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# Impact of postprandial glycaemia on health: Role in body weight control and diabetes prevention[1]

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## Background:

- Postprandial glycaemia (PPG) & insulinaemia/lipidaemia are implicated in the aetiology of metabolic chronic diseases
- Obesity & related health complications ⟶ reduced quality of life, massive healthcare costs & premature death
- Simple cost-effective strategies for prevention and management are needed
- The ILSI Europe Dietary Carbohydrates Task Force Expert Group (EG) established the state-of-the-science regarding these relationships.

## Methods:

The EG aimed to investigate: 1. Relationship between PPG and body weight & the aetiology of diabetes; 2. Whether physiological effects found in impaired glucose metabolism (e.g. people with diabetes) also occur in 'healthy' individuals. A summary of the literature was prepared.

## Results: Study findings were summarised in tabular format and analysed by the EG. The Table below presents an example using selected studies that show an effect on *insulin sensitivity*:

| Studies | Design, duration | Subjects Age BMI | Intervention | Postprandial glucose and insulin profiles | Effect on body weight | Effect on fasting glucose and insulinemia | Effect on insulin sensitivity (method used) | Effect on insulin secretion (method used) | Effect on hormone / mechanism |
|---|---|---|---|---|---|---|---|---|---|
| **Studies with data on postprandial kinetics** | | | | | | | | | |
| Brynes 2003 | R CO 24d with 24d wash- out | 17 M,overweight with at least one cardiac risk factor Age 45 BMI = 29 | HGI/LGI/HF/ Sucrose | Large difference on glucose and insulin profile | Decrease in LGI | No | Increase in LGI and HF (However, for HF, increase of NEFA and TG) (by HOMA PP) | | |
| Maki 2007 | R P 12wk | 40 M, 27 W Age 60 BMI = 32 | Beta glucan vs. control | No difference at D0 | | No | Increase with BG | **Decrease in BG (Test meal)** | |
| Weickert 2006 | R 3d | 17 healthy/ overweight Age 53 BMI = 30 | Cereal fibers | No change | NA | No | 13% increase (by clamp) | | |
| Weickert 2005 | CO 7d | 17 healthy/ overweight Age 53 BMI = 30 | Cereal fibers | Decrease AUC glycemia and earlier insulin secretion | NA | No | | | No change in ghrelin Effect through colonic fermentation |
| **Studies without data on postprandial kinetics** | | | | | | | | | |
| Pereira 2002 | R, CO 6wk with 6-9wk wash-out | 11 hyperinsulinic Age 42 BMI = 30 | Whole grain | Not reported | No | No effect on fasting glycemia Decrease of fasting insulin with LGI | Slightly increase insulin sensitivity (by clamp) | | |
| Reiser 1979 | R, CO 6wk with 4 wk wash-out | 19 healthy subjects 10 M / 9 W Age 42 | 30% Energy intake= starch or sucrose | Not reported | | Increase fasting glycemia and insulinemia after sucrose | Decrease after sucrose (by OGTT) | | |
| Frost 1998 | R, P (3wk) | 28 W healthy Age 35 BMI = 23-25 | LGI /HGI | Not reported | | No | Increase after LGI (Adipocyte Insulin sensitivity) | | |
| Garcia 2007 | R CO 6wk with 6 wk wash out | 11 IGT (9 W) Age 56 BMI=30 | Arabinoxylan concentrate | Not reported | Minus 1.9 to 3.7 kg | No | decrease glucose and insulin AUC (test meal) | | No change in ghrelin Decrease in lipolysis, SCFA |
| Robertson 2005 | R, CO 4wk with 4wk wash out | 10 healthy (6W) Age 49 BMI = 23 | Resistant Starch 30g/D | Not reported | No | No | Increase in insulin sensitivity after clamp test, but no change after test meal | No change (HOMA B) | Increase in ghrelin |

BMI, Body Mass Index; CO, cross-over; d, day; HF, High Fat; HGI, High GI; IGT, Impaired Glucose Tolerance; LGI, Low GI; M, men; P, parallel; R, randomised; W, women; wk, week

## Conclusions:

### Body weight control

Studies suggest that the types of foods which elicit a lower postprandial glucose response may be useful as part of an overall strategy for combating obesity, but the evidence for a role of PPG *per se* in these effects is considerably weaker. Short-term studies with different GI products suggest a role in appetite regulation, but the link to PPG is unclear. Suggested underlying mechanisms involve hormonal and metabolic changes. There is currently little data on the chronic effects of reducing PPG on body weight.

### Type 2 diabetes mellitus

There is mechanistic evidence from animal and human studies that elevated blood glucose and an altered pattern of PPG, together with an elevated insulin concentration, lead to transitory deleterious metabolic and hormonal state in healthy subjects. These phenomena are exacerbated in impaired glucose tolerant subjects. Thus, there exists evidence that reducing blood glucose and insulin responses is beneficial to prevent type 2 diabetes genesis. The relationship between postprandial glucose, diabetes and CVD is a continuum, indicating that early intervention to normalize postprandial glucose (and related indicators of hyperglycaemia and hyperlipidaemia) may be required.

***It is evident that more randomised controlled dietary intervention trials inducing effective low-glucose response diets are necessary to be able to draw more definitive conclusions on the role of postprandial glycaemia in relation to health.***

### Reference:

1. Blaak EE et al. Impact of Postprandial Glycaemia on Health and Prevention of Disease. (2012) *Obesity Reviews.* 13(10):923-84. Available by open-access: http://www.ilsi.org/Europe/Documents/Postprandial%20Glycaemia.pdf.

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