International Journal of Health Sciences and Research
www.ijhsr.org ISSN: 2249-9571

Review Article

Effect of Glycemic Index and Glycemic Load on Type 2 Diabetes Mellitus

Towhid Hasan[1], Marjia Sultana[1], Lincon Chandra Shill[1], Nafisa Habib Purba[1], Sara Sultana[2]

[1]Department of Food Technology and Nutrition Science, Noakhali Science and Technology University, Noakhali-3814, Bangladesh
[2]Department of Pharmacy, University of Dhaka, Dhaka-1000, Bangladesh

Corresponding Author: Marjia Sultana

ABSTRACT

Worldwide diabetes mellitus especially type 2 diabetes mellitus (T2DM) has become a common and rapidly growing chronic non-communicable disease with potentially devastating complications, posing major public health and socioeconomic challenges. The role of glycemic index (GI) and glycemic load (GL) in the control of the T2DM remain controversial. However, a number of mechanisms have been proposed through which dietary GI and/or GL can reduce the risk of T2DM, but all are suggestive. So, the present review is aimed to investigate the current understanding of the effect of GI and/or GL on the risk of T2DM.PubMed, Google Scholar, Cochrane library and several other databases were searched up to December 2018 to identify and select relevant studies. Research conducted using human are only considered eligible. Among the eighteen prospective cohort studies, eight have found no significant association of dietary GI and/or GL with T2DM risk, six studies revealed an association and four studies identified positive association only with GI but not with the GL. Among the ten interventional studies, only two indicated that the risk of T2DM increased with the increment of GI and GL. In conclusion, research on the glycemic response of foods needs to be expanded in order to get a broad and clear knowledge which will provide assurance that how dietary GI and/or GL have play role on the risk of T2DM.

Keywords: Glycemic index, Glycemic load, Type 2 diabetes mellitus, Carbohydrate.

INTRODUCTION

Diabetes mellitus, a chronic, metabolic disease, is characterized by elevated levels of blood glucose (or blood sugar), leading over time to serious damage to the heart, liver, blood vessels, eyes, kidneys, and nerves. Globally 1.6 million deaths are directly attributed to diabetes each year. [1,2] The number of people living with diabetes worldwide has been projected to increase from 366 million in 2011 to 552 million by 2030. [3] The most common type of diabetes is T2DM,usually occurs in adults, also referred to as "noninsulin-dependent diabetes mellitus" or "adult-onset diabetes mellitus" accounting for 90-95% of all diabetes. [4] In the past three decades, the prevalence of T2DM has risen dramatically in countries of all income levels particularly low- and middle-income countries have perceived the greater share of death and disability due to T2DM. [2] This form of diabetes encompasses individuals who have insulin resistance and usually relative

(rather than absolute) insulin deficiency and at least initially, and often throughout their lifetime, these individuals may not need insulin treatment for their survival. [4] The causes of T2DM are complex, but the main risk factors are overweight, including obesity, lack of physical activity, an increment in age (45 years or older), family background, high blood pressure (140/90 mm of Hg or above), and history of cardiovascular disease. [5]

Although many studies revealed that the cases of T2DM and its complications can be reduced by a healthy diet, regular physical activity, maintaining normal body weight and avoiding tobacco but the evidenceis not widely implemented. [1] An individual's diet is considered to contribute to the development of T2DM, in particular, the capacity that foods containing carbohydrates have to increase blood glucose level. [6,7] Carbohydrates are the main dietary components that have the greatest effect on glycemic response. [8] Glycemic response generally refers to the changes in blood glucose after consuming a carbohydrate-containing food. [9] GI and GL are two important measures of glycemic response to foods. In 1981, the concept of the GI was introduced by Jenkin to quantify the glycemic response to carbohydrates in different foods. [10] It measures the glycemic response to ingestion of a fixed amount of available carbohydrate in a test food compared with the same amount of available carbohydrate in a reference food (glucose or white bread) by the same subject. [11] GL is the mathematical product of the GI of a food and its carbohydrate content, has been proposed as a global indicator of the glucose response and insulin demand induced by a serving of food. [12] It has been suggested that diets with a high GI or high GL may predispose to higher postprandial blood glucose and insulin concentrations, which, in turn, increase glucose intolerance and impair pancreatic beta cell function and eventually lead to T2DM. [10]

Despite the existence of these two indexes for many years, the role of GI and GL in preventing type T2DM remains controversial. A number of studies have indicated an association of GI and/or GL with T2DM but there are many other large studies those have found no evidence to support the hypothesis. Under this circumstance, the present review is undertaken to reveal the current understanding between the effects of the glycemic index and/or glycemic load on type 2 diabetes mellitus.

MATERIALS AND METHODS

Sources of data

Several databases, including PubMed, Medline, Google scholar, Cochrane library etc. were searched for literature and extracted data till December 2018. Reference lists of the studies that were included in the analysis were also searched as well.

Search strategy

For an appropriate searching keyword like glycemic index, glycemic load, type 2 diabetes mellitus etc. were used.

Study selection

Any cross-sectional, case-control, cohort and experimental study on human providing sufficient information on the effect of GI and GL in preventing T2DMwas considered eligible for review.

RESULTS

Prospective cohort studies

In the current review, eighteen prospective cohort studies were explored to identify the association of GI and GL with the risk of T2DM (Table 1). Among the included studies, eight studies revealed no significant association but six studies showed that with the increment of dietary GI and GL, the risk of T2DM increased. Another four studies found a significant positive association of the risk the of T2DM only with dietary GI but not with the dietary GL.

Table 1: Prospective cohort studies identifying the effects of glycemic index and/or glycemic load on type 2 diabetes mellitus

Author, year [ref] Country	Subjects	Follow up period (year)	T2DM Assessment method	No. of T2DM cases	Results [HR/OR/RR (95% CI) in highest vs. lowest group of intakes]
Salmeron et al., 1997[11] USA	42759 M 40-75 y	6	Self-report and Confirmation	523	Dietary GI was positively associated with risk of T2DM, the combination of a high GL and a low cereal fiber intake further increased the risk of T2DM [GI: RR=1.37 (1.02-1.83); p<0.05, GL: RR=2.17 (1.04-4.54); p<0.05]
Salmeron et al., 1997[12] USA	65173 F 40-65 y	6	Self-report and Confirmation	915	Dietary GI and GL was positively associated with risk of T2DM [GI: RR=1.37 (1.09-1.71); p<0.05, GL: RR=1.47 (1.16-1.86); p<0.05]
Meyer et al., 2000[13] USA	35988 Postmenopausal women 55-69 y	6	Self-report	1141	Dietary GI and GL were not associated with T2DM [GI: RR= 0.89 (0.72-1.10); p>0.05, GL: RR=0.95 (0.78-1.16); p>0.05]
Stevens et al., 2002[14] USA	12251 M, F 45-64 y	9	Self-report and Confirmation	1447	There were no statistically significant associations of GI, or GL with incident of T2DM [GI: White: HR=1.002 (0.99–1.015); p>0.05, African American: HR=1.0 (0.98- 1.01); p>0.05, GL: White: HR=1.002 (1.000-1.004); p>0.05, African American: HR=0.999 (0.996–1.002); p>0.05]
Hodge et al., 2004[15] Australia	36787 M, F 40-69 y	4	Self-report and Confirmation	365	Dietary GI and GL were not associated with increased risk of T2DM [GI: OR=1.23 (0.98-1.54); p>0.05, GL: OR=1.04 (0.68-1.58); p>0.05]
Schulze et al., 2004 [16] USA	91249 female nurses 24-44 y	8	Self-report and Confirmation	741	Dietary GI was significantly associated with an increased risk of T2DM but GL was not significantly associated with risk [GI: RR=1.59 (1.21-2.10); GL: RR=1.33 (0.92-1.91); p>0.05]
Barclay et al., 2007[17] Australia	1833 M, F ⩾49y	10	Self-report or biochemical test	138	A significant positive association between GI and incident of T2DM was found for individuals aged <70 years, but no association was found for those aged ≥70 years [GI: <70 y HR=1.75 (1.05-2.92); p<0.05, GI: ≥70 y HR=0.80 (0.29-2.24); p>0.05]
Krishnan et al., 2007[18] USA	59000 Black women 21-69 y	8	Report of physician diagnosis	1938	Dietary GI but not GL were positively associated with risk of T2DM [GI: RR=1.23 (1.05-1.44); p<0.05, GL: RR=1.22 (0.98-1.51); p>0.05]
Mosdol et al., 2007[19] UK	7321 M, F 39-63 y	13	Self-report and Confirmation	329	Higher dietary GI and GL were not associated with an increased risk of T2DM [GI: HR=0.94 (0.71- 1.23); p>0.05, GL: HR=0.80 (0.51- 1.26); p>0.05]
Villegas et al., 2007[20] China	297755 F 40-70 y	5	Self-report and Confirmation	1608	Dietary GI and GL were all positively associated with the risk of T2DM [GI: RR=1.21 (1.03-1.43); GL: RR=1.34 (1.131.58)]
Sahyoun et al., 2008[21] USA	1898 M, F 70-79 y	4	Annual report of physician diagnosis	662	Neither dietary GI nor GL wasassociated with the risk of developing T2DM [GI: OR=1.0 (0.5-2.0); p>0.05, GL: OR=1.3 (0.6- 2.7); p>0.05]
Sluijs et al., 2010[22] Europe	37846 M, F 21-70 y	10	Self-report and Confirmation	915	Dietary GL and GI was associated with an increased T2DM risk [GL: HR=1.32 (1.14-1.54); p<0.001), GI: HR=1.08 (1.00-1.17); p=0.05]
Simila et al., 2011[23] Finland	25943 M Smokers 50-69 y	12	Identified from a national register	1098	Dietary GI and GL were not associated with T2DM risk [GI: RR=0·87 (0.71-1.07); p>0.05, GL: RR=0·88 (0·65-1·17); p>0.05]
van Woudenbergh et al., 2011[24] Netherlands	4366 M, F ≥55 y	6	Medical examinations	456	GI and GL were not associated positively with risk of T2DM [GI: RR=0.95 (0.75-1.21); p>0.05, GL: RR=1.00 (0.74-1.36); p>0.05]
Sakurai et al., 2012[25] Japan	1995 M 35-55 y	6	Annual medical examinations	133	GI, but not GL, had a significant positive association with the incidence of T2DM [GI: HR=1.96 (1.04-3.67), GL: HR=1.24 (0.65-2.34)]
Oba et al., 2013[26] Japan	64633 M, F 45-75 y	5	Self-report		Dietary GL and GI was positively associated with the risk of T2DM among women [GL: OR=1.52 (1.13-2.04);p<0.05, GI: OR=1.46 (0.94-2.28); p<0.05]
Rossi et al., 2013[27] Greece	22295 M, F 35-70 y	11	Self-report and Confirmation	2330	Low dietary GL adequately adheres to the principles of the traditional Mediterranean diet may reduce the incidence of T2DM [GL: HR=1.21 (1.05-1.40)]
Sluijs et al., 2013[28] Europe	519978 M, F 35-70 y	12	Self-report	11559	Higher dietary GI and GL was not significantly increased the risk of T2DM [GI: HR=1.05 (0.96-1.16); p>0.05, GL: HR=1.07 (0.95-1.20); p>0.05]

Note: F=Female, GI=Glycemic index, GL=Glycemic load, HR=Hazard ratio, M=Male, OR=Odds ratio,RR=Relative risk, T2DM= Type 2 diabetes mellitus, y=Years.

Interventional studies

A number of intervention studies have been evaluated to show the importance of low GIand low GL diets to improve glycemic control in people with T2DM(Table 2). These randomized controlled trials (RCTs) with interventions lasted 1-12 month compared low-GI diets with control diets including high-GI diets. Outcomes of interest were glycated hemoglobin (HbA1c) (chronic glucose control as estimated by HbA1c; higher amounts of HbA1c indicating poorer control of blood glucose levels) and fructosamine, both were used as the measure of glycemic control. Among the ten interventional studies, only two have shown a significant positive association between both dietary GI and GL, and the risk of T2DM. Other eight studies have shown no improvements in glycemic control following a low GI diet as compared to a high GI diet.

Table 2: Interventional studies investigating the effects of glycemic index and/or glycemic load on type 2 diabetes mellitus

Author, year [ref]	Subjects	Study duration	Dietary intervention	Results	Comments
Fontvieille et al., 1992[29]	6	6 weeks	The high GI diet (enriched in bread and potato) vs. low GI diet (pasta, rice, and legumes)	No significant differences were found in HbA1c value between the two different level of GI diet	GI has no effect on T2DM
Wolever et al., 1992[30]	6 (53-72 y) (overweight)	18 weeks	High GI vs. low-GI diets Both diets have similar composition (57% carbohydrate, 23%fat, and 34 g/day dietary fiber), but the low-GI diet had a GI of 58 compared to 86 for the high-GI diet	The mean levels of serum fructosamine on the low-GI diet (4.61±0.48) were significantly less than those on the high-GI diet (5.02±0.60) that was by 8% ($p<0.05$)	Low GI diets improves overall blood glucose control
Jarvi et al., 1999[31]	20	48 days	High GI vs. low GI diets	There was a significant difference in the changes of serum fructosamine concentrations between the diets ($p<0.05$)	Low GI diet lower glucose response and able to improve glycemic control
Heilbronn et al., 2002[32]	45 (overweight)	12 weeks	High GI (high GL) vs. low GI (low GL) diets	HbA1c was reduced twofold (-2.8%) more in subjects consuming a low-GI diet as compared to subjects consuming a high GI diet ($p<0.05$)	Low GI diet was able to improve glycemic control as compared to high GI diet
Jimenez-Cruz et al., 2003[33]	36 (obese)	18 weeks	High GI (high GL) vs. low-GI (low GL) diets	HbA1c was also significantly lower after the low GI period compared with after the High GI period ($p<0.05$)	Low GI diet may help to improve the metabolic control in T2DM obese subjects
Rizkalla et al., 2004[34]	12	12 weeks	High GI vs. low-GI diets	Improvement of HbA1c was lower at the end of the low GI diet than at the end of the high GI diet ($p<0.05$)	Low GI diet was able to improve glycemic control as compared to high GI diet
Jenkins et al., 2008[35]	210 (overweight)	6 months	High GI and GL diet (35 g of fiber, GI of 86, and GL of 201) vs. low GI and GL diet (42 g of fiber, GI of 62, and GL of 141)	The reduction in dietary GI and GL related positively to the reduction in HbA1c concentration ($r=0.35$, $p<0.001$) and ($r=0.24$, $p<0.05$)	Low GI and GL diet resulted in lower HbA1c levels compared with a high GI and GL diet thus improve glycemic control.
Wolever et al., 2008[36]	162	1 y	Low-GI diet (20% protein, 25% fat, 50% carbohydrates with 55% high GI) vs. High-GI diet (20% protein, 30% fat, 45% carbohydrates with 63% high-GI)	There was no significant difference in HbA1c value with the different diets ($p>0.05$)	In subjects with T2DM, long-term HbA1c was not affected by altering the GI.
Jenkins et al., 2012[37]	121	3 months	Low GI diet emphasizing legume consumption vs. high GI diet emphasizing high wheat fiber foods	The low GI legume diet reduced HbA1c values by 0.5% (95% CI, -0.6% to -0.4%) but high GI diet reduced HbA1c values by 0.3% (95% CI, -0.4% to -0.2%)	Low GI diet improved both glycemic control and reduced calculated CHD risk score in T2DM.
Farvid et al., 2014[38]	640	1 y	High GI vs. low-GI diets	OR=2.58 (1.08-6.15), p=0.02 for FSG and OR=3.05 (1.33-7.03), p=0.008 for HbA1c between the highest vs. the lowest quartile of GL	Dietary GL was positively associated with risk of hyperglycemia but no statistically significant association was observed for GI in relation to either FSG or HbA1c

Note: FSG= Fasting serum glucose, GI=Glycemic index, GL=Glycemic load, HbA1c = Glycated hemoglobin, T2DM= Type 2 diabetes mellitus, r= correlation coefficient, y=Year.

DISCUSSION

In this review, there lies an incongruity in the prospective cohort studies while exploring the relationship of GI and GL with the risk of T2DM. Some studies claimed that risk of T2DM increased as dietary GI and/or GL increased while others refuted that claim. Controversy persists among the interventional studies also. Several studies found a positive association of increased GI and/or GL in the diet with the T2DM risk, but others showed no association. However, the studies showing the effect of GI and/or GL on type 2 diabetes mellitus provide some promising evidence of how low dietary GI and/or GL lowered the risk of T2DM.

Decreasing insulin demand or improving insulin sensitivity may be the possible way by which the risk of T2DM may be scaling down. Over a period of years of exposure of hyperglycemia, pancreatic β-cells function becomes reduced, resulting in glucose intolerance and finally an irreversible state of diabetes. Thus, the mechanism for these phenomena is not entirely clear yet. Nevertheless, it is predicted that a diet producing higher blood glucose concentrations and greater demand for insulin may increase the risk of T2DM. [39] Several physiologic mechanisms have been proposed to explain the effect of GI and GL diet on the risk of T2DM. [39,40] High-GI and high-GL diets are known to stimulate the increased production of insulin, resulting in a state of hyperinsulinemia, which, in turn, can induce insulin resistance. In addition, the consumption of high-GI and high-GL diets for several years can increase the demand for pancreatic β-cells and lead to β-cell exhaustion and ultimate failure. [41] Furthermore, high-GI and high-GL diets are said to increase the concentration of blood glucose and free fatty acids, chronic exposure to these elevated concentrations can induce β-cell failure leading to T2DM. [10,39] The individual's glycemic response is also influenced by the degree of underlying insulin resistance that is further determined primarily by the degree of adiposity, physical activity, genetics, and other aspects of diet. Thus, it might be surmised that the negative metabolic effects of high-GIand high-GL foods would be inflamed in sedentary, overweight, or genetically susceptible persons. [39]

CONCLUSION

Diabetes particularly type 2 diabetes mellitus, is not a disease of affluence now, but has become a burden for the whole society affecting the rich and the poor alike. Numerous studies along with the World Health Organization (WHO) have reported the devastating health outcome of T2DM together with a serious economic crisis. As diabetes is a life-long disease and cannot be cured completely, several attempts have been taken to control diabetes and dietary measures have proved to be the most effective one. Dietary GI and GL are two valuable measures used to control blood glucose level which in turn control diabetes.

In the present review, an attempt has been made to provide a comprehensive detail of the association of overall dietary GI and/or GL with T2DM risk. The findings are inconsistency, yet, there is limited evidence on the optimal dietary approach to control hyperglycemia in T2DM.So, making any consensus about the effect of GI and/or GL on T2DM is very crucial. Therefore, to geta better understanding of this area of knowledge, further investigation is necessary to generate uncontroversial evidence.

Conflict of Interest: None.

REFERENCES

1. WHO. Diabetes [Internet]. World Health Organization; 2018. [updated 2018; cited 2018 Dec 2]. Available from: https://www.who.int/diabetes/en/.
2. Hasan T, Sultana M. Antidiabetic Potency of Bangladeshi Medicinal Plants. J Ayurvedic Herb Med. 2018;4(1):35-42.

3. Unwin N, Guariguata L, Whiting D, Weil C. Complementary approaches to estimation of the global burden of diabetes. Lancet. 2012;379(9825):1487-1488.
4. ADA. Classification and Diagnosis of Diabetes. American Diabetes Association. Diabetes Care. 2016;39 Suppl 1:S13-S22.
5. NIDDK. Symptoms and causes of diabetes [Internet]. National Institute of Diabetes and Digestive and Kidney Diseases; 2018. [updated 2016 Nov; cited 2018 Nov 30]. Available from: https://www.niddk.nih.gov/health-information/diabetes/overview/symptoms-causes.
6. Sheard NF, Clark NG, Brand-Miller JC, Franz MJ, Pi-Sunyer FX, Mayer-Davis E, et al. Dietary carbohydrate (amount and type) in the prevention and management of diabetes: a statement by the American Diabetes Association. Diabetes Care. 2004;27(9):2266-2271.
7. Greenwood DC, Threapleton DE, Evans CE, Cleghorn CL, Nykjaer C, Woodhead C, et al. Glycemic index, glycemic load, carbohydrates, and type 2 diabetes: systematic review and dose-response meta-analysis of prospective studies. Diabetes Care. 2013;36(12):4166-4171.
8. Bhupathiraju SN, Tobias DK, Malik VS, Pan A, Hruby A, Manson JE, et al. Glycemic index, glycemic load, and risk of type 2 diabetes: results from 3 large US cohorts and an updated meta-analysis. Am J Clin Nutr. 2014;100(1): 218-232.
9. Hubrich B, O'Brien L. Formulating Glycemic Strategies. Virgo Publishing; 2006.
10. Jenkins DJ, Wolever TM, Taylor RH, Barker H, Fielden H, Baldwin JM, et al. Glycemic index of foods: a physiological basis for carbohydrate exchange. Am J Clin Nutr. 1981;34(3): 362-366.
11. Salmeron J, Ascherio A, Rimm EB, Colditz GA, Spiegelman D, Jenkins DJ, et al. Dietary fiber, glycemic load, and risk of NIDDM in men. Diabetes Care. 1997;20(4):545-550.
12. Salmeron J, Manson JE, Stampfer MJ, Colditz GA, Wing A, Willett WC. Dietary fiber, glycemic load, and risk of non-insulin-dependent diabetes mellitus in women. JAMA. 1997;277(6):472-477.
13. Meyer KA, Kushi LH, Jacobs DR, Jr., Slavin J, Sellers TA, Folsom AR. Carbohydrates, dietary fiber, and incident type 2 diabetes in older women. Am J Clin Nutr. 2000;71(4):921-930.
14. Stevens J, Ahn K, Juhaeri, Houston D, Steffan L, Couper D. Dietary fiber intake and glycemic index and incidence of diabetes in African-American and white adults: the ARIC study. Diabetes Care. 2002;25(10):1715-1721.
15. Hodge AM, English DR, O'Dea K, Giles GG. Glycemic index and dietary fiber and the risk of type 2 diabetes. Diabetes Care. 2004;27(11):2701-2706.
16. Schulze MB, Liu S, Rimm EB, Manson JE, Willett WC, Hu FB. Glycemic index, glycemic load, and dietary fiber intake and incidence of type 2 diabetes in younger and middle-aged women. Am J Clin Nutr. 2004;80(2):348-356.
17. Barclay AW, Flood VM, Rochtchina E, Mitchell P, Brand-Miller JC. Glycemic index, dietary fiber, and risk of type 2 diabetes in a cohort of older Australians. Diabetes Care. 2007;30(11):2811-2813.
18. Krishnan S, Rosenberg L, Singer M, Hu FB, Djousse L, Cupples LA, et al. Glycemic index, glycemic load, and cereal fiber intake and risk of type 2 diabetes in US black women. Arch Intern Med. 2007;167(21):2304-2309.
19. Mosdol A, Witte DR, Frost G, Marmot MG, Brunner EJ. Dietary glycemic index and glycemic load are associated with high-density-lipoprotein cholesterol at baseline but not with increased risk of diabetes in the Whitehall II study. Am J Clin Nutr. 2007;86(4):988-994.
20. Villegas R, Liu S, Gao YT, Yang G, Li H, Zheng W, et al. Prospective study of

dietary carbohydrates, glycemic index, glycemic load, and incidence of type 2 diabetes mellitus in middle-aged Chinese women. Arch Intern Med. 2007; 167(21):2310-2316.
21. Sahyoun NR, Anderson AL, Tylavsky FA, Lee JS, Sellmeyer DE, Harris TB. Dietary glycemic index and glycemic load and the risk of type 2 diabetes in older adults. Am J Clin Nutr. 2008; 87(1):126-131.
22. Sluijs I, van der Schouw YT, van der AD, Spijkerman AM, Hu FB, Grobbee DE, et al. Carbohydrate quantity and quality and risk of type 2 diabetes in the European Prospective Investigation into Cancer and Nutrition-Netherlands (EPIC-NL) study. Am J Clin Nutr. 2010;92(4):905-911.
23. Simila ME, Valsta LM, Kontto JP, Albanes D, Virtamo J. Low-, medium- and high-glycaemic index carbohydrates and risk of type 2 diabetes in men. Br J Nutr. 2011;105(8):1258-1264.
24. van Woudenbergh GJ, Kuijsten A, Sijbrands EJ, Hofman A, Witteman JC, Feskens EJ. Glycemic index and glycemic load and their association with C-reactive protein and incident type 2 diabetes. J Nutr Metab. 2011;2011: 623076.
25. Sakurai M, Nakamura K, Miura K, Takamura T, Yoshita K, Morikawa Y, et al. Dietary glycemic index and risk of type 2 diabetes mellitus in middle-aged Japanese men. Metab. 2012;61(1):47-55.
26. Oba S, Nanri A, Kurotani K, Goto A, Kato M, Mizoue T, et al. Dietary glycemic index, glycemic load and incidence of type 2 diabetes in Japanese men and women: the Japan Public Health Center-based Prospective Study. Nutr J. 2013;12(1):165.
27. Rossi M, Turati F, Lagiou P, Trichopoulos D, Augustin LS, La Vecchia C, et al. Mediterranean diet and glycaemic load in relation to incidence of type 2 diabetes: results from the Greek cohort of the population-based European Prospective Investigation into Cancer and Nutrition (EPIC). Diabetologia. 2013;56(11):2405-2413.
28. Sluijs I, Beulens JW, van der Schouw YT, van der AD, Buckland G, Kuijsten A, et al. Dietary glycemic index, glycemic load, and digestible carbohydrate intake are not associated with risk of type 2 diabetes in eight European countries. J Nutr. 2013; 143(1):93-99.
29. Fontvieille AM, Rizkalla SW, Penfornis A, Acosta M, Bornet FR, Slama G. The use of low glycaemic index foods improves metabolic control of diabetic patients over five weeks. Diabet Med. 1992;9(5):444-450.
30. Wolever TM, Jenkins DJ, Vuksan V, Jenkins AL, Wong GS, Josse RG. Beneficial effect of low-glycemic index diet in overweight NIDDM subjects. Diabetes Care. 1992;15(4):562-564.
31. Jarvi AE, Karlstrom BE, Granfeldt YE, Bjorck IE, Asp NG, Vessby BO. Improved glycemic control and lipid profile and normalized fibrinolytic activity on a low-glycemic index diet in type 2 diabetic patients. Diabetes Care. 1999;22(1):10-18.
32. Heilbronn LK, Noakes M, Clifton PM. The effect of high- and low-glycemic index energy restricted diets on plasma lipid and glucose profiles in type 2 diabetic subjects with varying glycemic control. J Am Coll Nutr. 2002;21(2): 120-127.
33. Jimenez-Cruz A, Bacardi-Gascon M, Turnbull WH, Rosales-Garay P, Severino-Lugo I. A flexible, low-glycemic index mexican-style diet in overweight and obese subjects with type 2 diabetes improves metabolic parameters during a 6-week treatment period. Diabetes Care. 2003;26(7):1967-1970.
34. Rizkalla SW, Taghrid L, Laromiguiere M, Huet D, Boillot J, Rigoir A, et al. Improved plasma glucose control, whole-body glucose utilization, and lipid profile on a low-glycemic index diet in type 2 diabetic men: a

randomized controlled trial. Diabetes Care. 2004;27(8):1866-1872.
35. Jenkins DJ, Kendall CW, McKeown-Eyssen G, Josse RG, Silverberg J, Booth GL, et al. Effect of a low-glycemic index or a high-cereal fiber diet on type 2 diabetes: a randomized trial. JAMA. 2008;300(23):2742-2753.
36. Wolever TM, Gibbs AL, Mehling C, Chiasson JL, Connelly PW, Josse RG, et al. The Canadian Trial of Carbohydrates in Diabetes (CCD), a 1-y controlled trial of low-glycemic-index dietary carbohydrate in type 2 diabetes: no effect on glycated hemoglobin but reduction in C-reactive protein. Am J Clin Nutr. 2008;87(1):114-125.
37. Jenkins DJ, Kendall CW, Augustin LS, Mitchell S, Sahye-Pudaruth S, Blanco Mejia S, et al. Effect of legumes as part of a low glycemic index diet on glycemic control and cardiovascular risk factors in type 2 diabetes mellitus: a randomized controlled trial. Arch Intern Med. 2012;172(21):1653-1660.
38. Farvid MS, Homayouni F, Shokoohi M, Fallah A, Farvid MS. Glycemic index, glycemic load and their association with glycemic control among patients with type 2 diabetes. Eur J Clin Nutr. 2014; 68(4):459-463.
39. Willett W, Manson J, Liu S. Glycemic index, glycemic load, and risk of type 2 diabetes. Am J Clin Nutr. 2002;76(1): 274s-280s.
40. Ludwig DS. The glycemic index: physiological mechanisms relating to obesity, diabetes, and cardiovascular disease. JAMA. 2002;287(18):2414-2423.
41. Pawlak DB, Kushner JA, Ludwig DS. Effects of dietary glycaemic index on adiposity, glucose homoeostasis, and plasma lipids in animals. Lancet. 2004; 364(9436):778-785.

How to cite this article: Hasan T, Sultana M, Shill LC et.al. Effect of glycemic index and glycemic load on type 2 diabetes mellitus. Int J Health Sci Res. 2019; 9(2):259-266.

******