# Part 1: Dietary And Lifestyle Factors In The Management Of Type 2 Diabetes Mellitus

## Matthew Boylan

**Abstract**

Type 2 diabetes is reaching epidemic proportions in many Western countries. Though a multifactorial disease, diet and lifestyle are often major aetiological factors in the development of type 2 diabetes. It is accepted that modifications of diet and lifestyle will play a major role in preventing type 2 diabetes. The usefulness of the glycaemic index and glycaemic load in managing type 2 diabetes is discussed. It is concluded that the evidence regarding the usefulness of the glycaemic index and glycaemic load is equivocal, with their most effectual role being limited to preventive measures.

## Introduction

Diabetes is one of the 'most costly and burdensome chronic diseases of our time', and is increasing world-wide at epidemic proportions[1].

At the turn of the century it was estimated that 150 million people had developed diabetes, and that this number is expected to double by 2025. Type 2 diabetes is far more prevalent than type 1 diabetes, and in the developed world type 2 diabetes is the fourth or fifth leading cause of death, as well as a significant cause of morbidity[1,2].

Australia is unfortunately, but not unexpectedly, well entrenched in this world-wide predicament. Figures from the International Diabetes Institute released by the Federal Health Minister, Tony Abbott, on 15 May 2006 revealed that 100,000 Australian adults develop diabetes each year[3]. The Institute's Director, Professor Paul Zimmet, reported that in 1999/2000 one million Australians had diabetes and two million were pre-diabetic.

The current cost to the Australian health system is more than $3 billion and is expected to rise dramatically[4]. Clearly, the prevention and management of type 2 diabetes is a major health priority in Australia.

Offering more than just hope however is a significant body of evidence that shows that type 2 diabetes may be prevented or delayed by lifestyle changes[5]. Indeed lifestyle changes have been shown to be nearly twice as effective in preventing diabetes than glucose–lowering drugs[1]. Nutritional therapy has also long been accepted as an essential component of diabetes management[6,7].

This emphasis on lifestyle factors was re-iterated by Mr Abbott who called for a 'culture shift' in the Australian way of life. Mr Abbott said:

> In the end, this is not going to be beaten by governments. It's going to be beaten by people. Government can't make people walk to work. We can't make people walk to school. We can't make people give up the Magnum (ice cream)[3].

As natural therapists we too are unable to make people walk to school or work or eat less ice cream. However a key strength of our practising philosophy is the time we spend with our patients, and our emphasis on our patients achieving and maintaining a healthy lifestyle. Therefore we are well placed to lead the way in achieving the required culture shift to prevent and manage this epidemic.

In this series of articles, the role of diet and lifestyle modifications in the prevention, delay and management of type 2 diabetes will be reviewed. The literature on this topic is immense, and this series is not a systematic review of the literature. This series will however discuss the evidence regarding the efficacy of diet and lifestyle measures that are commonly considered for the management of type 2 diabetes. Although the focus will be exclusively on type 2 diabetes, some of the interventions discussed might also be appropriate in the management of type 1 diabetes. This series of articles will not discuss specific treatment options such as herbs, nutritional supplements etc.

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First, a very important caution. Under no circumstances should a patient with diabetes be advised to cease or reduce their prescribed medication. If insulin or other medication is ceased, the result may be fatal.

The dietary and lifestyle modifications to be discussed may prevent, or at least delay, the need for medication, for those who have not yet developed diabetes. These modifications will not replace the medication once it has been prescribed.

Following a brief overview of the aetiology and pathogenesis of type 2 diabetes, this first article will look at the role of the glycaemic index and glycaemic load in preventing and managing type 2 diabetes. The discussion is based on the literature and could be contentious and may surprise. In subsequent articles the roles of specific dietary components, exercise and other lifestyle factors will be examined.

## Glucose Metabolism in the Healthy Body

It is useful to very briefly outline normal physiology. Eating starch or sugar will, after metabolism primarily within the liver, elevate plasma glucose levels which in turn will result in an increased take up of glucose by many cells, including the β-cells of the pancreas.

Consequently, and after several intermediary steps, insulin containing vesicles located in the membrane of the pancreatic β-cells undergo fusion which causes their insulin contents to be dumped into the extracellular fluid. The released insulin then binds to receptors on target muscle and adipose tissue (fat) cells.

This binding by insulin stimulates intracellular glucose transporter proteins within the muscle and adipose cells membranes, allowing for an increased transport of glucose into the cells[8]. This process is tightly controlled in the healthy body so that plasma glucose levels are maintained within a narrow range of 3.5—6.5 mmol/L[9].

## Pathogenesis of Type 2 Diabetes Mellitus

Type 2 diabetes develops initially as an insulin resistance, where muscle and adipose tissue cells fail to respond normally to insulin. This resistance results in the pancreatic β-cells attempting to compensate for the excess plasma glucose by secreting additional insulin into the bloodstream.

Initially these compensatory mechanisms are successful and plasma glucose levels return to normal. However after a number of years the pancreatic β-cells are unable to cope with this extra workload and begin to fail. An insulin deficiency occurs, and plasma glucose levels rise and remain beyond the healthy range. Type 2 diabetes has manifested[8].

Type 2 diabetes may be characterised as defects in insulin secretion and insulin action. Indeed insulin resistance by itself is insufficient to result in diabetes. A concomitant pancreatic β-cell impairment is required.

Diabetes is often not seen alone, but is associated with obesity, hypertension and hyperlipidaemia. This cluster of conditions is commonly referred to as 'metabolic syndrome' or 'syndrome X'[9].

## Aetiology of Type 2 Diabetes Mellitus

Type 2 diabetes is a complex disease. The development of type 2 diabetes has been linked to obesity, as obese people experience both increased resistance to insulin and increased glucose production by the liver. Another theory links an increased concentration of free fatty acids to, ultimately, inhibition of glucose uptake by peripheral cells (insulin resistance), culminating in consequent pancreatic β-cell exhaustion. This leads to decreased insulin secretion, overproduction of glucose by the liver and peripheral under-utilisation of the increased glucose[10].

The main risk factors for type 2 diabetes are excessive energy intake with associated obesity and inadequate physical activity. Diets high in saturated fat and/or low in fibre also increase the risk for type 2 diabetes[11]. Increased age is an additional important risk factor for type 2 diabetes[9].

Genetics are also an important consideration. Although most middle aged type 2 diabetic patients are obese, most obese people are not diabetic. Thus obesity, as well as other environmental factors, may act as pre-cursors for the development of diabetes in those who are genetically predisposed to develop type 2 diabetes[9].

## Managing the Diabetic Epidemic

Because dietary and lifestyle factors play an important role in the pathogenesis of type 2 diabetes, there has been an increased focus on measures that are effective in preventing the development of type 2 diabetes[10]. This is an area where natural therapists are well placed to lead the way. This series of articles hopes to assist natural therapists take that lead, and begins by looking at two commonly referred to measures when discussing type 2 diabetes prevention and management, the glycaemic index and glycaemic load of foods.

## Glycaemic Index Defined

In simple terms, the glycaemic index (GI) is a measure of the impact of a food containing 50 g of available carbohydrate on the body's glycaemic response, expressed relative to that impact resulting from 50 g of oral glucose[12]. Foods whose available carbohydrate breaks down quickly may be said to consequently cause a high and fast blood glycaemic response, and are regarded as having a high GI.

The GI of glucose is expressed as 100. A GI value of or above 70 is regarded to be high, GI values of 56 to 69 are considered as medium, while GI values of and below 55 are low[13]. Some GI scales use 50 g of the available carbohydrate portion of white bread as the reference instead of glucose. This results in some foods which have a GI higher than that of white bread as also having a GI greater than 100. To avoid confusion, the use of 'the glucose = 100 scale is now recommended'[13].

## Glycaemic Load Defined

The blood glucose responses to a meal depend not only on the GI of the available carbohydrate, but also the amount of that available carbohydrate in the meal. This combined impact is described as the 'glycaemic load' (GL). The GL is obtained by multiplying the GI of a food by the amount of available carbohydrate per serving and dividing by 100[13].

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## Available Carbohydrate?

Both the above definitions refer to 'available carbohydrate'. By definition, this term means carbohydrate 'that is absorbed via the small intestine and used in metabolism'[12].

## GI and GL in the Management of Type 2 Diabetes

Perhaps surprising, the role of the GI and/or GL of foods is a much debated subject, with conflicting conclusions. In their authoritative book, Brand-Miller et al state in large font on page 35 'The GI is a clinically proven tool in its applications to diabetes, appetite and coronary health'[13]. It is consistently accepted in the literature that there is a body of studies which link high GI diets with the development of insulin resistance and type 2 diabetes.

Examples of these studies include a 2004 cohort study involving 2,834 subjects which revealed, among other things, that diets with a lower GI and GL were associated with lower insulin resistance[14]. Two other studies published by Salmeron et al in 1997 supported this view[15,16].

The first study was a cohort of 65,173 American women aged 40 to 65 years who had not developed diabetes as at 1986 when they completed diet questionnaires. The results obtained over a 6 year follow up suggested that 'diets with a high glycemic load and a low cereal fiber content increase risk of diabetes in women'[15].

In the second study a similar cohort was considered,  but this time involving 42,759 men aged 40 to75 years. Again the authors found that a diet with a high glycaemic load and low fibre increased the risk of men developing type 2 diabetes[16].

Livesy found that low GI carbohydrate diets eaten ad libitum resulted in lower body weight than high GI carbohydrate diets eaten ad libitum. He went on to suggest that perhaps GL might be a stronger explanation for this finding than GI, and that both restricted GI and GL diets might offer some protection from diabetes. However the data was insufficient to determine if the response to low and high GI diets might vary between people with high and low body mass indexes[12].

Another cohort study published in 2004 followed 91,249 women for 8 years following their completion of food questionnaires. This study found that 'after adjustment for age, body mass index, family history of diabetes, and other potential confounders, glycemic index was significantly associated with an increased risk of diabetes'. This association did not extend however to GL, and the authors conclusion was that 'diets with a high glycemic index and low in cereal fiber increase the risk of type 2 diabetes'[17].

Brand-Miller et al published in 2003 a meta-analysis of 14 studies comprising of 356 subjects. The studies were generally short term. Brand-Miller et al commented that these limitations 'tend to reduce rather than increase the chance of a significant finding.'

Taking these limitations into account, their analysis revealed that low GI foods in preference to high GI foods offer a 'small but clinically useful effect on medium term glycemic control in patients with diabetes.

The incremental benefit is similar to that offered by pharmacological agents …'[18]. This finding is consistent with the European, Canadian and British nutritional guidelines that promote the use of low GI foods in the management of diabetes[7]. Nevertheless some concerns must exist regarding the balance and quality of the trials considered in this meta-analysis.

## The Case Against GI/GL

The preceding summary provides a selection  of the evidence that suggests that dietary GI and GL might be associated with insulin resistance related diseases. However Liese suggests that the overall body of evidence 'is truly equivocal'[19].

This 2005 study by Liese et al involved 979 adults. A 'remarkable degree of consistency' in results demonstrating no association of GI, GL and carbohydrate intake with insulin sensitivity, insulin secretion and adiposity was found[19].

Another study published in 2005 considered food frequency questionnaires completed in 1999 by 5,675 subjects aged 30 to 60 years. The results did not support the position that a habitual intake of high GI and high GL foods increased a person's probability of experiencing insulin resistance[20].

A 2006 review analysed 1,255 subjects who completed food questionnaires during 1994—96, as well as 813 of the original participants who returned for follow up examinations during 1998—99. The results questioned the relationship between dietary GI and 'any measure of glycaemia' when examined in the context of the usual diet. Indeed the researchers went as far as to suggest that their results:

> taken together with the inconsistencies in the published literature for the effect of dietary GI on diabetes incidence, may be due primarily to inadequate utility of the GI to capture the true metabolic impact of foods consumed as part of a usual diet[21].

In her review of macronutrient considerations for persons who were pre-diabetic, Wien noted that the American Diabetes Association 2003 evidence-based review had concluded that the amount of carbohydrate was more important than its GI ranking in the medical nutrition treatment and prevention of diabetes. That Association's treatment strategy statement (as at 2006) advised that there was no strong evidence 'to recommend the use of a low-GI diet as a primary nutrition strategy in food/meal planning'[22]. This is in contrast to the European, Canadian and British nutritional guidelines mentioned above.

## Why the Discrepancies?

The short answer to this question is that the use of GI and GL in the setting of a typical daily diet for the average person is not an exact science. Several factors confound that situation and may have influenced the results summarised above.

Firstly, it is difficult to accurately compare different studies looking at dietary intake in relation to insulin response. One problem is that it 'must be considered that a gene-environment interaction may be of importance'[20]. The variations in genetic predispositions and environmental factors were generally, if at all, not considered in the preceding studies.

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A further problem exists in that the low GI (commonly 39—41) diets used in most experiments are well above the average low GI (commonly 48—50) in many populations. Thus 'diets consumed by free-living individuals may not approach the glycemic index values needed to achieve potential'[19].

Many of the studies relied upon food frequency questionnaires. These questionnaires were not usually designed to gather specific information on GI values, and various factors might have effected their accuracy[19,20]. For example the amount of available carbohydrate of a food may vary from region to region and season to season[20].

GI values will also vary depending upon how the food has been prepared eg porridge made from larger oats has a lower GI (40) than porridge made from finer oats (61); fibre produces different results, for example wheat fibre has only negligible GI effects when white bread is compared to wholemeal bread, yet potato skin has a significant effect on the GI of jacket potatoes; increasing maturity of a food tends to increase its GI[23]. The completion of the food frequency questionnaires may also have been subject to recall bias[20].

## Clinical Considerations

The above summary of the studies is not complete. There exist many more, both for and against the use of GI and GL in managing diabetes. No pre-determined selection criteria however was adopted in selecting the papers, other than to consider studies primarily published within the last 5 years. Thus it is hoped the above does present a balanced, albeit incomplete, view of the current body of evidence.

The preceding summary presents a very confusing position regarding the desirability or otherwise of utilising GI and GL values when preparing dietary advice for a person who has concerns about insulin resistance or diabetes. That confusion is further exacerbated when many of the studies recommend that more research be undertaken for clarification.

It is clear however that doubts have been established regarding the use of GI tables as a primary foundation for the formulation of dietary recommendations to patients with insulin resistance/diabetes concerns. It is apparent that the amount of carbohydrate eaten will be important, and thus the GL may be of more use than the GI. This will be especially so when considering foods within a narrow GI range.

It must also be kept in mind however that diets which have a low GI will generally be healthier in that there will be a lesser reliance on refined foods, and more reliance on unprocessed natural foods. To this end the relationship between GI and nutrient intake has been examined and briefly reported. In considering a diet with a mean GI of 59, it was found that a lower GI value was associated with higher amounts of fibre, lactose and galactose, and lower amounts of saturated fat and starch.

These 5 nutrients explained 62% of variance in GI. When whole foods were considered, lower amounts of low fat milk and fruits were associated with a lower GI value, while increased quantities of beer, white bread and fries was associated with higher GI values. These 5 foods explained 66% of variance in GI.

The authors concluded that the average GI of a diet appears to reflect more dimensions of diet than just quality of carbohydrates, such as the combination of foods consumed[24].

The preceding findings highlight that a diet with a low GI will generally be a diet that provides increased fibre. Fibre will be discussed more in the next article of this series, but if one consistent finding came out of the studies considered to date, it was that fibre plays an important role in the management of the body's insulin response.

The considered evidence is too conflicting and/or imprecise to make definite suggestions regarding the use of GI or GL in providing dietary advice. It is thus suggested that GI values not be used as a sole or primary reference when providing dietary advice for the diabetic, or at risk, patient.

GI values and particularly GL values still have a useful, even if secondary, role to play. This is because a low GI/GL diet will generally be a more healthy diet than the typical modern Western diet that is so significantly contributing to the current diabetes epidemic, and there is no suggestion that a low GI/GL diet might be harmful.

Indeed the recommendation of Diabetes Australia seems quite appropriate. This recommendation is:

> Diets of low energy density and containing a wide range of carbohydrate foods rich in dietary fibre and of low glycaemic index (cereals, vegetables, legumes and fruits) are recommended to reduce the risk of Type 2 diabetes[25].

Perhaps the most effectual role for GI/GL is therefore in the prevention, but not necessarily the treatment, of diabetes, and if ever it was true, then prevention is truly better than cure when it comes to this modern day, often largely self induced, scourge. That said, a diet overall lower in GI/GL will still likely represent a diet that is healthier than the typical Western diet.

Finally therapists need to be alert for further published research on this topic. It is possible that future better designed research will cause a re-think.

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# SUPPORT NETWORK SOUTH AUSTRALIA (SNSA)

## Message for South Australian Members from Sandra Sebelis

Following the idea I shared with our members at the South Australia Skills Update Seminar on 25 June 2006, the discussions that resulted and the report sent into our Sydney office for tabling at an ATMS Executive Management Committee meeting, I am happy to report that we 'have the green light'.

Could I now request that any SA members interested in providing their services/support to their colleagues, both on a regular basis as well as in possible times of crisis, to send their name, modality, clinic address, telephone number and email address to Raymond Khoury at the ATMS Sydney office for a list to be compiled. Raymond's email address is journal@atms.com.au and telephone number is (02) 9809 6800.

You are expected to request a 'token' fee for your service or, if you prefer, arrange a swap. It is at your discretion. I am happy to set the ball rolling and open my public Norwood yoga classes to our members for a gold coin; booking is unnecessary.

Thank you in advance to all who support this scheme.