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Effects of Dietary Insulin Index on Metabolic Parameters

Besin İnsülin İndeksinin Metabolik Parametreler Üzerine Etkileri

Betül GÜMÜŞ[1-2*], M. Emel ALPHAN[1]

[1] Istanbul Atlas University, Faculty of Health Science, Department of Nutrition and Dietetics, Istanbul, Turkey.
[2] Haliç University, Graduate Institute, Nutrition and Dietetics Doctoral Program, Istanbul, Türkiye.

Abstract

Carbohydrates are the main components that initiate insulin secretion, and the glycemic index and glycemic load ratios of a diet can be used to indirectly estimate the amount of insulin required after consuming carbohydrate-containing foods. However, insulin secretion is stimulated not only by carbohydrates but also by dietary proteins. When proteins are combined with carbohydrates, they play a synergistic role in increasing insulin levels and decreasing glycemia. The “food insulin index” is a concept developed to evaluate the postprandial insulin response of each food. This index directly measures the postprandial insulin response of the tested food compared to a reference food (glucose or white bread). Although carbohydrates are usually used as a measure of the glycemic index, the insulin index is calculated using energy, thus allowing the evaluation of foods that contain little or no carbohydrates. Considering that the insulinemic index values of foods are directly based on insulin release, it is thought that the insulinemic index may be more useful than the glycemic index ratio, especially in studies evaluating insulin exposure associated with conditions such as obesity. This review article aims to evaluate the effects of the food insulin index on blood parameters.

Keywords: Diet, Food insulin index, Glycemic index, Nutrition

Özet

Karbonhidratlar, insülin salınımını başlatan temel bileşenlerdir ve bir diyetteki glisemik indeks ve glisemik yük oranları, karbonhidrat içeren gıdaların tüketilmesinin ardından gerekli insülin miktarını dolaylı yoldan tahmin etmek için kullanılabilir. Ancak, insülin salınımı yalnızca karbonhidratlar tarafından değil, aynı zamanda diyetle alınan proteinler tarafından da uyarılmaktadır. Proteinler karbonhidratlarla birleştirildiklerinde, insülin seviyesinin yükselmesi ve gliseminin azalmasında sinerjik bir rol oynarlar. "Besin insülin indeksi”, her bir besinin postprandiyal insülin cevabını değerlendirmek amacıyla geliştirilmiş bir kavramdır. Bu indeks, referans bir besin (glukoz veya beyaz ekmek) ile karşılaştırıldığında test edilen besinin postprandiyal insülin cevabını doğrudan ölçer. Karbonhidratlar genellikle glisemik indeksin ölçütü olarak kullanılsa da, insülin indeksi enerji kullanarak hesaplanır, bu nedenle az karbonhidrat içeren veya hiç karbonhidrat içermeyen besinlerin de değerlendirilebilmesine imkan tanır. Besinlerin insülinemik indeks değerlerinin doğrudan insülin salınımına dayandığı göz önünde bulundurulduğunda, özellikle obezite gibi durumlarla ilişkili insülin maruziyetinin değerlendirildiği çalışmalarda, insülinemik indeksin glisemik indeks oranından daha faydalı olabileceği düşünülmektedir. Bu derleme makale besin insülin indeksinin kan parametreleri üzerindeki etkilerini değerlendirmeyi amaçlamaktadır.

Anahtar Kelimeler: Besin insülin indeksi, Beslenme, Diyet, Glisemik indeks

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*Corresponding Author: betul.kocak@atlas.edu.tr

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1. Introduction

Postprandial hyperglycemia and hyperinsulinemia play a significant role in the development of chronic diseases, including type 2 diabetes and coronary heart disease. Carbohydrates, the primary determinant of insulin secretion, exert this effect by directly increasing blood glucose levels. However, directly associating the amount of energy derived from carbohydrates with disease risk is incorrect, as dietary fiber and carbohydrates have important health effects (Caferoğlu & Özel, 2018).

The concept of "glycemic index" was first introduced by Jenkins et al. (Jenkins et al., 1981) to define carbohydrate quality. It describes how postprandial glycemia is affected by the food's form, dietary fiber content, and the type of carbohydrate present. The glycemic index (GI) of a food is defined as its ability to raise blood glucose levels within two hours after consumption (Rouhani et al., 2014). To calculate the glycemic index, the area under the blood glucose response curve for a test food containing 50 grams of carbohydrates is compared to the area under the curve for a reference food containing the same amount of carbohydrates (Akbulut et al., 2013).

Glycemic load (GL), on the other hand, considers both the quality and quantity of carbohydrates. It is calculated by multiplying the GI value of a food by the amount of carbohydrate (in grams) it contains (Rouhani et al., 2014). Consuming a high-GI diet can cause a rapid increase in postprandial plasma glucose levels, leading to a compensatory rise in plasma insulin levels (Kong et al., 2011). Studies have shown that in overweight and obese individuals with elevated insulin secretion, low-GI and/or low-GL diets have beneficial effects on weight loss (Joslowski et al., 2015).

The insulin response is not always proportional to the blood glucose response, as certain non-carbohydrate nutrients can also influence insulin secretion. In addition to carbohydrate-rich foods, protein- and fat-rich foods can also elicit a significant insulin response. Proteins, when consumed alongside carbohydrate-containing foods, enhance insulin secretion (Caferoğlu & Özel, 2018).

Hyperinsulinemia may contribute to albuminuria, which is measured by the albumin-creatinine ratio. It can also activate the renin-angiotensin system and the sympathetic nervous system, leading to hypertension (Kong et al., 2011).

Hyperinsulinemia is directly associated with metabolic disorders such as insulin resistance, hepatic steatosis, dyslipidemia, and hyperglycemia. These conditions may increase hepatic lipoprotein production and elevate glucose production. Additionally, hyperinsulinemia is linked to increased visceral adipose tissue, hypo-adiponectinemia, and hypercortisolemia. Due to the effects of hypercortisolemia, high-GI foods can disrupt the balance of appetite-stimulating (e.g., ghrelin) and appetite-suppressing (e.g., incretins) hormones, leading to less feeling of fullness and, consequently, obesity. Moreover, long-term consumption of high-GI foods may enhance chronic hunger signals accompanied by hyperinsulinemia and lipogenesis. These metabolic processes can overload pancreatic beta cells, potentially leading to the early onset of type 2 diabetes, particularly in genetically predisposed individuals (Kong et al., 2011).

*Corresponding Author: betul.kocak@atlas.edu.tr

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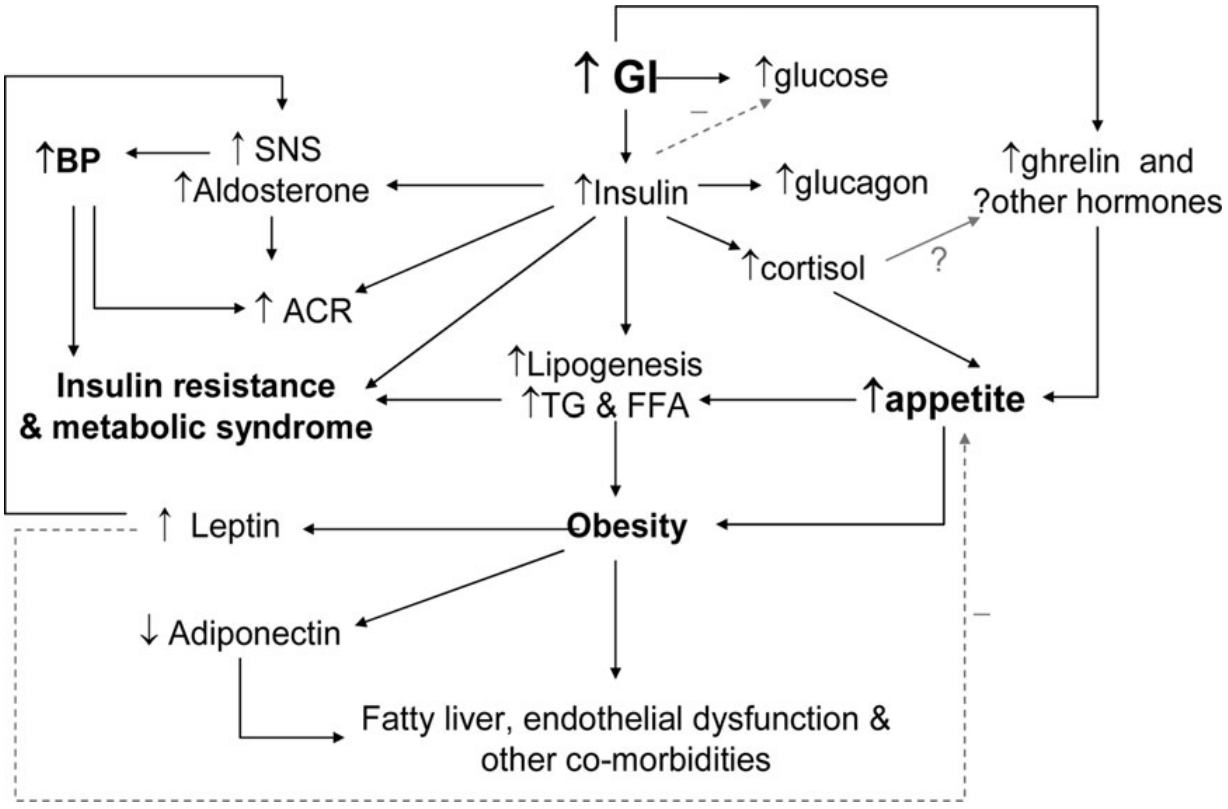

Figure 1. Associations between glycemic index (GI) and obesity

SNS: sympathetic nervous system, ACR: urine albumin-creatinine ratio, BP: blood pressure, FFA: free fatty acid, TG: triglyceride, Solid arrows indicate promotion or activation; dashed arrows with negative sign indicate suppression; question marks suggest possible mechanisms or involvements *(Kong et al., 2011).*

In individuals at risk for type 2 diabetes, dietary habits that lead to excessive insulin secretion may increase oxidative stress and impair the functionality of beta cells (Bao et al., 2009). Carbohydrates are the primary components that trigger insulin secretion, and the GI and GL ratios in a diet can be used to indirectly estimate the required amount of insulin following the consumption of carbohydrate-containing foods. However, insulin secretion is stimulated not only by carbohydrates but also by dietary proteins, which, when combined with carbohydrates, play a significant role in increasing insulin levels and reducing glycemia (Bao et al., 2009; Jakubowicz et al., 2015; Nimptsch et al., 2011).

Similarly, it has been suggested that adding fats to a carbohydrate-rich meal can reduce postprandial glycemia while increasing the insulin response (Bao et al., 2009; Jakubowicz et al., 2015).

A panel discussing the effects of carbohydrate quality on health recognized postprandial glycemia as a significant factor in overall health and considered dietary approaches that slow carbohydrate absorption as useful tools in reducing major chronic diseases and related risk factors. The panel concluded, based on clinical studies, that low-GI diets moderately improve glycemic control in type 1 and type 2 diabetes. However, evidence suggests that they have little or no effect on blood lipid levels and inflammation markers in both diabetic and non-diabetic individuals. The panel also acknowledged a strong association between a lower GI/GL diet and a reduced risk of developing T2DM in both men and women, as well as a lower risk of cardiovascular disease (CVD) in women. These health findings may highlight the need for more effective treatment strategies in sedentary, slightly overweight

*Corresponding Author: betul.kocak@atlas.edu.tr

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individuals with insulin resistance (Augustin et al., 2015). This review article aims to evaluate the effects of the food insulin index on blood parameters.

1.1. Food Insulin Index

The "food insulin index" is a concept developed to assess the postprandial insulin response of different foods (Jakubowicz et al., 2015). This index directly measures the postprandial insulin response of a test food compared to a reference food (glucose or white bread). While carbohydrates are generally used as a criterion for measuring GI, the insulin index is calculated based on energy intake, allowing the evaluation of foods that contain little or no carbohydrates (Nimptsch et al., 2011).

In the calculation, the insulin response obtained over a two-hour period after consuming a portion of the test food containing 1000 kJ (239 kcal) of energy is divided by the insulin response of the reference food and assessed based on the area under the curve (AUC) (Bao et al., 2009). To determine the insulin index, the cumulative changes in postprandial plasma glucose and insulin responses for each food are quantified as the incremental area under the 120-minute response curve (AUC). For each participant, the insulin score (%) for each test food is calculated by dividing the insulin AUC value of the test food by the insulin AUC value of white bread (reference food). The final insulin index is then expressed as a percentage using the following formula:

$$IS(\%) = \frac{\text{Area under the 120 min insulin response curve for 1000 kJ test food}}{\text{Area under the 120 min insulin response curve for 1000 kJ white bread}} \times 100$$

This formula is used to determine the insulin index (IS%) of a food. White bread is taken as the reference food and the insulin response of other foods is calculated as a percentage compared to white bread (Holt et al., 1997). In addition, the total insulin load (IL) of a diet can be calculated by combining the insulin indexes, energy contents and consumption frequencies of all consumed foods, thus estimating the overall insulin requirement of a diet (Jakubowicz et al., 2015). The total Glycemic Index (GI)/Glycemic Load (GL) and Insulin Index (II)/Insulin Load (IL) values of meals can be calculated using the GI and IL data of 121 different foods presented by Bao and colleagues (Bao et al., 2011) in 2011. The following formulas are used for these calculations. Here, "n" indicates the number of foods consumed, "GI_a" indicates the glycemic index of a food, and "II_a" indicates the insulin index of that food. In addition, "$Carbohydrate_a$" indicates the amount of digestible carbohydrates (total carbohydrates - dietary fiber) in a portion of food, "$Energy_a$" indicates the energy value in a portion of food, and "$Frequency_a$" indicates how often each food is consumed in a meal.

$$GI = \frac{\sum_{a=1}^{n}(GI_a \times Carbohydrate_a \times Frequency_a)}{\sum_{a=1}^{n}(Carbohydrate_a \times Frequency_a)}$$

$$II = \frac{\sum_{a=1}^{n}(II_a \times Energy_a \times Frequency_a)}{\sum_{a=1}^{n}(Energy_a \times Frequency_a)}$$

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$$GL = \frac{1}{100}\sum_{a=1}^{n}(GI_a \times Carbohydrate_a)$$

$$IL = \frac{1}{100}\sum_{a=1}^{n}(II_a \times Energy_a \times Frequency_a)$$

To this date, the insulin indexes (II) of 121 different foods and 13 mixed meals have been determined (Bao et al., 2011). Some foods with high II include baked beans (120), yogurt (115), and cornflakes (75), while examples of foods with moderate II include raw carrots (42) and corn (53). Foods with low II include milk (33), alcoholic beverages (white wine 3), and walnuts (7) (Nimptsch et al., 2011). The GI and II values of the 121 foods shared by Bao and colleagues in 2011 are presented in Table 1. Based on the obtained data, differences will emerge between a diet applied by considering the insulin index of foods and a diet based on low and moderate glycemic index foods. In light of the data, while milk, which has a low II value, is suitable for consumption, yogurt, which has a high II value, will not be suitable for a low II diet.

When protein-rich foods are examined, baked beans and white fish meat will not be suitable for a low-II diet due to their higher II values. However, since these foods fall into the low glycemic index category, they are used in glycemic index-based diets. When examining white fish and chicken meat, which have digestible carbohydrate amounts of 0, it is observed that fish meat, which contains 56 g of protein and has an II of 43, has a significantly higher II value compared to chicken meat, which contains 23 g of protein and has an II of 19. This is due to the fact that macronutrients other than carbohydrates in food also trigger an insulin response. As the protein content of foods increases, they stimulate the insulin response either synergistically or non-synergistically with carbohydrates. Therefore, GI and GL values, which are calculated solely based on carbohydrate levels, may not be sufficient to determine the insulin responses triggered by foods (Caferoğlu & Özel, 2018).

Table 1. Nutrient contents, GI and II values of foods

Foods	Weight (g or mL)	Protein (g)	Fat (g)	Dig CHO (g)	GI	II
Reference Food						
Glucose	59	0	0	59	100	100
Milk and Milk Products						
Milk (full fat)	368	11	14	17	31	24
Milk (1% fat)	558	20	6	27	29	34
Yogurt (low fat, strawberry)	260	12	5	38	31	84
Cheddar cheese	59	15	21	0	0	33
Cheddar cheese (93% fat free)	119	41	8	1	0	20
Cottage cheese/curd cheese low fat)	264	30	6	16	10	52
Cream/processed cheese	68	6	24	3	0	18
Ice cream (vanilla)	120	5	13	26	50	65

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Table 1. Nutrient contents, GI and II values of foods (Continue)

Cereals						
Cornflakes	67	5	0	55	81	82
Cereal (Special K)	63	13	1	44	69	86
Muesli bar	63	11	5	37	56	34
Fruits and Juices						
Apple	435	1	0	58	36	43
Orange	625	7	1	51	42	44
Banana	279	3	0	56	52	59
Melon	714	5	2	46	62	63
Black grapes	395	1	0	57	50	60
Raisins (seedless)	72	2	1	56	64	31
Avocado	112	2	25	0	0	4
Peaches (canned, in their own juice)	485	2	1	55	40	54
Raspberry jam	88	0	0	59	51	62
Vegetables						
Corn	222	6	3	43	47	39
Carbohydrate Rich Foods						
White bread	97	9	2	44	70	73
Whole grain bread	108	9	5	40	50	41
Potatoes (boiled, peeled)	368	10	1	49	78	88
White rice	203	5	1	56	72	58
White pasta	201	8	1	49	46	29
Protein-Rich Foods						
Eggs (boiled)	160	21	18	1	0	23
Beef steak	158	42	8	0	0	37
Chicken (fried in oil, with skin)	94	23	17	0	0	19
Chicken (oven-fried, skinless)	113	31	13	0	0	17
White fish	333	56	1	0	0	43
Tuna (canned, in water)	239	48	5	0	0	26
Tuna (canned, in oil, drained)	135	24	15	2	0	16
Shrimp (boiled, peeled)	235	48	4	2	0	21
Sausage (hot dog)	95	14	19	3	28	16
Baked beans (baked)	351	16	2	39	44	88
Lentils (in tomato sauce)	253	19	5	29	37	42
Peanuts (salted, roasted)	38	10	20	5	14	15
Peanut butter	41	9	20	7	14	11
Fat-Rich Foods						
Walnuts	35	6	23	1	0	5
Butter	33	0	27	0	0	2
Olive oil	27	0	27	0	0	3
Beverages						
Fruit juice	833	1	0	58	67	76
Ice tea (Ice tea)	622	0	0	59	59	69
Cola	595	0	0	59	53	44
Beer (4.9% alcohol)	671	2	0	13	66	20
White wine (11% alcohol)	362	1	0	1	0	3

Dig CHO= digestible carbohydrate, GI= Glisemic index, II=Insülin index

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Considering that the II values of nutrients are directly based on insulin release, it is thought that II may be more beneficial than GI, especially in studies evaluating insulin exposure related to conditions such as obesity (Nimptsch et al., 2011).

The important role of high postprandial insulin levels in increasing fat accumulation in the body can be explained by the fact that nutrients prefer to be stored in adipose tissue rather than muscle (Joslowski et al., 2012). In a study conducted on adults, it was reported that postprandial hyperinsulinemia seen at the 30th minute was a strong determinant of body weight gain and caused changes in waist circumference over a period of six years (Chaput et al., 2008). In addition, high insulin and low glucagon levels can limit glucose production in the liver and prevent the breakdown of fat. This situation is thought to be due to the high insulin demand for beta cells in the long term, reducing insulin sensitivity and increasing fat accumulation (Joslowski et al., 2012).

Another mechanism by which diets with high II/IL values may increase body fat may be through the mutually stimulating effects of insulin and insulin-like growth factor-1 (IGF-1) secretion. In vivo studies using adipocyte precursor cells have shown that increased IGF-1 levels may increase body fat by promoting the proliferation of preadipocyte cells. In addition, IGF-1 has been found to increase glucose uptake, accelerate lipogenesis, and inhibit lipolysis in preadipocyte and adipocyte cells (Joslowski et al., 2012).

1.2. Effects of Food Insulin Index on metabolic parameters

The first study investigating the effects of the food insulin index on postprandial glucose and insulin responses was published by Bell et al. in 2015 (Bell et al., 2015). In this study, both healthy individuals and individuals with type 2 diabetes were included, and two test meals were prepared with identical energy, macronutrient composition, and glycemic index (GI) values but different insulin index (II) values. The results showed that while the postprandial glucose responses of both meals were similar, the insulin response following the low-II meal was 53% lower in healthy individuals and 41% lower in individuals with type 2 diabetes (Bell et al., 2015).

Another study was conducted by Nimptsch et al., in which they analyzed blood samples from 4,002 healthy men and women. Dietary II and insulin load (IL) were assessed using food frequency questionnaires and data from directly analyzed or published II values of foods. It was found that participants in the highest quintile of II had triglyceride concentrations 26% higher compared to those in the lowest quintile. This relationship was more pronounced in obese participants. Additionally, dietary II was inversely correlated with HDL cholesterol in obese individuals. Similar relationships were observed for IL. However, dietary II and IL were not significantly associated with plasma C-peptide, HbA1c, LDL cholesterol, CRP, or IL-6. The study suggested that dietary II and IL may be particularly linked to plasma lipids in obese individuals (Nimptsch et al., 2011).

Another cross-sectional study was conducted with 8,691 adult participants aged 18–55 years. In terms of the dietary insulin index, men in the highest quintile of II were more likely to be overweight or obese compared to those in the lowest quintile. Furthermore, in a fully adjusted model, women in the highest

quintile of II were found to have a higher likelihood of general obesity compared to those in the lowest quintile (Anjom-Shoae et al., 2020). The studies concluded that adherence to a diet with a high II was associated with a higher likelihood of general obesity in women, whereas this association was not observed in men (Anjom-Shoae et al., 2020).

A study conducted in Tehran included 927 men and women aged 22–80 years. Fasting serum insulin and glucose levels were measured at baseline and after a three-year follow-up. Participants with higher IL had greater baseline weight and waist circumference. A borderline positive association was observed between II and the risk of insulin resistance. When comparing the highest and lowest tertiles of IL, the increase in insulin resistance risk was found to be statistically significant. The study concluded that dietary insulin load and II could be considered independent dietary risk factors for the development of insulin resistance (Mirmiran et al., 2016). This study highlights that II should not be overlooked in individuals with insulin resistance.

2. Conclusion

Although the studies conducted have shown the effects of II and IL on different parameters, the existence of studies showing that increased II and IL are associated with increased obesity prevalence and insulin resistance reveals the need for new studies on this subject. The effectiveness of a diet applied by considering II, is an open window waiting to be discovered in correcting anthropometric parameters as well as values related to hyperinsulinemia.

Authors Contributions

Topic selection: AME, GB; Study design: GB; Planning: AME, GB; Data collection and analysis: GB; Manuscript writing: AME, GB; Critical review: AME.

Conflict of Interest

There is no conflict of interest among the authors.

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