, hard cheese, and yogurt.
- High-calcium supplemented/low-dairy. Total calcium intake of 1,200-1,300 milligrams per day made up of no more than one serving of dairy per day plus an 800-milligram calcium supplement.
- Low-calcium/low-dairy. Total calcium intake of 400-500 milligrams per day with no more than one serving of dairy per day and a placebo supplement.

Researchers say participants were free to choose from fat-free, low-fat, and regular milk, cheese, and yogurt. They typically picked fat-free and low-fat milk and yogurt and regular cheeses, while keeping their overall fat intake the same. Serving sizes were 8 ounces or a cup for milk and yogurt and 1.5 ounces of hard cheese or 2 ounces of processed cheese, such as two slices of American cheese.

The study showed that all of the groups lost weight, but those who ate the dairy-rich diet lost the most with an average of 24 pounds compared with 19 pounds in the calcium supplement group and 15 pounds in the low-calcium/low-dairy group. In addition, researchers found the high-dairy group lost significantly more body fat than those in the other groups, particularly from the midsection.

Dairy's Role in Weight Loss

Previous studies have shown that calcium can boost weight loss by increasing fat breakdown in fat cells. This study suggests that taking in calcium from dairy products may actually improve on those effects.

Dairy for some reason, yet unexplained, has a greater effect on fat loss and specifically trunk fat loss than does calcium alone. Although the bulk of dairy's weight-loss enhancing effects is caused by its calcium content, researchers say there are a variety of potential mechanisms that may explain those additional benefits and merit further research.

For example, some of the minerals in dairy products, such as phosphorous and magnesium, may enhance calcium's beneficial effects on fat breakdown within the cells. In addition, the proteins in dairy products may help preserve muscle and increase metabolism. But this results shouldn't be interpreted as license to indulge in dairy products in hopes of spurring weight loss. The bottom line of successful weight loss is still burning more calories than we take in.

SOURCES: M.Obesity Research, April 2004; vol 12: pp 582-590.

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