POLISH JOURNAL OF FOOD AND NUTRITION SCIENCES
Pol. J. Food Nutr. Sci. 2006, Vol. 15/56, SI 2, pp. 59-61

# THE VALUE OF GLYCEMIC INDEX AND GLYCEMIC LOAD OF DIET IN THE GROUP OF PATIENTS WITH COLON ADENOMAS

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Key words: glycemic index, glycemic load, colonic adenoma, colon cancer

High dietary glycemic index (GI) may increase colorectal cancer risk by affecting insulin and insulin-like growth factor-I levels. Colon polyps are a pre-malignant condition. We examined carbohydrate intake, the value of GI and glycemic load (GL) in patients with colon polyps recognized as adenomas (n = 83) in comparison with patients with normal result of colonoscopy (n = 73). The mean intake of carbohydrates in study group was 256.5±36.5 g/d, in control group 305.9±39.1 g/d (NS). The mean value of GI in study group was 59.8±4.1, in control group – 60.4±5.2 (NS). The value of the mean GL was 128.4±8.6 and 130.2±9.1, respectively (NS).

## INTRODUCTION

The strong relationship between Westernization and colon cancer incidence has spawned a number of explanatory hypotheses, many focused on the influence of dietary calcium, fat, fiber and carbohydrates on the colonic carcinogenesis.

Although diet is believed to influence colorectal cancer risk, the long-term effects of a diet with a high glycemic index and glycemic load are unclear. There is some evidence that glycemic effects of diets high in refined starch may increase colorectal cancer risk by affecting insulin and insulin-like growth factor-I levels. However, the results of epidemiological and clinical studies are conflicting.

Colon polyps are a pre-malignant condition of colon cancer. The aim of the study was to compare carbohydrate intake, the value of dietary glycemic index and glycemic load in patients with colon adenomas and in control group without adenomas.

## MATERIALS AND METHODS

We examined carbohydrate intake, the value of dietary glycemic index (GI) and glycemic load (GL) in the group of patients with colon polyps recognized as adenomas (study group, n=83) in comparison with the group of patients with the absence of any pathological changes in colonoscopy (control group, n=73).

The participants from both groups comprised patients, in whom colonoscopy was performed because of different suspicions. The exclusion criteria involved another than polyps changes, like colon cancer or inflammatory bowel diseases.

The mean age of the study group was 56.4 years and control group – 56.3 years. The M/F proportions in both groups was similar (1/1.20 and 1/1.23).

Dietary data were assessed by using dietary history method. In both groups dietary history was obtained with an inquiry form including data on usual consumption of dishes and drinks and their amounts. The intake of nutrients in everyday diet was established by interviewing the patients using an album of food products [Szczygłowa *et al.,* 1982].

These data were used to estimate the carbohydrates value of habitual daily diet. The calculation were obtained using tables of composition and nutrients value of products and dishes [Kunachowicz *et al.,* 1998; Nadolna *et al.,* 1994; Piekarska & Łoś-Kuczera, 1983].

For the purpose of the present analysis the GI values were taken from International table of glycemic index and glycemic load values [Foster-Powell *et al.,* 2002]. GIs of foods (especially for Polish products and dishes) for which no published data were available were arithmetically estimated [Buyken *et al.,* 2001]. Glucose was the standard food on which all GI's were based.

The GI value was calculated by using the method described for mixed meals, *i.e.*, the value of each food was multiplied by its carbohydrate content expressed as a proportion of the total carbohydrate for a day [Wolever *et al.,* 1991]. The GL value of daily diet was calculated by summing individual GL of food (GL = GI x carbohydrate content in portion of food / 100).

In statistical analysis ANOVA and Kruskal-Wallis tests were used.

## RESULTS AND DISCUSSION

The mean intake of carbohydrates in study group was

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256.5±36.5 g/d, in the control group 305.9±39.1 g/d. The difference was not statistically significant. The mean value of glycemic index in study group was 59.8±4.1, in control group – 60.4±5.2 (difference not statistically significant). The value of the mean glycemic load was 128.4±8.6 and 130.2±9.1, respectively (not statistically significant). The differences in the values of carbohydrate consumption, glycemic index and glycemic load values in the groups of male and female patients taken separately were also not statistically significant, which is presented in Table 1. An increasing body of evidence indicates that variations in the levels of insulin and insulin-like growth factors (IGF) could account for many risk factors of colon cancer.

Insulin and the IGF axis each play important and complementary roles in metabolism and growth. Insulin influences metabolism on a short-term basis (*e.g.*, after a meal), whereas the IGF axis exerts a longer-term effect on growth. IGF-1 inhibits apoptosis and is required for cell cycle progression.

Insulin and the IGF axis mediate many of the physiologic consequences of nutritional status. As major determinants of proliferation and apoptosis, there is a strong rationale to suspect that these factors influence carcinogenesis. In animal models, modulation of insulin and IGF-1 levels influences colonic carcinogenesis. Human studies consistently show that high levels of insulin and IGF-1 increase risk of colon cancer. People with type 2 diabetes, who have high levels of insulin and IGF-1, are at elevated risk of colon cancer in most studies. Many studies indicate that high intake of sucrose, starches and diets with a high glycemic index that stimulate insulin secretion are associated with a higher risk of colon cancer. These patterns may be particularly deleterious in those with a sedentary lifestyle.

Several clinical studies were performed to asses the role of dietary GI or GL in the pathogenesis of colon carcinoma or colon polyps.

Slattery *et al*. [1997] evaluated mean GI of diet in American population using food frequency questionnaire. In the group with highest quintile of GI and sedentary life-style, higher risk of colon cancer was found (the relative risk for highest quintile in comparison to the lowest quintile was 3.46 among men and 2.00 among women). But it must be admitted that in the group of physically active people GI value was not related to the risk of cancer. High mean glycemic load of diet was connected with increasing the risk of 69% in females and 87% in males. The relationship between sugar consumption and colon cancer was much lower.

In the study performed by Franceschi *et al*. [2001], risk of colon cancer was 70% higher in the group of people consuming diet with high GI value in comparison to the group with low GI (highest and lowest quintile was compared). Levi *et al*. [2002] studied mean GI value in over 300 patients with colon cancer in comparison to the paired control group. For people with high and moderate tercile of GI value higher risk was found that in the group with the lowest GI value. Relative risk was 2.2 and 1.8, respectively.

Higgingbotham *et al*. [2004] have chosen 174 women with colon cancer diagnosed with 8 years of observation from the group of 40 thousands women participated in Women's Health Study. It appeared that the group in highest quintile of mean glycemic load value had relative risk 2.85 higher that the group with lowest glycemic load value. Terry *et al*. [2003] published the results of prospective study involving nearly 50 thousands of women in the age of 40-59 years in Canada. The mean observation period was 16 years. In this time about 600 cases of colon cancer were diagnosed. The risk of cancer was evaluated in relation to total carbohydrate consumption, sugar intake and mean glycemic load of the diet. The comparison was performed between groups with the highest and lowest quintile of the results. In none of the evaluated factors any statistically significant differences were seen (the relative risk difference was not higher than 5%). The results of this study not confirm the role of GI idea in the etiology of colon cancer [Terry *et al.,* 2003]. Up to now, only one study was devoted to the role of glycemic load in pathogenesis of colon polyps. In the group of 34 thousands of women who were initially free of cancer or polyps 1715 cases of distal colon polyps were diagnosed during 18 years of follow-up. Dietary GI, GL, and carbohydrate intake were not related to risk of colorectal adenoma after adjustment for age and established risk factors [Oh *et al.,* 2004].

Our findings are in agreement with the results of the two last-mentioned studies.

## CONCLUSIONS

The value of GI and GL in the group of patients with and without colonic adenomas was similar. Our data do not support the hypothesis that diet high in carbohydrates with high glycemic index and glycemic load increases the risk of colon adenomas.

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TABLE 1. Mean value of carbohydrates intake, glycemic index (GI) and glycemic load (GL) values in men and women population from the studied groups.

| | Men | | Women | |
|---|---|---|---|---|
| | Study group (n = 34) Mean value ± SD | Control group (n = 27) Mean value ± SD | Study group (n = 49) Mean value ± SD | Control group (n = 43) Mean value ± SD |
| Carbohydrates intake (g) | 282.7 ± 40.8 | 336.4 ± 37.0 | 234.5 ± 35.8 | 274.2 ± 36.9 |
| GI value | 59.5 ± 4.7 | 61.7 ± 5.3 | 59.5 ± 4.7 | 61.7 ± 5.3 |
| GL value | 132.4 ± 8.8 | 133.9 ± 9.2 | 125.7 ± 8.6 | 128.1 ± 9.0 |

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## WARTOŚĆ INDEKSU GLIKEMICZNEGO I ŁADUNKU GLIKEMICZNEGO DIETY U CHORYCH Z GRUCZOLAKAMI JELITA GRUBEGO

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Istnieją przesłanki na znaczenie stosowania diety o wysokim indeksie glikemicznymi (IG) i ładunku glikemicznym (ŁG) na wzrost ryzyka raka jelita poprzez wpływ na wytwarzania insulinopodobnego czynnika wzrostu I. Celem badania była ocena spożycia węglowodanów, średniej wartości IG oraz ŁG diety w grupie chorych z gruczolakami jelita grubego (n=83). W grupie kontrolnej (n=73) nie stwierdzono żadnych nieprawidłowości w kolonoskopii. Średnie dzienne spożycie węglowodanów w grupie badanej wyniosło 256.5±36.5 g a w grupie kontrolnej 305.9±39.1 g. Średnia wartość IG w grupie badanej wynosiła 59.8±4.1, zaś w grupie kontrolnej 60.4±5.2. Średni ŁG diety w grupie badanej osiągnął wartość 128.4±8.6 a w grupie kontrolnej 130.2±9.1. W żadnym przypadku różnice nie były istotne statystycznie.