The goals of noninsulin-dependent diabetes mellitus (NIDDM) treatment are to reduce the incidence of symptomatic glycemic abnormalities, delay or lessen microvascular complications and reduce risk factors for macrovascular complications (eg, atherosclerotic vascular disease). The gastrointestinal tract has no clear role in the pathophysiology of noninsulin-dependent diabetes mellitus (NIDDM), but it may be an appropriate site for therapeutic intervention. Studies suggest that for patients with NIDDM, a calorie-restricted, high carbohydrate diet low in fat and rich in fibre may improve glycemic control, mitigate the risk of atherosclerosis and retard such diabetic complications as nephropathy and retinopathy. Increased meal frequency slows the rate of carbohydrate absorption, flattens blood insulin responses and reduces serum cholesterol. New therapeutic interventions, such as soluble fibre, low glycemic index foods or alpha glucosidase inhibitors, can further slow carbohydrate absorption and thus reduce secondary risks from hyperglycemia and hyperinsulinemia.

**Key Words:** Alpha glucoside inhibitors, Diabetes mellitus, Hyperglycemia, Hyperinsulinemia

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**PATHOPHYSIOLOGY OF NIDDM**

Individuals with NIDDM have impaired nutrient metabolism primarily because of insulin resistance in key target tissues (eg, liver, muscle, adipose tissue) (1). Insulin resis-

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**TMS WOLEVER.** Diet and the role of altered carbohydrate absorption in the treatment of noninsulin-dependent diabetes mellitus. *Can J Gastroenterol* 1996;10(1):29-36. The gastrointestinal tract has no clear role in the pathophysiology of noninsulin-dependent diabetes mellitus (NIDDM), but it may be an appropriate site for therapeutic intervention, specifically changes in diet, meal frequency and medications. Studies suggest that for patients with NIDDM, a calorie-restricted, high carbohydrate diet low in fat and rich in fibre may improve glycemic control, mitigate the risk of atherosclerosis and retard such diabetic complications as nephropathy and retinopathy. Increased meal frequency slows the rate of carbohydrate absorption, flattens blood insulin responses and reduces serum cholesterol. New therapeutic interventions, such as soluble fibre, low glycemic index foods or alpha glucosidase inhibitors, can further slow carbohydrate absorption and thus reduce secondary risks from hyperglycemia and hyperinsulinemia.

**Key Words:** Alpha glucoside inhibitors, Diabetes mellitus, Hyperglycemia, Hyperinsulinemia

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**RÉSUMÉ :** Le tractus gastro-intestinal ne joue pas de rôle défini dans la physiopathologie du diabète sucré non insulino-dépendant (DSNID), mais il peut constituer un domaine d’intervention thérapeutique approprié, spécialement en ce qui concerne l’alimentation, la fréquence des repas et la prise des médicaments. Selon certaines études menées chez des patients atteints de DSNID, un régime alimentaire pauvre en calories et riche en glucides, faible en gras et riche en fibres peut améliorer la maîtrise de la glycémie, réduire les risques d’athérosclérose et retarder les complications du diabète, dont la néphropathie et la rétinopathie. La prise de repas plus fréquents ralentit le taux d’absorption des glucides, régularise les réponses insuliniques sanguines et abaissent le cholestérol séré. De nouvelles interventions thérapeutiques comme la prise de fibres solubles, d’aliments à faible indice glycémique ou inhibiteurs de l’alpha-glucosidase, peuvent ralentir davantage l’absorption des glucides et ainsi réduire les risques d’hyperglycémie et d’hyperinsulinémie.

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Normal serum insulin, but insufficient to maintain euglycemia in response to Toxic effects of hyperglycemia on pancreatic function

<table>
<thead>
<tr>
<th>Insulin secretion</th>
<th>Fasting blood glucose (mmol/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Fasting</td>
<td>Normal to increased</td>
</tr>
<tr>
<td>2. Postprandial</td>
<td></td>
</tr>
<tr>
<td>a. Early phase</td>
<td>Normal to increased</td>
</tr>
<tr>
<td>b. Late phase</td>
<td>Normal to increased</td>
</tr>
<tr>
<td>c. Postprandial</td>
<td>Normal to increased</td>
</tr>
</tbody>
</table>

TABLE 1

*Normal serum insulin, but insufficient to maintain euglycemia in response to this glucose load

TABLE 2

CLINICAL IMPLICATIONS OF HYPERGLYCEMIA

For many years clinicians have suspected a relationship between glycemic control and the development of diabetic microvascular complications (e.g., retinopathy, neuropathy, nephropathy). Only recently, however, have convincing data become available to support the therapeutic value of good control of blood sugar levels in clinical practice.

The Diabetes Control and Complications Trial (6) and the Stockholm Diabetes Intervention Study (7) are two long term, multicentre studies that independently demonstrated that better than usual ('tight') glycemic control delayed the onset and slowed the progression of microvascular complications in persons with insulin-dependent diabetes mellitus (IDDM) (Tables 2, 3). Intensive therapy reduced the development of hypocholesterolemia, defined as a serum concentration of low density lipoprotein (LDL) cholesterol greater than 4.14 mmol/L, by 34%. Cardiovascular events were reduced by intensive therapy but this change was not statistically significant (0.5 events versus 0.8 events/100 patient-years). The relative youth of the patients made the detection of treatment-related differences in rates of macrovascular events unlikely.

Although individuals with NIDDM did not participate in these trials, it is believed that the results may apply to them because the mechanisms by which glucose causes complications are presumed to be the same in both types of diabetes (8,9). Glycemic control may be only one of several factors that contribute substantially to the onset or progression of microvascular complications in NIDDM (10,11). Further research is underway to determine the relationship between glycemic control and complications for people with NIDDM (12-14).

CLINICAL IMPLICATIONS OF HYPERINSULINEMIA

There are good data to suggest that hyperinsulinemia promotes the development of accelerated atherosclerosis in NIDDM (1,15). In one animal study on the effects of hyperinsulinemia, insulin administration stimulated proliferation of atheromas within blood vessels and increased cholesterol synthesis. Studies in humans confirm that insulin also plays a critical and permissive role in the endogenous synthesis of cholesterol (16), an important risk factor in atherogenesis.

Results of the Stockholm Diabetes Intervention Study: Glycemic control as secondary intervention in insulin-dependent diabetes mellitus

<table>
<thead>
<tr>
<th>Complications</th>
<th>Conventional therapy</th>
<th>Intensive therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients with development of serious retinopathy (%)</td>
<td>52</td>
<td>27</td>
</tr>
<tr>
<td>Nephropathy – patients developing albuminuria &gt;200 μg/min</td>
<td>9 patients</td>
<td>1 patient</td>
</tr>
<tr>
<td>Neuropathy – patients reporting symptoms at 7.5 years (%)</td>
<td>11</td>
<td>2</td>
</tr>
<tr>
<td>Mean glycosylated hemoglobin (%)</td>
<td>8.6</td>
<td>7</td>
</tr>
</tbody>
</table>

Data from reference 7

For years before the diagnosis of NIDDM is made, chronic mild elevation of blood glucose may contribute to the development of insulin resistance in the periphery. Chronic hyperglycemia is also believed to affect beta cells' ability to secrete insulin (Table 1).
Insulin resistance may also contribute to the hypertension that is observed in NIDDM and other insulin-resistant states by enhancing renal sodium reabsorption or by directly stimulating the sympathetic nervous system (18).

Although the relationship among human hyperinsulinaemia, insulin resistance and clinical diseases is not yet completely understood, these studies illustrate some of the ways in which hyperinsulinaemia may contribute to accelerated atherosclerosis in patients with NIDDM (15-18).

**ROLE OF DIET IN THE CLINICAL EXPRESSION OF NIDDM**

**Diet/gene interaction:** In animals with experimentally induced beta cell dysfunction, a high fat intake caused marked fasting hyperglycemia and exaggerated glycemic responses to stress. When animals with equivalent beta cell defects were fed high starch diets, they maintained normal insulin sensitivity and fasting blood sugar, and displayed only mild impairment of glucose tolerance (19).

Human studies also suggest that dietary intake affects the clinical expression of NIDDM. Among Pima Indians, known to be genetically predisposed to NIDDM, diet can be a critical factor in whether diabetes is expressed (20). One study showed declines in glucose tolerance, glucose disposal and beta cell sensitivity to insulin after subjects were switched from the traditional Pima diet (70% of calories from carbohydrates, 15% from fat) to a high fat modern diet (30% from carbohydrates, 50% from fat). Similar diabetogenic effects of the latter high fat diet were also found in Caucasians entered in that study (20).

**Diet and development of complications:** Diet may also play a role in the development and progression of diabetic complications although the results are not entirely consistent and more work is needed in the area. In one retrospective study (21), diabetics who developed retinopathy were found to have lower daily intakes of carbohydrate and fibre, and more calories from protein than diabetics who did not develop retinopathy.

Another study showed that lipid lowering by a low fat diet and/or hypolipidemic drugs improved hard retinal exudates, decreased microaneurysms and improved overall vision in patients with NIDDM with established diabetic retinopathy (22). It has also been reported that there is an increased incidence of certain types of cataracts among diabetic individuals who have diet-related vitamin and mineral deficiencies (23).

**CURRENT NUTRITIONAL RECOMMENDATIONS AND CONTROVERSIES**

**Weight loss and glycemic control:** The nutritional intervention recommended most frequently for patients with NIDDM is weight loss. Obesity complicates and worsens glycemic control for persons with NIDDM by increasing insulin resistance in both the liver and peripheral tissues (2).

There are many approaches to weight loss including low calorie diets, low fat diets combined with exercise and protein-sparing modified fasts. Although long term compliance with weight loss plans is difficult for many obese persons with NIDDM, current recommendations favour an energy controlled, high fibre diet in combination with exercise and/or behaviour modification counselling (24). For sustained weight reduction, a weight loss of no more than 1 kg/week is recommended. Theoretically this can be achieved with a reduction in energy intake of 500 Kcal/day, but the level of energy intake recommended needs to be individualized and depends on many factors, such as the level of physical activity undertaken by the individual. Glycemic improvement due to weight reduction may be predicted after a weight loss of only 2.3 to 4.5 kg.

Despite the theoretical value of weight loss for obese persons with NIDDM, some who lose weight do not show improvement in blood glucose control (25). The effectiveness of weight loss as a clinical intervention may depend on the duration of diabetes, and the individual’s insulin reserve and maintenance of the lower body weight (26). Recent studies suggest that the improvement in blood glucose control is more a function of reduced nutrient load rather than reduced adipose tissue mass per se (27). Thus, after a period of weight loss on a hypocaloric diet, when a weight-maintaining diet is reestablished, blood glucose levels begin to rise again (28).

Patients whose blood sugar control does not improve after a trial of weight loss may require exercise, oral hypoglycemics or other pharmacological interventions to achieve glycemic control (25).

**Canadian Diabetes Association dietary recommendations:** The Canadian Diabetes Association recommends a calorie-restricted, low fat, high carbohydrate, protein-limited, fibre-rich diet for all individuals with NIDDM (29). According to these recommendations, less than 30% of total calories should come from fat, 55 to 60% from carbohydrates and 12 to 20% from protein, with up to 40 g/day of fibre. The reduction of fat should be achieved primarily by a reduction of saturated fatty acids to less than 10% of energy. Polyunsaturated fat should contribute no more than 10% of energy with the remainder supplied by monounsaturated fat.

**Dietary carbohydrate:** There is some controversy regarding the purported benefits of the high carbohydrate intake recommended for persons with NIDDM. Some data suggest that an increased carbohydrate intake may increase blood glucose and insulin, temporarily increase triglyceride concentrations and reduce beneficial high density lipoprotein (HDL) cholesterol (30). However, these changes may be transient and are not universal findings (31,32).

Some research suggests that, in subjects with NIDDM, replacing saturated fat with monounsaturated fat may benefit lipid metabolism more than increasing the percentage of carbohydrate consumed (33). However, other studies were unable to demonstrate significant glycemic or lipid improvements due to increasing monounsaturated fat intake compared with high carbohydrate intake (31). A recent, large, multicentre trial compared the effects of a diet containing 55% of energy as carbohydrate and 30% as fat with a diet of 40% carbohydrate and 45% fat (high monounsaturated fat) in 42 subjects with NIDDM who consumed each
diet for six weeks (34). There was no significant difference
between the diets on serum LDL or HDL cholesterol. The
high carbohydrate diet produced a statistically significant,
but small, increase in serum triglycerides from 1.75 to 2.19
mmol/L (P<0.0001). There was a significant increase in
postprandial glucose and insulin with the high carbohydrate
diet; however, a significant effect on the major component
of adult hemoglobin (HbA1C) could not be demon-
strated (7.9% for the monounsaturated fat diet versus 8.2%
for the high carbohydrate diet [not significant]). Thus, the
issue of how much carbohydrate should be in the diabetic
diet is still a matter of debate. If high carbohydrate intake is
attempted, it may be important that the type of carbohydrate
be unrefined, high in fibre and low in glycemic index. These
types of carbohydrate are preferable because they may allow
carbohydrate intake to be increased without increasing
blood glucose, insulin and triglycerides (33).

The use of sugar by diabetic patients has traditionally
been forbidden. However, consumption of sucrose (table
sugar) produces a blood glucose response similar to or even
lower than an equal amount of carbohydrate from most ‘co-
plex’ carbohydrate foods such as bread (35). It has actually
been shown recently that some sucrose-sweetened breakfast
cereals produce a lower blood glucose response than an
equivalent amount of the same unsweetened cereal (36).
This is primarily because fructose produces a very low blood
glucose response (35), and the molecule of sucrose contains
glucose and fructose. In controlled studies in diabetes,
isoenergetic replacement of starch with modest amounts of
sucrose (28 to 45 g/day) has no effect on blood glucose or in-
sulin in patients with diabetes (37-39). Even as much as 220
g sucrose/day in NIDDM patients has no effect on blood glu-
 cose control or blood lipids compared with a diet contain-
ing less than 3 g sucrose/day (40). There is some evidence
that fructose as a sweetener actually improves glycemic con-
trol (41,42). There is some concern that fructose may raise
blood lipids in susceptible individuals (41) but this is not a
universal finding (42). Thus, dietary recommendations are
beginning to acknowledge that the use of modest amounts of
sugar is acceptable. The problem is in defining what ‘modest’
means. The Canadian Diabetes Association suggests that
the rather unrealistically low amount of 3 to 5 g of sucrose may
be used by people with diabetes if glycosylated hemoglobin is
normal or near normal. The concern of most dietitians re-
garding sugars is that it is easy to consume a large amount of
carbohydrate and thus energy, which in turn influences
blood glucose responses. Thus, while equal carbohydrate por-
tions of whole fruits and fruit juices produce nearly the same
blood glucose responses (35,43), in practice, for example, a
serving of apple juice (250 mL) has a greater effect on blood
glucose than one whole apple because the juice contains
nearly twice as much carbohydrate as a serving of a whole
apple.

Dietary protein: Low protein intake has been proposed as a
strategy to reduce the risk of diabetic nephropathy. Al-
though not yet demonstrated in subjects with NIDDM, low
protein intake has been shown to preserve renal function
and slow the progression from early nephropathy to renal
failure in persons with IDDM (44-46).

There is some evidence to suggest that reduced protein
intake slows the development of diabetic retinopathy. How-
ever, other data suggest that the development of retinopathy
may also be affected by hypertension, ageing, long term lev-
els of glycemic control or some combination of all three
(11,21).

Another proposed rationale for reducing the protein in-
take of persons with NIDDM is to decrease the stimulating
effects that ingested protein may have on insulin secretion
and cholesterol synthesis (47). It remains unknown whether
the theoretical advantage of protein restriction has practical
value in devising food plans for individuals with NIDDM.

Dietary fat and cholesterol: Low fat diets have long been
proposed as a method to reduce serum LDL cholesterol, and
hence the risk of cardiovascular morbidity and mortality, in
the general adult population. This may be a particularly im-
portant risk reduction strategy for persons with NIDDM.

Cardiovascular disease (CVD) risk in diabetic patients is di-
rectly related to their levels of blood pressure, cigarette
smoking and serum cholesterol. However, at any level of
these risk factors, diabetic patients probably have three times
the risk of CVD versus nondiabetics (48). Thus, levels of se-
rum cholesterol that would be considered acceptable in a
nondiabetic may impart considerable CVD risk in an indi-
vidual with diabetes because in diabetics, lipoprotein parti-
cles are modified by glycation, aggregation and oxidation;
glycated LDL particles are cleared very slowly from the body,
contribute to the production of foam cells and are thought to
stimulate atherogenic pathophysiology (49).

There is some uncertainty concerning the significance of
fasting versus postprandial lipid abnormalities in NIDDM.
However, some research in patients with NIDDM suggests
that fasting hypertriglyceridemia may be a very important
predictor of rapidly evolving atherogenic changes in post-
prandial lipids and lipoproteins. These postprandial lipid
changes can potentiate the already unfavourable athero-
genic fasting lipid profiles of many patients with NIDDM
(50). Detection of fasting hypertriglyceridemia may be an
important opportunity for intensification of diet and other
therapeutic efforts (eg, smoking cessation) to mitigate excess
risk of atherosclerosis among patients with NIDDM.

For many patients with NIDDM blood lipids can be man-
aged through weight loss, adherence to a low fat, high fibre,
high carbohydrate diet or through pharmacological achieve-
ment of glycemic control (29,47). For others, an individu-
alized evaluation of the etiology of the lipid abnormality and
introduction of selected antilipemic medications may be
necessary.

Increased meal frequency: Some researchers have explored
increased meal frequency as a therapeutic strategy in
NIDDM. The consumption of one large meal produces a gly-
cemic challenge. In response, post-meal rises in insulin are
followed by a rebound of counterregulatory hormones 180
mins later (eg, glucagon and growth hormone). However,
when a person eats or sips the same nutrients slowly, blood in-
sulin responses are flattened and there is continued suppression of counterregulatory hormones during the postprandial phase.

Healthy volunteers who consumed 17 small meals/day for two weeks had a 25% reduction in daytime insulin levels compared with a group who ate the identical diet as three meals/day (51). The former subjects experienced a 20% decrease in cholesterol and marked reductions in LDL and apolipoprotein-B due to increased meal frequency (51). These lipemic effects of increased meal frequency may be especially beneficial for patients with NIDDM because of their increased risk of accelerated atherosclerosis (49,52).

A recent study in patients with NIDDM confirmed that a daily diet of 13 small meals lowers blood glucose, serum insulin, C-peptide and triglycerides compared with an isocaloric, three meals daily diet of identical composition (53). These and other studies suggest that higher than normal meal frequency may be a useful way to limit glucose excursions and to reduce resulting insulin and free fatty acid levels in patients with NIDDM (54,55).

The long term importance of increased meal frequency remains unknown because there are no long term trials in diabetic subjects to indicate the optimal number of meals/day. The Canadian Diabetes Association recommends at least three meals/day, with snacks added according to patient preference and the hypoglycemic agents used (56). The British Diabetes Association recommends small, frequent meals (47). The American Diabetes Association is flexible in the number of daily meals but recommends that calories be spread evenly throughout the day, which helps to avoid a large concentration at one meal and allows time for postprandial glucose levels to return to preprandial levels before eating again (57).

Compliance with small, frequent meals may be difficult given our modern lifestyle. Erratic eating patterns, fasting, binging and gross malnutrition are not uncommon among North American adults. When frequent meals are considered as a treatment modality the therapeutic benefit:risk ratio is high and the cost is low. Nonetheless, compliance with frequent meals may be as difficult for patients as compliance with multiple medication dosages. However, if compliance can be elicited through effective provider-patient communication, increased meal frequency may be useful in the treatment of NIDDM (58,59).

### TABLE 4
Examples of common foods and their glycemic index*

<table>
<thead>
<tr>
<th>Food</th>
<th>Glycemic index</th>
<th>Food</th>
<th>Glycemic index</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breads</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rye (whole grain pumpernickel)</td>
<td>68</td>
<td>Snack foods</td>
<td></td>
</tr>
<tr>
<td>Rye (crispbread)</td>
<td>95</td>
<td>Corn chips</td>
<td>99</td>
</tr>
<tr>
<td>Wheat (white or wholemeal)</td>
<td>100</td>
<td>Potato chips</td>
<td>77</td>
</tr>
<tr>
<td>Pasta</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Macaroni</td>
<td>64</td>
<td>Vegetables</td>
<td></td>
</tr>
<tr>
<td>Spaghetti (wholewheat)</td>
<td>61</td>
<td>Potato (instant)</td>
<td>120</td>
</tr>
<tr>
<td>Spaghetti (white)</td>
<td>67</td>
<td>Potato (mashed)</td>
<td>98</td>
</tr>
<tr>
<td>Pasta</td>
<td></td>
<td>Potato (boiled)</td>
<td>80</td>
</tr>
<tr>
<td>Macaroni</td>
<td>64</td>
<td>Potato (sweet)</td>
<td>70</td>
</tr>
<tr>
<td>Spaghetti (wholewheat)</td>
<td>61</td>
<td>Baked beans (canned)</td>
<td>70</td>
</tr>
<tr>
<td>Spaghetti (white)</td>
<td>67</td>
<td>Chick peas (canned)</td>
<td>60</td>
</tr>
<tr>
<td>Cereal grains</td>
<td></td>
<td>Green peas (frozen)</td>
<td>65</td>
</tr>
<tr>
<td>Barley</td>
<td>36</td>
<td>Kidney beans (canned)</td>
<td>74</td>
</tr>
<tr>
<td>Buckwheat</td>
<td>78</td>
<td>Lentils (red)</td>
<td>38</td>
</tr>
<tr>
<td>Bulgur</td>
<td>65</td>
<td>Peanuts</td>
<td>15</td>
</tr>
<tr>
<td>Millet</td>
<td>103</td>
<td>Fruits</td>
<td></td>
</tr>
<tr>
<td>Rice (polished or brown)</td>
<td>81</td>
<td>Apple</td>
<td>52</td>
</tr>
<tr>
<td>Rice (parboiled)</td>
<td>68</td>
<td>Banana</td>
<td>84</td>
</tr>
<tr>
<td>Corn</td>
<td>80</td>
<td>Raisins</td>
<td>93</td>
</tr>
<tr>
<td>Breakfast cereals</td>
<td></td>
<td>Sugars</td>
<td></td>
</tr>
<tr>
<td>All Bran</td>
<td>74</td>
<td>Fructose</td>
<td>26</td>
</tr>
<tr>
<td>Corn Flakes</td>
<td>121</td>
<td>Glucose</td>
<td>138</td>
</tr>
<tr>
<td>Muesli</td>
<td>96</td>
<td>Sucrose</td>
<td>83</td>
</tr>
<tr>
<td>Oat bran</td>
<td>80</td>
<td>Lactose</td>
<td>57</td>
</tr>
<tr>
<td>Puffed rice</td>
<td>132</td>
<td>Dairy products</td>
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<tr>
<td>Puffed wheat</td>
<td>110</td>
<td>Ice cream</td>
<td>69</td>
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<tr>
<td>Shredded wheat</td>
<td>97</td>
<td>Skim milk</td>
<td>46</td>
</tr>
<tr>
<td>Cookies</td>
<td></td>
<td>Whole milk</td>
<td>44</td>
</tr>
<tr>
<td>Oatmeal</td>
<td>78</td>
<td>Yogurt</td>
<td>52</td>
</tr>
<tr>
<td>Matzo</td>
<td>100</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Shortbread</td>
<td>88</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*The glycemic index (GI) is a classification of the blood glucose raising potential of different foods defined as: GI = 100 x F/B, where F is the incremental area under the blood glucose response curve to a 50 g available carbohydrate portion of a food and B is the glycemic response to a 50 g carbohydrate portion of white bread taken by the same subject. Based on reference 35
NEWER THERAPEUTIC APPROACHES VIA THE GUT

There are several nutritional and pharmacological means to mimic the actions of increased meal frequency and to slow the rate of carbohydrate absorption from the gut (53). Clinical studies using soluble fibre, low glycemic index foods or alpha glucosidase inhibitors have demonstrated that carbohydrate absorption can be slowed and redistributed throughout the intestine with significant therapeutic benefit. Slowing absorption lowers peak postprandial serum blood sugar levels and shortens the duration of hyperglycemia. These changes slow the secretion of insulin, and reduce postprandial rebound of free fatty acids and counterregulatory hormones. This results in more efficient carbohydrate metabolism and reduced secondary risks from hyperglycemia and hyperinsulinemia.

DIETARY FIBRE

Viscous forms of dietary fibre have been used to improve metabolic control in diabetes by slowing carbohydrate absorption. Guar gum, gum tragacanth and psyllium fibre reduce postprandial blood glucose and insulin levels in patients with NIDDM and may decrease cholesterol and triglyceride levels (60,61). In general, purified viscous fibres have been shown to produce statistically significant long term reductions in blood glucose and cholesterol in 60 to 80% of studies, whereas the more palatable nonviscous fibres (eg, wheat bran) have shown positive results in only about 20% of studies (62). However, viscous fibre may not be effective in poorly controlled diabetic patients (63) nor provide any additional benefit compared with a low calorie diet in newly diagnosed or obese patients (64).

A major problem with the use of purified viscous fibres in diabetes management is its poor availability. Palatable, fibre-enriched foods are not widely available except in the form of a commercial psyllium-enriched cereal (65).

The way in which fibre is consumed has also been shown to be important. Fibre must be intimately mixed with food to have a salutary effect on blood glucose (65) and serum cholesterol (66). Attempts to ingest fibre packaged in gelatin capsules have generally not been shown to be effective.

The use of foods naturally high in fibre may be preferable to the use of purified fibre supplements. However, wheat and other cereal fibres, which are most familiar to the North American palate, have minimal short or long term effects on blood glucose. Diets rich in foods that are high in soluble fibre (eg, legumes and barley) have been shown to improve blood glucose control, but are less familiar and unpalatable to some. Thus, the practical benefits of high fibre diets have been seen as equivocal by diabetologists throughout North America.

GLYCEMIC INDEX AND DIET

Independent of fibre content, some foods are naturally absorbed at slower rates (Table 4). For example, breads containing a high proportion of whole cereal grains are more slowly absorbed and result in lower postprandial glucose levels versus breads made with milled flour (35,67).

An intake of low glycemic index foods has been shown to reduce fasting blood glucose and HbA1C, reflecting improved long term glycemic control (68). Low glycemic index foods may also have useful effects on blood lipids and renal function (68). The inclusion of low glycemic index foods into the diet may be an effective, inexpensive way to improve blood glucose control without increasing insulin demand (69). However, before the full use and clinical evaluation of the glycemic index can be implemented, further research is needed to categorize foods fully and to assess their effect in mixed meals (eg, carbohydrates taken with proteins and fats) (70).

ALPHA GLUCOSIDASE INHIBITORS

Also known as intestinal disaccharides inhibitors, alpha glucosidase inhibitors have been shown to blunt postprandial hyperglycemia by delaying the absorption of carbohydrates. Acarbose, a new and promising alpha glucosidase inhibitor, is an oligosaccharide extracted from culture broth of the fungus actinomycetes. Acarbose is taken as a chewable tablet or sprinkled onto food and is minimally absorbed. It competitively inhibits carbohydrate absorption in the proximal small intestine by preferentially binding to brush border enzymes (ie, glucoamylase, sucrase maltase, isomaltase). This drug’s net effect is to delay carbohydrate absorption until nutrients reach the ileum (71,72). Thus, acarbose prolongs absorptive time and flattens the blood sugar, insulin and gastric inhibitory peptide responses. The effect of acarbose on blood lipids is variable and there is no evidence that insulin sensitivity in the peripheral tissues of NIDDM patients is improved by the use of alpha glucosidase inhibitors (71).

Used alone or in combination with oral agents, acarbose has been shown to improve blood glucose control in patients with NIDDM (71,72). Other studies have shown monotherapy with acarbose to be equal in efficacy to sulphonylureas, as demonstrated by improvements in fasting and postprandial blood sugar levels and glycohemoglobin.

Several other alpha glucosidase inhibitors are also under development (eg, AO-128, emiglitate and miglitol). In addition to glycemic control benefits, some of these may promote weight loss in obese patients with NIDDM (73).

PRACTICAL ISSUES IN NUTRITIONAL APPROACHES TO DIABETES MELLITUS MANAGEMENT

Compliance: Patient compliance with dietary regimens is notoriously difficult. However, research has shown that intensive patient education, behaviour modification counselling or both can elicit significant improvements in glycemic control, weight loss and quality of food choices in NIDDM patients (74,75). Meal planning is often based on the Canadian Diabetes Association recommendations, but can also be individualized to accommodate cultural and personal preferences. Compliance is improved when the patient and family...
participate in setting weight loss goals and meal planning. Efforts to improve physician-patient communication may also be essential to the success of low calorie, weight loss diets and other dietary interventions for NIDDM (58,59).

Home blood glucose monitoring and patient response: Home blood glucose monitoring to guide dietary intake and/or physical exertion may be underused by patients with NIDDM (76,77). A recent survey of diabetes management practices suggests that most diabetics are not instructed to use home blood glucose monitoring data in this manner (76). Consequently, many patients who self-monitor blood glucose may modify their insulin or oral hypoglycemic agent dosages, but not alter their food intake or exercise patterns in response to blood sugar abnormalities (77). Further research is needed to identify and educate NIDDM patients who would benefit from self-adjustment of dietary intake (and/or exercise patterns) on the basis of home blood glucose monitoring (76).

Counselling to promote weight loss or maintain ideal body weight is also important to NIDDM management. However, not all physicians have the time or skills to counsel patients effectively. One study reported a 50% discrepancy between patient and physician understanding of overall treatment goals, weight loss goals and blood glucose goals (78).

Diabetes educators may be helpful to promote weight loss and early symptom recognition, to teach home blood glucose monitoring and to enhance family support for NIDDM patients (79). Patient counselling by physicians alone may not be effective or sufficient, and early referral to dieters or other health care educators is important within the context of contemporary diabetes management (80,81).

CONCLUSIONS

In addition to the standard treatment options, there are several innovative dietary and pharmacological ways to approach the management of NIDDM.

Research has shown that hyperinsulinemia, hyperglycemia and other metabolic abnormalities can be significantly affected by manipulating gastrointestinal function. It may be possible for patients with NIDDM to improve glycemic control and reduce atherosclerotic risk factors through an intake of low glycemic index foods, a high fibre diet, increased meal frequency or the use of alpha glucosidase inhibitors.

As a broader array of treatment approaches becomes available for patients with NIDDM, physicians and other members of the diabetes health care team will need to work closely with patients and their families to realize the benefits of advances in NIDDM management (82).

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