Coll. Antropol. **35** (2011) 4: 1363–1368
Review

Glycemic Index in Diabetes

Dario Rahelić[1], Alexandra Jenkins[2], Velimir Božikov[1], Eva Pavić[3], Klara Jurić[1], Christopher Fairgrieve[2], Dominik Romić[1], Slaven Kokić[4] and Vladimir Vuksan[2]

[1] University of Zagreb, Dubrava University Hospital, Division of Endocrinology, Diabetes and Metabolic Disease, Zagreb, Croatia
[2] University of Toronto, School of Medicine, St. Michael's Hospital,Risk Factor Modification Centre, Toronto, Canada
[3] University of Zagreb, Dubrava University Hospital, Departament of Nutrition, Zagreb, Croatia
[4] University of Split, Split University Hospital Centre, Division of Endocrinology, Diabetes and Metabolic Disease, Split, Croatia

A B S T R A C T

The Glycemic Index (GI) is a rating system that ranks carbohydrate-containing foods according to their postprandial blood glucose response relative to the same quantity of available carbohydrate of a standard such as white bread or glucose. The concept of GI was first introduced in the early 80's by Jenkins and coworkers. Since then, numerous trials have been undertaken, many indicating benefits of a low GI diet on glycemic control, as well as lipid profiles, insulin and C-peptide levels, inflamatory and thrombolytic factors, endothelial function and regulation of body weight. As a result, a low-GI diet may prevent or delay the vascular complications of diabetes. However, despite many studies supporting the benefits of the Glycemic Index as part of the treatment of diabetes mellitus, several areas of controversy have been raised in the literature and are addressed here. Clinicians treating diabetic patients should be aware of the potential benefits of low-GI foods in the prevention and treatment of diabetes and its complications.

Key words: *Glycemic Index, Diabetes, Low-GI diet, High-GI diet*

Introduction

The dramatic increase in the incidence of type 2 diabetes represents one of the most significant global health issues of the twenty-first century, and has prompted revisions in the prevention and treatment of this devastating disease[1].

Recommendations for carbohydrate levels in the diet have see-sawed between the historically high-fat, low--carbohydrate diet[2] to a high carbohydrate, low fat[3], in an effort to reduce the incidence of cardiovascular disease. Recommended levels of carbohydrate intake now seem to have settled in between 45 and 60% of energy depending on individual preference and not unlike the diet reccommended for the general population[4].

Carbohydrate Counting (or Carbohydrate Exchanges) is a concept that was introduced in the 1950's in order to achieve consistent carbohydrate intakes leading presumably to predictable post prandial blood glucose excursions[5]. In addition the assumption was made that complex carbohydrates cause a smaller rise in blood glucose concentration than simple, rapidly-absorbable carbohydrates. However, this concept suggests that all complex carbohydrates in equal portions produce the same effect on blood glucose concentration. At the same time, it also assumes that all simple carbohydrates in equal portions produce the same effect on blood glucose concentration[6]. In 1981 this concept was challenged with the introduction of the Glycemic Index (GI)[7] which demonstatred that, despite equicarbohydrate amounts, foods may result in very different postprandial blood glucose repsonses. The GI allow the ranking of carbohydrate-containing foods based on the glycemic response they illicit when consumed. The GI of a given food is determined by the blood glucose response relative to a standard food such as white bread or pure glucose[7]. As in a standard oral glucose tolerance test, blood glucose is measured at regular intervals over a two-hour period following consumption. The glycemic index is calculated as the area under the glucose curve of the test food, divided by the area under the glucose curve of the control food, and expressed as a percent[7,8]. The generally accepted convention is that foods with a GI of 55 or less are considered to be »low-GI«, foods with a GI between 56 and 69 are con-

Received for publication November 5, 2010

sidered »medium-GI«, and foods with a GI above 70 are considered »high-GI«. Pure glucose control would have a GI value of 100, while white bread control is 71 of its value. Clearly, foods with a lower GI value would be better choices for patients trying to lower their blood glucose levels.

Mechanisms of High-GI and Low-GI Diets

The metabolic effects of low and high GI diets are hypothesies to be related to the rate of glucose absorption from the small intestine[9]. The rapid rate of glucose absorption after consumption of a high-GI meal causes a spike in blood glucose concentration. This transient hyperglycemia stimulates the rapid release of insulin from pancreatic beta cells and simultaneously inhibit secretion of glucagon from pancreatic alpha cells. The rise in insulin secretion facilitates the uptake of glucose by the liver, muscle, adipose and other insulin-dependent tissues[10], thus quickly lowering blood glucose levels. However, two to four hours after a high-GI meal the high insulin and low glucagon levels result in blood glucose levels falling below starting levels often into the hypoglycemic range[8]. In turn, this stimulates a counter-regulatory hormone response to achieve normal glycemic levels and increases glycogenolysis and gluconeogenesis. As a result, high-GI carbohydrates increase the concentration of free fatty acids[8], which causes insulin resistance and impaired glucose tolerance[11] in subsequent meals[12]. Furthemore, the hypoglycemia following the consumption of high-GI foods may induce feelings of hunger and may even preferentially stimulate the consumption of more high-GI foods, thus perpetuating the vicious cycle[8]. In contrast, hypoglycemia does not occur during the postprandial period after consumption of low-GI carbohydrates due to a slower and more gradual absorption of glucose from the gastrointestinal tract. Consequently, there is less stimulus for insulin release, lower levels of free fatty acid and an increase in insulin sensitivity[9]. The effects of ingesting a low GI meal may therefore impact the metabolic response to the next meal[13]. The mechanisms for this »second-meal effect« are likely mediated by slower rates of absorption and digestion, which result in a delayed period of fasting between meals. The ingestion of a low-GI food at bedtime has been shown to suppress both nocturnal free fatty acid levels and postprandial glucose levels at breakfast, possibly due to reduced nocturnal lipolysis[14]. A low-GI food taken in the evening can also prevent nocturnal hypoglycemia in patients with insulin-dependent diabetes mellitus[15].

More recently, reduced oxidative stress has been proposed as an additional mechanism by which low-GI foods attenuate insulin sensitivity and blood glucose levels[16]. Oxidative stress is defined as a disturbance in the balance between the production of free oxygen radicals and antioxidant capacity. Diabetes *per se* is associated with increased oxidative stress[17] which appears to have a major role in the micro- and macro-angiopathic complications of diabetes[18]. It has been demonstrated that low-GI foods can decrease oxidative stress by increasing the total antioxidant capacity[16] as well as reducing the concentration of lipid peroxidation markers[19].

The mechanisms of how the Glycemic Index affects glucose absorption and metabolism are are thus beginning to be understood, but ongoing investigation is needed.

Controversies and the Glycemic Index

Some investigators have criticized the usefulness of the Glycemic Index in the context of a mixed meal. It has been argued that the GI of each component of a meal cannot be used to predict the glycemic response to a mixed meal[20]. Indeed, there are discrepancies within the literature about determining the GI of a composite meal[20]; these have since been attributed, however, to methodological differences in calculating the area under the glycemic response curve (e.g. whether or not the area under the baseline is included in the calcualtion of the incremental area), blood sampling (arterial vs. venous blood) and the duration of time between the meal and the last glycemic measurement. When using a consistent methodology, it has been shown that the GI of a mixed meal can be accurately predicted by calculating the mean GI value of each component divided by its carbohydrate content[21].

In addition to the methodology involved in determining the composite GI of a meal, it has been well-established that many factors (including gastrointestinal motility, cooking methods, and the presence of other nutrients) can also influence the postprandial glucose response[9,22]. Extrinsic factors, such as the methods of treating, storing and cooking carbohydrate-containing foods, can affect the particle size and the integrity of the starch granules[23] and plant cell walls[24], making the carbohydrate portion more accessible to digestive enzymes[25] which would effectively raise the GI value of the food.

Furthermore, the presence of other macronutrients can influence the glycemic response. The potential effects of protein and fat on the glycemic response to a given carbohydrate food may be explained by increased insulin secretion[26] and a delayed effect on gastric emptying[27], respectively. Proteins typically induce a greater degree of insulin secretion from pancreatic beta cells compared with carbohydrates, despite an unchanged or even lower blood glucose concentration[28]. Dietary fats reduce the rate of gastric emptying, which consequently slows down the absorption of carbohydrates[28]. As a result, many investigators have expressed concern about the influence of dietary fats and proteins, as well as interactions with other macronutrients such as fiber, on glycemic response[28].

These concerns are supported by at least one study, which showed that the GI of mixed meals calculated from table values did not predict the measured GI. The authors found that the GI of mixed meals was more strongly correlated with either fat, protein or energy content than with carbohydrate content alone[29]. However, in

studies in which 8–24 g fat was added to mixed meals containing 38–104 g carbohydrate, the added fat had little effect on predicted glycemic response[30]. This discrepancy may again be the result of methodological differences, but more study is needed in this area.

There are also concerns about the apparent variability in the GI for some foods[31], and it has been suggested that differences in GI values of similar foods reported by different investigators could be due to real differences in starch structure or digestibility, variation in methodology, or the effects of random variation. In a more recent, multicenter study involving 7 different centers from around the world, in which the GI values of four centrally-provided foods (instant potatoes, rice, spaghetti and barley) were measured, the GI values of the foods did not vary significantly among the different sites[32]. Demonstrating that consistent results are achieved when the foods are the same. On the other hand, addition of some whole grains, like *Salvia hyspanica L.*, can decrease postprandial glycemia of tested food[33].

The Glycemic Index is therefore a source of several areas of controversy, and is not without its critics. Some investigators contend that the GI is highly variable, not physiological, and difficult to learn and follow[28,34]. On the other hand, Jennie Brand Miller's study in type 1 children demonstrating that low GI diets were easy to follow and effective[35].

Despite the critisisms and controversies which will probably continue for some time to come, the GI concept has been accepted by many diabetes associations around the world [26–38] as being useful, and an integral part of the dietary treatment of diabetes.

Glycemic Index and Type 2 Diabetes Mellitus

The results of some epidemiological studies suggest that long-term consumption of low-GI carbohydrates could reduce the risk of type 2 diabetes[39], while other prospective studies showed that a high-GI diet can increase the risk of type 2 diabetes by 37%[40]. However, some other studies have shown that Glycemic Index was also not significantly associated with the incidence of diabetes[41].

There are numerous mechanisms of glucose metabolism that may explain the possible link between high-GI diets and the increased risk of diabetes. High-GI diets may promote weight gain[42], which can lead to insulin resistance. In addition, high-GI diets can stimulate insulin secretion, which can contribute to pancreatic β-cell dysfunction[43] and the down-regulation of insulin receptors and so further increase in insulin resistance[44]. The Insulin Resistance Atherosclerosis Study was the first study to compare the effect of high-GI and low-GI diets on insulin sensitivity. However, no relation between GI and insulin sensitivity was found[45,46]. A large observational study also failed to find an association between GI and insulin resistance[47].

Glycemic Index and Glycemic Control

Studies on GI have focused mainly on its ability to improve glycemic profiles. Mechanisms by which the GI affect fasting plasma glucose levels are still largely unknown; however, some theories have been hypothesized as previously described above. Fasting and postprandial blood glucose, as well as glycated hemoglobin, are considered the most important parameters of glycemic control, and the role of the Glycemic Index will be discussed with respect to each.

Glycemic Index and Fasting Blood Glucose

Several randomized controlled trials have demonstrated the strong correlation between GI and fasting blood glucose in patients with diabetes, including one study in which blood glucose increased from 9.4 to 9.8 mmol/L among diabetic patients eating a high-GI diet, and decreased from 10.1 to 9.2 mmol/L among those eating a low-GI diet during the study period of four weeks[48]. Another study showed a 30% reduction in fasting blood glucose with a low-GI diet compared to 8% for the high-GI diet after two weeks[23]. This reduction has been shown to be even greater when a low-GI diet was consumed by diabetic patients for twelve weeks in combination with dietary education about GI[6]. Similar results were found by David Jenkins and his group[49].

There is thus a large amount of data in support of the ability of a low-GI diet to significantly reduce fasting blood glucose. Potential areas of criticism for these studies include their short-term nature and lack of possible mechanisms of action.

Glycemic Index and Postprandial Blood Glucose

Results of numerous studies have confirmed the beneficial effect of a low-GI diet on postprandial glycemia[23,48,50,51], and there is consensus that a low-GI diet can reduce both blood glucose and insulin by 30% compared to a high-GI diet[48]. A study by Wolever and collegues found that a significant reduction in postprandial blood glucose was sustained after one year[52]. Other studies, however, have failed to find a difference in postprandial blood glucose[20], while some investigators have suggested that much better improvements in glucose and lipid metabolism can be achieved by modest weight reduction in patients with type 2 diabetes[53].

Several studies have demonstrated decreased microvascular complications with improved glycemic control[54,55]. Measures which improve glycemic control, including low GI diets, may therefore be helpful in the prevention of complications associated with diabetes. Conversely, numerous studies have demonstrated that acute glucose peaks sucha as those associated with high-GI foods may contribute to the development of diabetic complications[56,57]. The oxidative stress caused by acute postprandial hyperglycemia also contributes to macrovascular damage

through oxidation of low-density lipoprotein (LDL), endothelial dysfunction and other pro-atherogenic mechanisms[57]. It is thus apparent that postprandial hyperglycemia is an important risk factor for cardiovascular morbidity and mortality[58,59].

Glycemic Index and HbA1c

Numerous clinical trials have investigated the relationship between the Glycemic Index and glycated hemoglobin (HbA1c) in patients with type 1 and type 2 diabetes. Low-GI diets have been shown to reduce the level of HbA1c by absolute amounts varying from 3%[60] to 19%[25,49,61] in clinical trials, which have been supported by cross-sectional trials as well[62]. This improvement in HbA1c may be due to an incremental reduction of glycemic responses as a result of consuming a low-GI diet, which has been shown to significantly reduce blood glucose and fructosamine (a related marker of glycemic control) after only two weeks, relative to a high-GI diet[63]. A null effect of a low-GI diet on HbA1c, however, has been reported in some longer-term studies of three months[64] and six months[50]. Similar conclusions were made in the Canadian Trial of Carbohydrates in Diabetes in which patients with type 2 diabetes were treated with a low-GI diet alone[52], although this may have been the result of the very low starting HbA1c levels of 6.1%, this study did demonstrate long-term reductions in postprandial blood glucose and C-reactive protein (CRP). Another study which followed 102 subjects with type 2 diabetes for 6 months demonstrated that reductions in A1c levels were maintained[49]. Despite different results there is nevertheless compelling evidence for the use of a low-GI diet in the dietary management of diabetes, and recommendations for its use have been made by numerous diabetes associations.

Glycemic Index and the Risk of Diabetic Complications

The increased risk of microvascular and macrovascular complications in patients with diabetes is well established[54,55]. Many trials have suggested that a low-GI diet can improve glycemic control[65]. Better glycemic control, futhermore, have influence on prevention or postponing the development of diabetic complications.

In a meta-analysis of 37 prospective observational studies, a high-GI diet was shown to be an independent risk factor for coronary heart disease (CHD)[39]. Possible beneficial effects of a low-GI diet in the prevention of CHD could be explained by improvements in blood glucose and insulin levels[25]. This is significant in that postprandial blood glucose appears to be a strong predictor of cardiovascular disease[58,66,67], and hyperinsulinemia is a well-known independent risk factor for CHD[68].

In addition, a low-GI diet has been shown to improve the lipid profile[63,69]. In a meta-analysis of 11 studies, lowering the composite dietary GI by at least 12 points was shown to reduce triglycerides by an average of 9%[70], while another study found that a high-carbohydrate, low-GI diet increased the level of high-density lipoprotein by 5.4% relative to an isocaloric high-GI diet[71]. Possible mechanisms for the influence of a low-GI diet on lipid profile could be a reduction in the amount of insulin-stimulating HMG-CoA reductase activity, impairment of cholesterol reabsorption from the ileum as a result of high fibre content in low-GI foods, or inhibition of cholesterol synthesis in the liver by the short-chain fatty acid propionate, a product of colonic fermentation[25].

A low-GI diet may also have affect other cardiovascular markers, including CRP, thrombolytic factors and endothelial function, which could further reduce the risk for diabetic complications. Several studies have found a positive correlation between GI and CRP[52,72] as well as plasminogen activator inhibitor 1 (PAI-1), suggesting that a low-GI diet may reduce the level of low-grade inflammation and coagulation, respectively. There is also evidence that hyperglycemia and hyperinsulinemia can lead to impaired fibrinolysis and thrombosis as well, which would further increase the risk of CHD[73]. A high-GI diet has also been shown to affect endothelial function through the increased production of oxygen free radicals, as a result of hyperglycemia[74], as well as a reduction in flow-mediated dilation (FMD)[75].

In summary, there is support in the literature for the mechanisms by which a low-GI diet may prevent or delay the development of diabetes and its complications.

Recommendations

In the Nutrition Recommendations and Interventions for Diabetes 2008, a position statement by the American Diabetes Association, it is suggested that low-GI foods that are rich in fiber and other important nutrients should to be encouraged in the prevention and nutritional therapy of diabetes[36]. The Joint Food and Agriculture Organization/World Health Organization Expert Consultation on Carbohydrates, the European Association for the Study of Diabetes, Canadian Diabetes Association, Diabetes UK and Diabetes Australia also encourage the use of the Glycemic Index in the prevention and treatment of diabetes[36–38,51,76].

Conclusions

Despite the controversy within the literature, there is substantial evidence that a low-GI diet can improve glycemic control in patients with diabetes. Further investigation is needed, however, to continue to support the use of the Glycemic Index in the prevention and treatment of diabetes and its complications health care professionals treating patients with diabetes should be aware of the beneficial effects of a low-GI diet.

REFERENCES

1. DEVILLE-ALMOND J, TAHRANI AA, GRANT J, GRAY M, THOMAS GN, TAHERI S, Am J Mens Health, 5 (2011) 30. — 2. ARKY RA, Nutritional management of the diabetic. In: ELLENBERG M, RIFKIN H (Eds) Diabetes mellitus: theory and practice (Medical Examination Publishing Company, New York,1983). — 3. NUTTALL FQ, BRUNZELL DJ, Diabetes, 28 (1979) 1027. — 4. AMERICAN DIABETES ASSOCIATION, Diabetes Care, 29 (Suppl 1) (2006) S4. — 5. MITCHELL, RD, NOWAKOWSKA JA, HURST AJ, J Hum Nutr Dietetics, 3 (1990) 19. — 6. FROST G, WILDING J, BEECHAM J, Diabetic Medicine, 11 (1994) 397. — 7. JENKINS DJA, WOLEVER TMS, TAYLOR RH, BARKER H, FIELDEN H, BALDWIN JM, Am J Clin Nutr, 34 (1981) 362. — 8. LUDWIG DS, JAMA, 287 (18) (2002) 2414. — 9. JENKINS DJ, KENDALL CW, AUGUSTIN LS, VUKSAN V, Am J Med, 113 (Suppl 9B) (2002) S30. — 10. MEYER C, WOERLE HJ, DOSTOU JM, WELLE SL, GERICH JE, Am J Physiol Endocrinol Metab, 287 (2004) E1049. — 11. PIATTI PM, MONTI LD, PACCHIONI M, PONTIROLI AE, POZZA G, Metabolism, 40 (1991) 926. — 12. WOLEVER TM, JENKINS DJ, OCANA AM, RAO VA, COLLIER GR, Am J Clin Nutr, 48 (1988) 1041. — 13. JENKINS DJA, OCANA A, JENKINS AL, WOLEVER TM, VUKSAN V, KATZMAN L, Am J Clin Nutr, 55 (1992) 461. — 14. AXELSEN M, WESSLAU C, LONNROTH P, ARVIDSSON LENNER R, SMITH U, Intern Med, 245 (1999) 229. — 15. AXELSEN M, ARVIDSSON LENNER R, LONNROTH P, SMITH U, Eur J Clin Nutr, 53 (1999) 706. — 16. BOTERO D, EBBELING CB, BLUMBERG JB, RIBAYA-MERCADO JD, CREAGER MB, SWAIN JF, Obesity, 19 (9) (2009) 1664. — 17. COMINACINI L, GARBIN U, PASTORINO AM, FRATTA PASINI A, CAMPAGNOLA M, DE SANTIS A, Diabetes Res, 26 (1994) 173. — 18. BAYNES JW, Diabetes, 40 (1991) 405. — 19. HU Y, BLOCK G, NORKUS EP, MORROW JD, DIETRICH M, HUDES M, Am J Clin Nutr, 84 (1) (2006) 70. — 20. COULSTON AM, HOLLENBECK CB, SWISLOCKI AL, REAVEN GM, Diabetes Care, 10 (1987) 395. — 21. TRUSWELL AS, Eur J Clin Nutr, 46 (Suppl. 2) (1992) S91. — 22. KREZOWSKI PA, NUTTALL FQ, GANNON MC, BARTOSH NH, Am J Clin Nutr, 44 (1986) 847. — 23. JENKINS DJA, WOLEVER TM, BUCKLEY G, LAM KY, GIUDICI S, KALMUSKY J, Am J Clin Nutr, 48 (1988) 248. — 24. ELLIS PR, DAWOUD FM, MORRIS ER, Br J Nutr, 66 (1991) 363. — 25. AUGUSTIN LS, FRANCESCHI S, JENKINS DJA, KENDALL CWC, LA VECCHIA C, Eur J Clin Nutr, 56 (2002) 1049. — 26. GANNON MC, NUTTALL FQ, NEIL BJ, WESTPHAL BA, Metabolism, 37 (1988) 1081. — 27. WELCH IM, BRUCE C, HILL SE, READ NW, Clin Sci, 72 (1987) 209. — 28. PI-SUNYER FX, Am J Clin Nutr, 76 (2002) S290. — 29. FLINT A, MØLLER BK, RABEN A, PEDERSEN D, TETENS I, HOLST JJ, Br J Nutr, 91 (6) (2004) 979. — 30. WOLEVER TM, BOLOGNESI C, J Nutr, 126 (1996) 2807. — 31. WOLEVER TM, KATZMAN-RELLEA L, JENKINS AL, VUKSAN, JOSSE RG, JENKINS DJ, Nutr Res, 14 (1994) 651. — 32. WOLEVER TM, VORSTER HH, BJÖRCK I, BRAND-MILLER J, BRIGHENTI F, MANNI JI, Eur J Clin Nutr, 57 (3) (2003) 475. — 33. VUKSAN V, JENKINS AL, DIAS AG, LEE AS, JOVANOVSKI E, ROGOVIK AL, Eur J Clin Nutr, 64 (4) (2010) 436. — 34. GANNON MC, NUTTALL FQ, Diabetes Care, 10 (1987) 759. — 35. GILBERTSON HR, THORBURN AW, BRAND-MILLER J, CHONDROS P, WERTHER GA, Am J Clin Nutr, 77 (1) 2003 83. — 36. AMERICAN DIABETES ASSOCIATION, Diabetes Care, 31(Suppl.1) (2008) S61. — 37. CANADIAN DIABETES ASSOCIATION, Can J Diab, 32 (Suppl.1) (2008). — 38. THE DIABETES AND NUTRITION STUDY GROUP (DNSG) OF THE EUROPEAN ASSOCIATION FOR THE STUDY OF DIABETES (EASD), Eur J Clin Nutr, 54 (2000) 353. — 39. BARCLAY AW, PETOCZ P, MCMILLAN-PRICE J, FLOOD VM, PRVAN V, MITCHELL P, Am J Clin Nutr, 87 (3) (2008) 627. — 40. SALMERON J, ASCHERIO A, RIMM EB, COLDITZ GA, SPIEGELMAN D, JENKINS DJ, Diabetes Care, 20 (1997) 545. — 41. STEVENS J, KYUNGMI A, JUHAERI M, HOUSTON D, STEFFAN L, COUPER D, Diabetes Care, 25 (2002) 1715. — 42. BRAND-MILLER JC, HOLT SHA, PAWLAK DB, MCMILLAN J, Am J Clin Nutr, 76 (2002) S281. — 43. WILLETT W, MANSON J, LIU S, Am J Clin Nutr, 76 (Suppl.1) (2002) S 274. — 44. VIRKAMÄKI A, YKI-JÄRVINEN H, Endocrinology, 134 (5) (1994) 2072. — 45. LIESE AD, SCHULZ M, FANG F, WOLEVER TM, D'AGOSTINO DB, SPARKS KC, Diabetes Care, 28 (2005) 2832. — 46. RICCARDI G, RIVELLESE A, GIACCO R, Am J Clin Nutr, 87 (Suppl 1) (2008) S 269 — 47. LAU C, FAERCH K, GLUMER C, TETENS I, PEDERSEN O, CARSTENSEN B, Diabetes Care, 28 (2005) 1397. — 48. RIZKALLA SW, LAROMIGUIERE M, HUET D, BOILLOT J, RIGOIR A, ELGRABLY F, Diabetes Care, 28 (2004) 1866. — 49. JENKINS DJA, KENDALL CWC, MCKEOWN-EYSSEN G, JOSSE RG, SILVERBERG J, BOOTH GL, JAMA, 300 (23) (2008) 2742. — 50. TSIHLIAS EB, GIBBS AL, MCBURNEY MI, WOLEVER TM, Am J Clin Nutr, 72 (2000) 439. — 51. WOLEVER TMS, BARBEAU MC, CHARRON S, HARRINGTON K, LEUNG S, MADRICK B, CJDC, 23 (2000) 56. — 52. WOLEVER TM, GIBBS AL, MEHLING C, CHIASSON JL, CONNELLY PW, JOSSE RG, Am J Clin Nutr, 87 (2008) 114. — 53. BOSELLO O, ARMELLINI F, ZAMBONI M, FITCHET M, Int J Obes, 21 (Suppl 1) (1997) S10. — 54. UK PROSPECTIVE DIABETES STUDY (UKPDS) Group, Lancet, 352 (1998) 837. — 55. LACHIN JM, GENUTH S, NATHAN DM, ZINMAN B, RUTLEDGE BN, Diabetes, 57(4) (2008) 995. — 56. MONNIER L, COLETTE C, Diabetes Care, 31 (Suppl. 1) (2008) S 150. — 57. BROWNLEE M, Nature, 414 (6865) (2001) 813. — 58. DE VEGT F, DEKKER JM, RUHE HG, STEHOUWER CD, NIJPELS, BOUTER LM, Diabetologia, 42 (1999) 926. — 59. BONORA E, Int J Clin Pract, 29 (Suppl. 1) (2002) S5. — 60. CALLE-PASCUAL AL, GOMEZ V, LEON E, BORDIU E, Diabetes Metab, 14 (1988) 629. — 61. COLLIER GR, GIUDICI S, KALMUSKY J, WOLEVER TMS, HELMAN G, WESSON V, Diab Nutr Metab, 1 (1988) 11. — 62. WOLEVER TM, HAMAD S, CHIASSON JL, JOSSE RG, LEITER LA, RODGER NW, J. Am Coll Nutr, 18 (1999) 242. — 63. WOLEVER TMS, JENKINS DJ, VUKSAN V, JENKINS AL, BUCKLEY GC, WONG GC, Diabetic Medicine, 9 (1992) 451. — 64. YUSOF BN, TALIB RA, KAMARUDDIN NA, KARIM NA, CHINNA K, GILBERTSON H, Diabetes Obes Metab, 11 (4) (2009) 387. — 65. LIU S, WILLETT WC, STAMPFER MJ, HU FB, FRANZ M, SAMPSON L, Am J Clin Nutr, 71 (2000) 1455. — 66. ORENCIA AJ, DAVIGLUS ML, DYER AR, WALSH M, GREENLAND P, STAMLER J, J Clin Epidemiol, 50 (1997) 1369. — 67. BALKAU B, SHIPLEY M, JARRETT RJ, PYORALA K, PYORALA M, FORHAN A, Diabetes Care, 21 (1998) 360. — 68. HU G, QIAO Q, TUOMILEHTO J, BALKAU B, BORCH-JOHNSEN K, PYORALA K, Arch Intern Med, 164 (2004) 1066. — 69. THOMAS DE, ELLIOTT EJ, BAUR L, Cochrane Database Syst. Rev, 18 (3) (2007). — 70. BRAND MILLER JC, Am J Clin Nutr, 59 (Suppl. 1) (1994) S747. — 71. LUSCOMBE ND, NOAKES M, CLIFTON PM, Eur J Clin Nutr, 53 (1999) 473. — 72. DU H, VAN DER ADL, VAN BAKEL MM, VAN DER KALLEN CJ, BLAAK EE, VAN GREEVENBROEK MM, Am J Clin Nutr, 87 (3) (2008) 655. — 73. CALLES-ESCANDON J, MIRZA SA, SOBEL BE, SCHNEIDER DJ, Diabetes, 47 (1998) 290. — 74. GRAIER WF, SIMECEK S, KUKOVETZ WR, KOSTNER GM, Diabetes, 45 (1996) 1386. — 75. LAVI T, KARASIK A, KOREN-MORAG N, KANETY H, FEINBERG MS, SHECHTER M, J Am Coll Cardiol, 53 (24) (2009) 2283. — 76. DIABETES AUSTRALIA. PRACTICE GUIDELINES FOR THE NUTRITIONAL MANAGEMENT OF TYPE 2 DIABETES MELLITUS FOR ADULTS 2006. Available from: URL: http://www.diabetesaustralia.com.au/education_info/ nebg.html.

D. Rahelić

University of Zagreb, Dubrava University Hospital, Division of Endocrinology, Diabetes and Metabolic Disorders, Av. Gojka Šuška 6, 10000 Zagreb, Croatia
e-mail: drahelic@kbd.hr

GLIKEMIČKI INDEKS U ŠEĆERNOJ BOLESTI

S A Ž E T A K

Glikemički indeks (GI) je sustav rangiranja ugljikohidratnih namirnica prema njihovom postprandijalnom odgovoru. Koncept GI je prvi put predstavljen u ranim ´80-tim godinama od strane prof. Jenkinsa i suradnika. Od tada su provedene brojne studije, koje govore u prilog učinkovitosti dijete niskog GI na regulaciju glikemije, na lipidni profil, serumske koncentracije inzulina i C-peptida, trombolitičke čimbenike, funkciju endotela i regulaciju tjelesne težine. Prema podacima iz literature čini se da dijeta niskog GI može djelomično spriječiti ili odgoditi vaskularne komplikacije šećerne bolesti. U literaturi postoje i kontradiktorni rezultati što je u ovom osvrtu i navedeno. Ipak, kliničari koji sudjeluju u liječenju bolesnika sa šećernom bolešću trebali bi biti svjesni moguće koristi dijete niskog GI u prevenciji i liječenju šećerne bolesti i njenih komplikacija.