# THE FOOD GUIDE PYRAMID: HAS IT BECOME OUR TOMBSTONE?

*For the past two decades, the message from nutrition opinion leaders has been clear: cut down on fat, eat more carbohydrate.*

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Dietary guidelines for optimal health suggest 50 - 55% of total energy from carbohydrates, 25 - 30% from fat, and 15 - 20% from protein as portrayed by the well-used Food Guide Pyramid.[1] The authors wish to re-examine these guidelines, by revisiting the role of high carbohydrate intake in the development of the insulin-resistant syndrome (also referred to as the metabolic syndrome).

The cluster of obesity, hypertension, varying degrees of glucose intolerance and dyslipidaemia with resultant heart disease, has collectively caused an enormous burden to health care cost in most Western societies. In 1988, Reaven refocused attention on this cluster and named it 'syndrome X'. As central obesity, omitted by Reaven from his original description, is a common component of the cluster, the term metabolic syndrome is now favoured. Reaven related the above-mentioned 4 components directly to high levels of insulin. It is estimated that this syndrome affects 70 - 80 million Americans (is this the tip of the iceberg?).[2,3]

Carbohydrate foods, consumed in isoglucidic amounts, have been shown to produce different glycaemic responses, depending on the nature of the food and the amount of processing involved. The concept of glycaemic index (GI) was proposed by Jenkins as early as 1981.[4] The GI can be defined as a quantitative assessment of carbohydrate-rich foods based on postprandial blood glucose response to an equivalent portion of a reference food, either white bread or glucose. In South Africa, glucose is the preferred standard. The ability of carbohydrates to raise blood glucose depends both on the type of constituent sugars (glucose, fructose, and galactose) and the physical form of the food (particle size and degree of hydration), and is affected largely by processing and refining. Although the GI of carbohydrates is influenced by the chemical composition of the starch, the processing of cereals and starches remains the largest manipulable influence on the GI. We have learned that not all 'complex carbohydrates' are equal.[5]

## ROLE OF INSULIN

Insulin has been called the feasting hormone because its release is enhanced by high blood glucose levels, and to a lesser degree by protein ingestion. Insulin is the major anabolic hormone that promotes the storage of nutrients, and at the same time, insulin acts to inhibit the mobilisation of these fuels.[6]

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It has been suggested that in about 25% of a normal population, insulin response to carbohydrates might be blunted – these are the people who fare well on high-carbohydrate diets, without developing hypoglycaemia or gaining weight. On the other hand, 25% of an otherwise normal population is genetically predisposed to an exaggerated insulin response to high carbohydrate intake. Typical of the insulin resistance syndrome, this group gains weight very easily, and struggles with weight loss management.

Between these two extremes is the other 50% of an otherwise normal population, who respond normally to high carbohydrate intake. They have an elevated insulin response to high carbohydrate intakes, not as exaggerated as the unlucky 25%, but sufficiently elevated to lead to the metabolic syndrome.

## WHERE DID WE GO WRONG?

It has become clear in the GI list of South African foods that these previous complex carbohydrates, e.g. popular breakfast cereals, instant noodles, rice cakes, regular white, brown and whole-wheat bread and rolls, are high GI foods, and therefore culprit foods that provoke large insulin responses.

Is it possible that by following prudent dietary guidelines for more than 2 decades, and encouraging carbohydrate intake in the form of cereals and breads (while food processing continued to refine these products), insulin surges were stimulated, resulting over time in the development of the insulin resistance syndrome? Did the much debated Dr Atkins (as early as 1972) not maybe have a point in stating: 'The total turnabout in man's diet, to the point where refined carbohydrates *dominate* it, has caused an evolutionary maladaption that shows itself not just in adults but even in the young. For carbohydrates – not fat – are the principal elements in food that fatten fat people'?[7]

## WHAT ABOUT THE FATS?

The eicosanoids are a group of hormone-like compounds, derived from the essential fatty acids linoleic acid (omega 6 series) and alpha-linolenic acid (omega 3 series). The balance between the activities of the eicosanoids is important to maintain normal vascular function.[8]

Linoleic acid supports thromboxane A2 (TXA2) and prostacyclin 2 (PCI2) and leukotriene B4 (LTB4) production, through arachidonic acid (AA) formation, all of which have potent inflammatory effects, and especially the vasoconstrictive and thrombotic activity of TXA2. The metabolic syndrome is also associated with a highly procoagulant state, endothelial dysfunction, and abnormalities suggesting a low-grade, inflammatory state characterised by elevated C-reactive protein and cytokine levels. Evidence exists that improved glycaemic control (with insulin or oral agents) improves the hypercoagulable state, and improves endothelial dysfunction.[9]

On the other hand, alpha-linolenic acid is converted to eicosapentanoic acid (EPA), and metabolised to eicosanoids of the 3-series, TXA3, PCI3, and LTB5. The thrombotic and inflammatory potency of these eicosanoids is much less than from the eicosanoids derived from AA.

Furthermore, omega-3 (EPA) and omega-6 (AA) fatty acids compete with each other for the enzymes involved in eicosanoid production, favouring EPA over AA.

Epidemiological studies have shown the relation between omega-3 fatty acid or fish intake, and reduced risk of CVD. EPA causes a reduction in AA and thus a reduction in the formation of TXA2. The precursor of AA, dihomo gamma linolenic acid (DGLA), leads to the formation of eicosanoids with opposite effects to those produced from AA.

The omega-3 fatty acids in plant oils are easily destroyed by cooking and heat and for this reason the best source of omega-3 fat is fatty fish (see below).

## PRINCIPLES OF DIETARY ADAPTATION

Bearing in mind that not all complex carbohydrates are created equal, the emphasis should be placed on low GI dietary intake.

Processes such as milling, beating, grinding, liquidising, mixing, mashing and refining foods raises the GI by making the starch more available to the body. Gastric emptying is slowed down by soluble fibre (oat bran, legumes, less processed oats, deciduous fruit), prolonging the digestion time, and lowering the GI. Digestive bran has no effect on the digestibility of the carbohydrate food in which it is found, so foods containing wheat bran will not have a lower GI than foods with no bran, unless bran is present in large quantities. Intact grains such as whole-wheat, whole corn, whole rye, whole oats and whole barley have much lower GI values than flours made from the same grains, because they take much longer to digest. The presence of both protein and fat slows down the rate of gastric emptying, and has a lowering effect on the GI of carbohydrate foods.

The principle is that the slower the rate of carbohydrate absorption (low GI),

the lower the rise in blood glucose, and the smaller the magnitude of the insulin response. Low GI foods are associated with earlier satiety compared with high GI foods, and studies have shown that in iso-energetic meals, slowly digested carbohydrate-rich foods may allow a sense of satiety to last longer than foods that are digested fast.

Fat restriction has been advocated for decades as a means of caloric restriction, to aid in weight loss. We should advocate a relatively higher intake of essential fats, with emphasis on omega-3 (EPA) such as high-fat cold water fish like salmon, sardines, mackerel, trout and eel. We should also advise a lower intake of saturated fats in animal products such as red meat, full cream dairy products, butter, cream and high-fat cheeses.

The practical and effective implementation of any of these principles is best facilitated through consultation with a qualified dietician.

Radical lifestyle changes to facilitate weight loss have been shown to be more effective than pharmacological agents in lowering insulin levels and preventing the onset of type 2 diabetes.[10] Therapy should be focused on weight loss and increased physical activity. Because insulin resistance often precedes the development of its consequences by years, if not decades, early detection and treatment encourages patients to develop good habits at a young age.

*References available on request.*

## IN A NUTSHELL

For over 2 decades, nutritional guidelines for optimal health have emphasised low-fat, high-carbohydrate intake.

During this time the incidence of obesity has increased, despite reduced fat intake.

The metabolic syndrome was identified in 1988, and linked with high circulating levels of insulin and insulin resistance.

In 1981 the concept of the glycaemic index showed that all complex carbohydrates are not equal and that processed carbohydrate products produce large glycaemic responses with high insulin demands.

It is possible that by encouraging increased carbohydrate intake, the incidence of metabolic syndrome was increased.

Through fat restriction, the intake of fish oil (EPA) has been low. EPA has been shown to be beneficial in reducing cardiovascular disease risk, which is high in patients suffering from the metabolic syndrome.

Nutritional guidelines for optimal health should be based on low GI, and allow for higher intakes of fish oil or omega-3 rich fats.

## SINGLE SUTURE

### STEROIDS, ASTHMA AND SMOKERS

It may be best for smokers with mild asthma to start immediately with high-dose steroid inhalers. A multicentre, randomised, double-blind trial of low-dose versus high-dose inhaled steroids taken for 12 weeks reports that, compared with non-smokers, smokers with mild asthma are insensitive to the therapeutic effects of low-dose steroids. Daily peak-flow readings were lower, and the number of exacerbations of asthma increased for smokers taking lose-dose inhalers.

*Tomlinson JEM, et al. Thorax 2005; 60: 282-287.*