Comparisin of Low Energy Breakfast Based on Special Egg White Spread Product With a Standard Breakfast

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Abstract

Aim: The aim of the study was to evaluate the influence of a new form of low calorie breakfast in comparison to standard low fat cheese breakfast. A special spread based on egg white was used (Energy content per 100 g: 319 kJ versus 802 kJ, fat content 1.15 g versus 15 g). Egg white derived products are widely used for weight reduction in Czech Republic.

Methods: 12 non-diabetic patients were included into the study (7 men and 5 women), mean age 38.5±10.7 years, and mean BMI 24.6 kg/m². Standard breakfast was served first in a time interval of 1 week (Krajanka cheese) and after a week wash-out period low energy breakfast based on egg white was served for the same time. Glucose level, insulinaemia and C peptide were evaluated in 0, 60, 120 minute after breakfast. Psychological questionnaire was used to evaluate the taste and satiety.

Results: Standard breakfast: insulinaemia 6.3…34.6…11.0 IU, C peptide 0.6…1.6…1.0 pmol/l, glucose 5.0…5.4…4.8 mmol/l Low energy breakfast: insulinaemia 6.6…24.6…10.7 IU, C peptide 0.6…1.4…1.0 pmol/l, glucose 4.8…5.1…5.0 mmol/l. Paired t-test was used for evaluation. No difference in blood glucose level was found, borderline significances of insulinaemia in 60 minute (p= 0.056) and in C peptide (p= 0.089). Significant difference in decrease of insulinaemia between 60 and 120 minute (p=0.03) and borderline significant decrease in C peptide (p=0.055) shows shorter insulin secretion after low calorie breakfast and good insulin sensitivity in this group of lean subjects. Higher satiety and less-worthier taste were found using low energy breakfast.

Conclusion: Low calorie breakfast induces lower and shorter insulin secretion and has a good metabolic effect in lean subjects. Testing in obese subjects will follow. Egg white derived low calorie products have a beneficial effect on insulin response without any difference in ingested carbohydrate quantity.

Introduction

Obesity (defined as body mass index >30 kg/m²) and its associated disorders (diabetes mellitus type 2 or impaired glucose tolerance, arterial hypertension, dyslipidaemia and elevated uric acid level – part of so called metabolic syndrome) are problems of utmost significance in current medicine [1]. The prevalence of obesity and complication of obesity tends to increase not only in developed countries with highest rates in USA and Europe, but also in a substantial number in developing countries. Obesity is one of the most significant risk factor decreasing mean age of patients [2, 3]. It was shown, that body weight reduction in obese patients not only leads to significant improvement of diabetes mellitus type 2 compensation but also of other parameters of the metabolic syndrome [4]. The first and most important step during reduction of total body weight is starting the reduction diet. The reduction of body weight by 10–15% significantly decreases the risk for diabetes mellitus type 2 development [5].

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Here we should stress, that most of obese patients diet themselves repeatedly during their lives but the results of these time-limited and nutritionally not very balanced diets are at better under optimal if any. It is frequently seen, that patients trying to reduce their body weight do hunger-strike or eat daily as little as 2000 KJ, which is the reason why after finishing diet effort there is often a so-called “yo-yo effect”.

Lowering diets can be classified from different points of view. We may divide them for example according to the fat or sugar content, the approach which is used mainly in obese diabetic patients. The most important criterion of diet classification is the energy value of diet. Before starting dieting, it is always advisable to know eating habits of the patient which is meant to trace both the amount of energy ingested before starting on the diet and the frequency of dishes. It generally advised to lower the energetic intake stepwise in steps of 2000–4000 KJ rather than unpremeditated start with sudden decrease to diet with energy content 4000 KJ. Stepwise lowering of energy intake leads not only to marked loss on weight but also to better compliance of patients as there is a lesser occurrence of starvation and binge eating. Another precaution in body weight loss is regularity in feeding. It is recommended to eat 3–5 times a day smaller helpings. If the patient does not start to eat regularly, the hope for long-time effect of weight reduction is minimal. The drawback of all reducing diets is that there is no single universal diet after that all patients would have similar weight loss. Different weight loss in different patients is caused by several factors – mainly life style reflected by different moving activities and dietary habits before starting diet. Another factor that influences the weight loss rate is a patient’s age and other comorbidities, mainly diabetes mellitus type 2. Generally we may expect faster and more marked loss on weight in younger patients than in seniors.

Selection of food suitable for lowering weight should prefer low fat content food – the upper limit of fat content should be utmost 30%. Frankfurters, butter, margarine and fat meat – especially pork – are ranked among improper food. Unfortunately the selection of low fat food is not as wide in the market. The very often used argument against this food is the price. Practically we proved as very good protein slices commercially available under trade name “Šmakoun” which are made from pure egg white or meat substitute widely known under the name “Robimaso” which is essentially a vegetable protein.

To our best effort we were not able to find any work that would focus on comparison of influence of standard breakfast and a low calorie breakfast based on egg white on postprandial serum levels of glucose, insulin and C peptide.

**Patients and Methods**

12 non-diabetic patients were included into the study (7 men and 5 women), mean age 38.5±10.7 years, and mean BMI 24.6 kg/m². Standard breakfast was served first for a time interval of 1 week (Krajanka cheese) and after a week wash-out period low energy breakfast based on egg white was served for the same time. Nutritional...
content of both products is presented in Table 1. Serum levels of insulin, glucose and C-peptide were evaluated in timecourse 0, 60 and 120 minutes after breakfast. Paired t-test was used for evaluation. Satiety and taste were evaluated during personal interviews. Serum concentrations of traced biomarkers were evaluated either by commercially available RIA kits (insulin by RIA kit, Immunotech, Czech Republic) while the rest by standard laboratory methods.

The results are reported as $\bar{x} \pm SD$. Paired t-test with adjustments was used for comparison of the groups as appropriate (AnalystSoft, StatPlus – statistical analysis program. Version 2007. See http://www.analystsoft.com).

**Results**

Results in healthy population (non-diabetics) show the different influence of normal and low-fat diet on insulin secretion. Although the differences in C-peptide (Figure 1) and insulin concentrations were during the timecourse statistically insignificant in times 0 and 30 minutes due to data dispersion ($p=0.056$ for insulin; $p=0.089$ for C-peptide) there is an obvious p-trend visible. There was no difference present in terms of baseline serum glucose concentrations which supports our statement on good insulin sensitivity in our patients (Figure 2).

Statistically significant difference in serum insulin (Figure 3) decrease followed by return towards basal line in both breakfasts in 60 and 120 minutes 23.1 (S) vs. 13.9 (P) ($p=0.03$) reflects good insulin sensitivity in this group of lean subjects.

**Table 1 – Nutritional content of tested products**

<table>
<thead>
<tr>
<th></th>
<th>Normal breakfast (cheese)</th>
<th>Low energy breakfast (spread)</th>
</tr>
</thead>
<tbody>
<tr>
<td>protein (g/100 g)</td>
<td>12.5</td>
<td>13.1</td>
</tr>
<tr>
<td>fat (g/100 g)</td>
<td>15</td>
<td>1.15</td>
</tr>
<tr>
<td>sugar (g/100 g)</td>
<td>2</td>
<td>3.16</td>
</tr>
<tr>
<td>energy (kJ)</td>
<td>802</td>
<td>319</td>
</tr>
<tr>
<td>Dry matter (g/100 g)</td>
<td>29</td>
<td>15.13</td>
</tr>
</tbody>
</table>

**Figure 1 – C-peptide serum levels during timecourse NB vs. S. *$p=0.003$ for S vs. NB at timecourse 60 min.**

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C-peptide concentrations revealed a P-trend between 60 and 120 minutes 0.64 vs. 0.40 (p=0.055). Higher satiety but less-satisfying taste was found using low energy breakfast.

Discussion

Obesity and mainly its complications defined altogether as metabolic syndrome (diabetes mellitus type 2 or impaired glucose tolerance, arterial hypertension, dyslipidaemia and elevated uric acid level) are considered a hot topic of contemporary medicine. It has been shown in dozens of clinical trials that in patients with fully developed metabolic syndrome, a significant decrease in expected mean age is found and no one can be surprised that this disease attracts nowadays much attention. The major problem in fully developed diabetic syndrome is the treatment of diabetes mellitus type 2.

Several factors contribute to the genesis of diabetes mellitus type 2 – insulin resistance and impaired glucose secretion and the dysfunction of beta cells. Beta
cell dysfunction reflects the decrease in number of beta cells, the impairment if their function by a long-term hypergycæmia (glucotoxicity) raising continuous hyperinsulinaemia and increased levels of fatty acids (lipotoxicity).

At the same time there is a visible depletion of insulin-containing granules in the cells which is reflected by an impaired response to glucose stimulation. In diabetes type 2 patients it is an insufficient early secretion phase responsible for blunting of postprandial glycemic changes which impaires vigorously the normal pulsatile insulin secretion [6]. Similar changes are visible – though not so profound – both in patients with impaired glucose tolerance where both qualitative and quantitative defects in insulin secretion are observed and in close relatives of diabetic patients where a lower insulin secretion in early phase after peroral glucose administration.

Much effort was put in recent time to elucidate problems connected with postprandial status both in diabetic and non-diabetic populations. It was shown that increased postprandial glycaemia is significant and independent prognostic factor contributing to cardiovascular diseases development. Postprandial hyperglycaemia acts together with hyperinsulinaemia on microvascular complications development. The increase in postprandial glycaemia by 1 mmol/l doubles the relative risk of death from cardiovascular diseases. Postprandial hyperglycaemia not only reflects the lack of insulin itself needed for processing of glucose ingested by food but also enhanced endogenous glucose production by liver gluconeogenesis.

In non-diabetic population roughly 50% of the total day dose of insulin is secreted equally during the whole day. The rest is secreted postprandially. As a response to food intake there is a fast release of insulin from storage granules of beta cells and this early phase of insulin secretion allows peripheral glucose utilization, decreases liver gluconeogenesis and limits postprandial increase of serum glucose levels. This early phase starts not later than 2 minutes after food intake and lasts further for approximately 10–15 minutes. It is followed by a late phase of secretion that lasts until normal glucose levels are achieved. The impaired early phase is a marker of beta cell dysfunction and appears a long time before a clear-cut change in serum glucose levels is observed. Postprandial hyperglycaemia might be influenced (lowered) in several ways – either medically (dipeptidylpeptidase IV inhibitors, exenatid or liraglutide [7, 8, 9]) or by a diet regimen.

The second proposed mechanism of decreasing a postprandial glycaemia – namely a diet – plays in this time a significant role as a cheap though efficacious approach. We showed in our work that after a low calorie protein breakfast a significant decrease in C-peptide and insulin secretion is present in comparison to standard breakfast where an opposite effect was seen. The results of our work if extrapolated on patients with impaired glucose tolerance or impaired insulin sensitivity show that low calorie breakfast might effectively lower glycaemia in this group and the only tiny contribution of patient would be a change a dietary habits.

In our comparative work, a protein mass slice “Šmakoun” was used for preparation of the spread. Slice was finely ground and resulting substance was
fortified by linseed oil, starch and soluble dietary fibre. Final flavour was improved by adding spices. Nutritional data of the consumed 50 g of the spread in test are as follows: proteins 6.25 g, fats 7.5 g, saccharides 1 g, total dry matter 14.75 g, energy 400 kJ. This slice has been validated in our body mass reduction program both in diabetic and non-diabetic patients due to its low calorie content and effect on postprandial insulinaemia [10, 11].

It is tempting to study the same approach in patients with impaired glucose tolerance, where a difference in glucose levels should be present in comparison to our group. The differences in satiety between both groups could simply reflect a significant difference in taste with less-worthier taste in “Šmakoun” slice. The continuous research on new low calorie foods is nowadays highly appreciable as a fight with widely accepted junk-food/fast-food.

Conclusion
Low calorie breakfast induces lower and shorter insulin secretion and has a good metabolic effect in lean subjects. Testing in obese subjects will follow. Egg white derived low calorie products have a beneficial effect on insulin response without any difference in ingested carbohydrate quantity.

References