Nutritional Management of Postprandial Glycaemia

Dr Wendy Russell

(on behalf of the ILSI Europe Expert Group)
Background to the Activity

Currently 366 million people have diabetes

Projected to reach 552 million in 2030

Leading cause of death in developed countries

Epidemic in newly industrialised nations

Poorly controlled diabetes:
- macrovascular disease
- vision loss
- renal failure
- neuropathy
- amputations
Background to the Activity

**Question 1.** Is post-meal hyperglycaemia harmful?

**Question 2.** Is the treatment of post-meal hyperglycaemia beneficial in improving clinical outcomes of glycaemic control?

**Question 3.** Which therapies are effective in controlling post-meal plasma glucose?

**Question 4.** What are the targets of post-meal glucose and how should they be addressed?
Background to the Activity

Advocated control of post-meal glucose by nutritional means

ILSI Metabolic Syndrome and Diabetes Task Force

“provide an in-depth understanding of the potential risk factors for the development of the metabolic syndrome and type 2 diabetes mellitus”

Expert Group: Nutritional Management of Postprandial Glycaemia

• develop nutritional strategies based on evaluation of the evidence
• interaction of macro-, micro- and non-nutrients within the food matrix
Diet and Health
Regulation of Glucose Homeostasis

Central Nervous System

Hepatic System

Muscle

Adipose Tissue

Pancreatic System

Gastrointestinal Tract
Pathophysiology

Insulin Resistance and β-Cell Dysfunction

- Increased Lipolysis
- Reduced Incretin Secretion
- Increased Glucagon Secretion
- Neurotransmitter Disfunction
Carbohydrate

Typically provide the major energy contribution to our diet

Primary determinants of the glycaemic response

Reduce the size/duration of this in people with diabetes

Consider:
- Simple Sugars
- Oligosaccharides
- Polysaccharides
  (Dietary Fibre)
## Glycaemic Index/Load/Glucose Equivalents

<table>
<thead>
<tr>
<th>Food Dose</th>
<th>Glycaemic Index (% basis)</th>
<th>Food Dose</th>
<th>Relevant Quantity</th>
</tr>
</thead>
<tbody>
<tr>
<td>50 g Carbohydrate</td>
<td>Glycaemic Response Relative to 50 g Glucose</td>
<td>X 1/100 Available Carbohydrate</td>
<td>Weight of Glucose = Response (g)</td>
</tr>
<tr>
<td></td>
<td>X 100</td>
<td>Glycaemic Index (% basis)</td>
<td>Glycaemic Load (per g basis)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Glycaemic Glucose Equivalents (per weight of food)</td>
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</table>
Relevance of Glycaemic Response

How meaningful are GI, GL and GGE?

Consider:

- Other Food Components/Food Matrix
- Amount/Frequency of Food Consumed
- Gastric Emptying/Digestive Response
- Previous Food Ingested
- Non-glucose Insulin Stimulation
- Rate of Absorption
- Inter-individual Variation
Carbohydrate: Total Carbohydrate/Weight Loss

Low-Fat High-Carbohydrate Diets


Low-Carbohydrate High-Protein, Low GI and Low GL Diets


High-Fat Mediterranean-Style Diet


Macronutrient Composition/Calorie Restriction?

Appropriate interventions required

- weight maintenance
- isocaloric
- long-term
Carbohydrate: Mono- and Disaccharides

**Prospective studies:**

- glucose and fructose have deleterious consequences
- no conclusive evidence for sucrose, maltose & lactose

**Intervention studies:**

- no short term deleterious effect of fructose
- increased postprandial glycaemia/insulinemia with sucrose


Studies with surrogate endpoints have shown detrimental effects of fructose on insulin resistance/sensitivity

Laville et al *Obs Rev* (2009)
Carbohydrate: Dietary Fibre

High Intake of Dietary Fibre = Reduced Incidence of T2DM

Acute glycaemic/insulinaemic response effected by:

• type and amount of dietary fibre

  Reduction in glucose/insulin response, dependent on T2DM status

• food matrix

  Soluble dietary fibre effect food matrix, digestion/absorption and in turn PPG
Studies suggest that soluble fibre improves acute glucose/insulin response

These include: beta glucan, guar gum, psyllium, glucomannan

- Pastors (1991)

Actual postprandial response may be masked by food matrix

Longer term interventions have produced conflicting results
Gut microbiota is recognised as a major contributor to human health

Preclinical studies suggest a role in regulation of glycaemia


Patients with diabetes have altered microbiota

Very few studies addressing the effect of prebiotics on PPG

- Consumption of short chain fructans decreased basal hepatic glucose
  Luo et al. (1996)

- Fructans reduced PP insulin response in hypercholesterolemic patients
  Giacco et al. (2004)

- Inulin-type fructans increased GLP-1 and decreased PP glucose
  Cani et al. (2009)
# Probiotics and Glycaemic Control

No direct clinical evidence that probiotic bacteria effect PPG

**Evidence of effect on related outcomes:**
- Fasting glycaemia
- Insulin resistance

<table>
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<tr>
<th><strong>Reduced fasting glycaemia in elderly T2DM with FOS rich dairy product</strong></th>
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<td><em>L. acidophilus, B. bifidum</em></td>
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<th><strong>Reduced fasting glucose (but not HbA1c) in T2DM</strong></th>
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<td><em>L. acidophilus (La-5), B. Lactis (Bb-12)</em></td>
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<th><strong>Improvement in insulin sensitivity in non-T2DM and T2DM</strong></th>
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Protein and Amino Acids

Amino acids stimulate insulin release

Leucine is particularly insulinotropic


Protein and/or amino acid ingestion strongly stimulates post-prandial insulin release in both healthy and T2DM subjects

Must consider protein content in addition to glycaemic load
Fatty Acids

- saturated fatty acids (SFA)
- trans-unsaturated fatty acids (TFA)
- monounsaturated fatty acids (MUFA)
- polyunsaturated fatty acids (PUFA)

High Intake of Fat = Reduced Insulin Sensitivity

Lower Intake of Fat = Composition Dependant
Saturated Fatty Acids

Direct association with T2DM  
Vessby et al Diabetologia (2001)

Mechanisms:

• impact on cell membrane composition/function
• intercellular enzyme activity and transcription
• exert damaging effects on β-cells
• ER stress
• inflammation
Trans Fatty Acids

Nurses’ Health Study associated TFA with T2DB

Iowa Study showed no association

Postprandial Insulin levels higher compared to MUFA

Chrisiansen et al *Diabetes Care* (1997)
Lefevre et al *Metabolism* (2005)
Monounsaturated Fatty Acids

No direct association with T2DB risk (Nurses’ Health Study)

Replacing SFA with MUFA improvement in insulin sensitivity

Potential Mechanism:

- incretin response/gastric emptying
- cytoprotection of β-cells
- improved β-cells function
- Increased glucose uptake (GLUT1/4)

Paniagua et al Diabetes Care (2007)
Polyunsaturated Fatty Acids

PUFAs (n-6; plant)
- improved insulin sensitivity
- reduced risk of T2DM

PUFAs (n-3; fish)
- inconstant results

Experimentally n-3 PUFA:
- anti-inflammatory activity
- increased leptin/adiponectin expression
- regulate genes involved in carbohydrate metabolism
Micronutrients: Vitamins

Strong evidence associating vitamin D with T2DM

- meta analysis – low vitamin D status and T2DM
- interventions - beneficial effects of vitamin D repletion

Mechanism:
- β-cell function
- insulin resistance
- inflammation

No data supporting benefits of vitamins on postprandial glycaemia
Micronutrients: Minerals

**Cr**
- Important role in glucose homeostasis
- Supplementation beneficial in T2DM
- Reduction in fasting and postprandial glucose

**Mg**
- Involved in glucose transport and metabolism
- Reduced Mg levels in T2DM
- Lack of data regarding postprandial glycaemia
- Sales et al (2011)

**Zn**
- Important role in insulin synthesis
- Plasma/Tissue concentrations low in T2DM
- Reduction in fasting and postprandial glucose
- Gunasekara et al (2011)
Non-nutrients: Phytochemicals

Most widely studied phytochemicals are phenylpropanoid-derived

- flavanoids
- phenolic acids

Human Intervention Studies (acute effect on PPG)

- chocolate
- grape seed, fig, mixed berries
- nuts (almonds, pistachio, mixed)
- tea, coffee
- cinnamon, ginseng, salacia
- seaweed, fraxinus

Non-nutrients: Phytochemicals

Action unknown - poor classification/metabolism/bioavailability

Potential Mechanisms:

- inhibition carbohydrate digestion
- modulation of glucose release
- stimulation of insulin secretion
- delayed gastric emptying
- inhibition of lipolysis
- decrease in oxidative stress
- inhibition of inflammation
Miscellaneous: Alcohol

J-shaped association with T2DM

Moderate consumption improved insulin sensitivity

Potential Mechanisms:

• acetate, reduces fatty acid release
• inhibits uptake of circulating fatty acid by muscle
• enhance glucose oxidation and insulin sensitivity

Addition of water to a test meal increased glucose response in:

- healthy individuals
- well controlled T2DM

No significant effect in poorly controlled T2DM

Torsdottir and Andersson *Diabetologia* (1989)
Miscellaneous: Sweeteners

**Aspartame**

- no increase in blood glucose (healthy; T2DM)
- no effect on postprandial glycaemia (T2DM)


- no effect in fasting or postprandial glycaemia
- no effect on HbA1c levels (T2DM)

  Nehrling et al *Diabetes Care* (1985)

- postprandial glycaemia, insulinemia and lipidemia significantly decreased compared to sucrose (obese)

  Raben et al *Food & Nutr Res* (2011)

**Stevia**

- postprandial glycaemia significantly lowered compared to sucrose (healthy; obese)

  Anton et al *Appetite* (2010)
Provisional Summary

Results are convincing for dietary fibre

Macronutrient balance is likely to be critical

Results are encouraging for a variety of phytochemicals

Gut microbiota appears to play an important role

Requirements:

• more well-planned studies are required
• better characterisation of potential bio-actives
• magnitude of effect between dietary groups should be addressed
• acute postprandial effects to be correlated with long-term outcomes
Expert Group

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