Association between dietary glycemic index and glycemic load, insulin index and load with incidence of age-related cataract: Results from a case-control study

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Highlights:

- The highest quartiles of dietary glycemic index were significantly, positively associated with the risk of age-related cataracts, compared to those in the lowest quartile, in an Iranian population.
- There was a positive association between higher dietary glycemic load and cataract risk.
- We found a significant, direct, relationship between dietary insulin load and risk of cataract, but the association between dietary insulin index and the risk of cataract was not significant.

Abstract

Aim: To identify the association between the dietary carbohydrate indexes, such as dietary glycemic index (DGI) and load (DGL), dietary insulin index (DII) and load (DIL), with the possibility of cataract.

Method: This case–control study consisted of 101 new cases of cataract and 202 controls. DGI and DGL were computed through DGI values previously published. DII was also calculated based on dietary insulin index data published previously.

Results: There was a significant positive association between the highest quartiles of DGI (OR=6.56; 95% CI=2.67-16.06; P<0.001), DGL (OR=6.17; 95% CI=1.93-19.37; P= 0.002) and DIL (OR=4.17; 95% CI=1.41-12.27; P= 0.004) with risk of cataract, compared to those on the lowest quartile, but not for DII (OR=0.85; 95% CI=0.39-1.86; P= 0.82). Furthermore, after stratifying groups by BMI, a significant direct association between highest quartile of DGI (OR=6.76; 95% CI=2.49-18.38; P<0.001) and DGL (OR=3.45; 95% CI=0.96-12.37; P=0.05) with risk of cataract was evident in individuals with elevated BMI (BMI≥25).

Conclusion: We found a significant, direct, relationship between DGI, DGL and DIL with risk of cataract. However, the association between DII and the risk of cataract was not significant, even after adjusting for related confounders.

Keywords: GI, GL, DII, DIL, cataract

Introduction

Age-related cataract is considered as one of the most prevalent causes of poor vision and loss of eye sight in the world (1-3). In Iran, approximately 31.7% of loss of vision and 47.5% of ophthalmic disorders are attributable to cataract, respectively (1, 4). The most effective treatment for this disease is cataract surgery, which imposes a heavy societal economic burden (5). Therefore identification of modifiable cataract risk factors would be pragmatic in the development of nonsurgical strategies for postponing or preventing the incidence of age-related cataract (6). There are various risk factors for development of this disease, including genetic factors, aging, exposure to UV-B light, and diabetes (1, 6). In addition it has been suggested that other factors, such as environmental agents and metabolic related disease may be related to cataract incidence (1). Amongst the various environmental agents, diet is purported to possess an imperative function in development of this disease, acting, either, as a protective agent, or a risk factor (1, 7).

Some studies have provided strong evidence to indicate a direct association between high carbohydrate intakes and cataract incidence (3, 6, 8, 9). Among the macronutrients, carbohydrate is the principle factor for regulation of postprandial blood glucose levels and consequently postprandial insulin secretion (10). Furthermore, other macronutrients, such as protein and fat, play a role in increasing insulin secretion, despite generating modest blood glucose responses (10, 11). GI is defined as a measurement index of how much each carbohydrate-containing food elevates blood glucose in comparison with a standard food of either glucose or white bread (12, 13). In addition, Glycemic Load (GL), which represents both the quantity and quality of carbohydrate in the diet, is measured utilizing the GI of food and its non-fiber carbohydrate content values (13). However, in addition to postprandial insulin response to blood glucose levels, there are other factors which impact on insulin secretion, such as fructose, certain amino acids, and fatty

acids (11, 14). Accordingly, to evaluate postprandial insulin responses to all of the mentioned insulinotropic agents, the dietary insulin index (DII) was conceived, which may be used to investigate the insulin response of healthy people after consumption of a specific food rather than a reference food with equal energy (identical to the GI, either white bread or glucose) (11, 15).

There are some conceivable associations between several food groups and nutrients with risk of age-related cataract (9); indeed, some studies have reported that high consumption of vegetables and fruits may be protective factors against cataract (16-18), however others found no association (19, 20). Furthermore, Theodoropoulou reported that increased intake of fish, and decreased consumption of meat, are associated with lower risk of cataract (9). Laboratory and epidemiological data have indicated that high glycemic index (GI) diets, and consequently high blood sugar levels, may be risk factors for the progression of age related diseases, such as cataract (21, 22). Some studies have demonstrated a direct relationship between higher GI diets and cataract incidence (6, 8), although Turati et al reported a positive association between higher glycemic load (GL) and the risk of cataract, but no association with GI (23).

Currently, little is known about the relationship between DGI, DGL, DII and DIL and the risk of age-related cataract. Therefore, clearly, the association between cataract progression and the mentioned dietary indexes is a matter of great scientific interest. However, because of the inconsistency reported in the little available evidence, we sought to investigate the association between the above indexes with cataract incidence.

Subjects and methods

The present case-control study was carried out in Farabi ophthalmology and Shariati hospitals, in Tehran. The participants were chosen using convenience sampling. In total, 202 controls and 101 cases enrolled the study. The formulas for calculating sample size was mentioned in a previous study by detail (1). During this study, newly diagnosed cataract patients (<1 month) were recruited. Cataract diagnosis was confirmed according to the gradual opacity of eye lenses, leading to blurred eyesight, detected by an ophthalmologist (4, 24). The case group was entered into the study according to the following inclusion criteria: the diagnosis of the cataract should not exceed one month prior to the study commencing, the diagnosis of cataract in at least one eye, the eyesight degree should not exceed more than 0.6, in addition, only participants aged ≥ 40 years were recruited. The subjects in the control group were chosen from non-hospitalized subjects or hospitalized patients not suffering from cataract, the age range more than 40 years, and the eyesight degree ≤ 0.6. The exclusion criteria for both groups were: suffering from another eye disease, adherence to any non-normal dietary habit less than one year prior to the interview, former history of eye injury, radiation therapy or eye surgery. At the end of the study, eight participants from cases and controls (four from each group) were omitted from the study because of their daily abnormal caloric intakes, which were less than 702 kcal, or more than 5016 kcal, per day.

The control group was matched for age (with a five-year interval) and sex with the case group, resulting in double the number of control vs case participants.

Information regarding; age, the history of cataract in the family, heart disease, hypertension (systolic blood pressure ≥ 160 mm Hg or diastolic blood pressure ≥100 mm Hg or antihypertensive medicine consumption), diabetes, hyperlipidemia, arthritis, smoking, alcohol consumption, physical activity, prior intake of vitamin C and multivitamin supplement, corticosteroids consumption, oral contraceptives, estrogen therapy, as well as the number of hours exposing to

sunlight, and using special equipment against the sun, was obtained by an interview with the subjects. The weight of each subject was measured, while wearing light clothing and unshod, using a 100g sensitivity scale, and height using a tape measure with the sensitivity of 0.1 cm, respectively. Body mass index (BMI) was then calculated by dividing weight in kilograms by height in meters squared. Written informed consent was attained from all subjects prior to study commencement. The study was approved by the ethics committee at the National Nutrition and Food Technology Research Institute of Shahid Beheshti University of Medical Science. (Ethic number: 051525)

Dietary assessment

All participants' dietary intakes in the preceding year were assessed using a validated and reliable 147-item food frequency questionnaire (FFQ) (25-27). The amounts of each nutrient was converted into grams by a household scale guideline, and the quantity of each nutrient which is consumed per day was computed in grams for each subject (28). For all food items, calories and sub nutrients were measured by the table of food ingredients of United States department of agriculture (29, 30). Next, the data were transferred into SPSS software for analysis. Dietary factors of food groups, such as total fat, grains, dairy, meat, fruit and vegetable, saturated fatty acids, and cholesterol intake were examined in the current study.

Calculation of GI and GL

Dietary GI and GL were derived from the FFQ as follows (31) :

Dietary GI = [(carbohydrate content of each food item) × (number of servings/d) × (GI)] / total daily carbohydrate intake

Dietary GL= (carbohydrate content of each food item) × (number of servings/d) × (GI)

The GI and GL values were calculated based on Tehran Lipid and Glucose Study (12). The GI values of food items were prepared from the GI international table (31), the online database of GI, which is maintained by the University of Sydney (32), and from studies reporting Iranian foods' GI (33). The GI for whole and refined grain, potatoes, starchy vegetables, legumes and some of fruits was obtained from the studies that reported the Iranian foods' GI (33), and the GI for fruits, dairy products, and nuts were gathered from the GI international table (31). The reference of GI values was white bread (GI for white bread =100). In instances of multiple GI values for one food, we computed the mean of GI values for analysis. If the GI value of a food was not specified, some other food item was allocated based on similarity to the food item. In addition, food items which had a very low carbohydrate content were omitted from the analysis because of inability to the precise measurement of their GI values (12).

Calculation of dietary insulin index (DII) and dietary insulin load (DIL)

Dietary insulin index value for each food component compares the postprandial insulin response relative to a reference food such as glucose or white bread (15). Insulin index values for foods found in the food-frequency questionnaire were determined, either, from published estimates (31 foods) (34, 35), or, from direct testing of food items at the University of Sydney, Australia (73 foods; provided by Jennie Brand-Miller). The test procedure is provided the Holt study in detail. Briefly, the study was conducted in a 2-hour duration with 15-minute intervals of evaluating insulin levels after consumption of isoenergetic food groups, then, the data was utilized as below (34):

DII = area under 120-min insulin response curve for 1000 KJ test food / area under 120-min insulin response curve for 1000 KJ reference food (white bread)

Insulin index values for each meal showed an average response of 11 to 13 subjects.

For calculation of dietary insulin load (DIL), we first computed the insulin load of each food as follows (15):

Dietary insulin load = Σ [insulin index of food × energy content of food (Kcal/serving) × frequency of consumption (servings of food/day)

Each unit of DIL equal the insulin response produced by 1 kcal glucose (15). In the current study, the DIL for 31 food items were computed.

Eventually the dietary insulin index of overall diet which represents the mean of insulin index value for each food component was measured by dividing DIL by total energy intake according to the following formula:

DII= dietary insulin load / {Σ [energy content of food (Kcal/serving) × frequency of consumption (servings of food/day)}

Statistical analysis:

Statistical analyses were conducted using SPSS version 19.0 (SPSS Inc., Chicago, IL, USA). For determination of differences in continuous variables, including dietary intakes among quartiles of DGL, DGI, DIL and DII, one-way analysis of variance (ANOVA) was used. Each of the DIL, DII and DGL, DGI were categorized into quartiles based on the distribution among controls. We used binary logistic regression to estimate odds ratios and 95% confidence intervals for quartiles of DIL, DII and DGL, DGI. Covariates considered included body mass index (continuous), total energy intake (continuous), history of cataract in the family, education, physical activity, gender, the access to equipment, diabetes, and hypertension. Next, the odds ratio was adjusted for these confounding factors. In addition, analyses were stratified by BMI (BMI<25 compared with BMI≥25) to ascertain the impact of obesity on risk of cataract.

Result:

General characteristics of the participants are reported in the previous study (1). Table 1 presents dietary intakes across quintiles of energy-adjusted DGL and DGI. Participants in the highest quartile of DGL had higher intake of grains, dairy, legumes, red meat, fruits, vegetables, and nuts in comparison with those in the lowest quartile. Furthermore, grains intake of those in the highest quartile of DGI was more than those in the lowest quartile. Dietary intakes across quintiles of energy-adjusted DIL and DII are shown Table 2. A statistically significant difference in dietary intake of grains, dairy, legumes; red meat, fruits, vegetables and nuts was found among quartiles of DIL. Participants in the highest quartile of DII consumed more grains compared with those in the lowest quartile. Multivariable-adjusted odds ratios (ORs) and 95% CIs for cataract, across quartiles of DGL, DGI, DIL and DII, are shown in table 3. After adjusting for confounders, individuals in the highest quartile of DGL (OR=6.17; 95% CI=1.93-19.37), DGI (OR=6.56; 95% CI=2.67-16.06) and DIL (OR=4.17; 95% CI=1.41-12.27) had a significantly higher risk of cataract, compared to those in the lowest quartile of intake. Table 4 details the multivariable-adjusted odds ratios (95% CI) for cataract by quintiles of DGL, DGI, DIL and DII stratified by BMI (<25 and ≥ 25 Kg/m^2). There was no significant relationship between DGL, DGI, DIL and DII and risk of cataract in individuals with BMI<25. A positive association was found between the highest quartile of DGL (OR=3.45; 95% CI=0.96-12.37), DGI(OR=6.76; 95% CI=2.49-18.38), DIL (OR=1.94; 95% CI=0.58-6.87) and risk of cataract in individuals with elevated BMI (BMI$\geq$25); moreover, this association remained significant after controlling for potential confounders (age, energy, the history of cataract in the family, education, physical activity, gender, the access to equipment, BMI, gender, diabetes, and hypertension).

Discussion

There is currently a paucity of empirical data regarding the relationship between DGI, DGL, DII and DIL and the risk of age-related cataract. Thus, the association between cataract progression and the mentioned dietary indexes is a matter of great scientific interest. Indeed, age related cataracts are a large contributor to poor vision and blindness, whilst diet is a modifiable risk factor which may offer a non-surgical and cost-effective opportunity to delay the onset or prevent this condition (1-3). However, because of the equivocal nature of what little evidence currently exists, we sought to investigate the relationship between the dietary carbohydrate indexes, such as DGI, DGL, DII, DIL, with the odds of being diagnosed with cataracts in accord with the aforementioned aim, the current study found that poorer dietary quality through the higher dietary indexes of DGI, DGL, DIL, in a non-diabetic population, increased the risk of age-related cataracts.

The highest quartile of DGI (OR=6.56; 95% CI=2.67-16.06; P<0.001) was significantly, positively associated with the risk of age-related cataracts, as compared to those in the lowest quartile, in an Iranian population. In a prospective study, an increased risk of nuclear cataracts was evident in those with higher DGI, however, a consistent dose-response relationship was not present in an Australian population (36). Previously, a positive association of DGI and cataract risk was reported in a meta-analysis of seven studies (6). However, a caveat to this meta-analysis is that data were limited to the United States, Australia, and China; therefore, the conclusions should be interpreted with caution for other ethnic populations. Thus, is the present study builds upon the existing literature, and provides a novel addition, by confirming a key dietary index (DGI) is positively associated with cataract risk in an Iranian population (6).

Contrasting the positive association finding of the current study, Turati and colleagues found no association between DGI and cataract risk in a case control study with an Italian population (23). Differences in populations and dietary cultural practices may explain the difference in findings.

The current study found a positive association between higher DGL (OR=6.17; 95% CI=1.93-19.37; P= 0.002) and cataract risk, which is consistent with previous research (8, 23). However, this finding contradicts previous epidemiological studies, where relationships between other dietary indexes and cataract extractions, but not DGL (8, 37, 38). However, the difference in findings may be due to the variation in diagnoses of cataracts, cataract extractions, and varying severities and time of removal. All previous studies in the field of DGL and DGI and cataract were conducted in developed countries, underpinning the inconsistencies between the current and previous studies. Indeed, some studies have reported that dietary patterns have changed among the developing countries, from a healthy, low carbohydrate, low fat, and high fiber diet, into increasing consumption of calorie-dense foods, including refined carbohydrates, fats, meats, and low fiber, which has been attributed to increased food availability (39, 40). Such changes to dietary patterns have corresponded to a rise of obesity, metabolic syndrome, and diabetes mellitus, which are independent key factors of age related cataract (9, 41).

The dietary index, DII, was not associated with the risk of age-related cataract, which may be explained by the notion that consumption of whole grains can reduce the opacity of the lens (1); indeed, we found that participants in the highest quartile of DII consumed more grains compared with those in the lowest quartile. The polyol pathway has been found to be key in cataractogenous in diabetic patients, and this mechanism may still be relevant in the non-diabetic population (42). The enzyme, aldose reductase (AR), catalyzes the reduction of glucose to sorbitol through the polyol pathway (43). The accumulation of sorbitol results in osmotic changes and an intracellular increase of fluid, leading to lens swelling, hydropic lens fibers degeneration, and the formation of cataracts (44, 45). Higher AR levels has been found to increase cataract risk (46), and AR inhibitors have been effective in reducing cataract risk in diabetic patients through preventing the

accumulation of sorbitol (47). Notwithstanding, despite the biological plausibility, the literature on dietary indices and their association with age related cataracts is still emerging in the non-diabetic population. The same biological pathways may be responsible for the finding of an increased risk of cataracts in non-diabetic patients with higher dietary indexes of DGI, DGL, DIL, but clearly warrants further investigation.

The present study found a significant positive association between the highest quartile of DGI (OR=6.76; 95% CI=2.49-18.38; P<0.001) and DGL (OR=3.45; 95% CI=0.96-12.37; P=0.05) with risk of cataract in individuals with elevated BMI (BMI$\geq$25). This finding is supported by a meta-analysis by Ye and colleagues (48), who suggest that elevated leptin, inflammation, and obesity complications may explain this finding. The underlying biological pathways which lead to obesity-related complications of diabetes, hyperlipidemia and hypertension (49, 50), all risk factors for cataracts, may promote cataracts in populations with high BMI, even in the absence of these conditions. Furthermore, elevated leptin, which has been linked to lens opacity and inflammation cytokines and C-reactive proteins, may promote cataract development (37, 50).

Although this study presents a novel addition to the literature, there are some limitations that must be considered. Firstly, the findings of this study are likely not generalizable to populations in other countries, highlighting the need for more research into different ethnic groups. Further research with longitudinal studies is also warranted, so that the association between dietary indexes and different cataract types may be discerned. In addition, studies which examine the age of onset and severity of cataracts and dietary factors are also needed.

Based on the findings of this study, it appears that dietary glycemic load can be reduced by the addition of whole grains, increasing fruit and vegetable intake, as well as reducing simple sugar

intake, to reduce cataract incidence. These changes may be beneficial in reducing the risk of cataract, by reducing dietary glycemic load and insulin load.

Conclusion

We found a significant, direct, relationship between DGI, DGL and DIL with risk of cataract. However, the association between DII and the risk of cataract was not significant, even after adjusting for related confounders. This study demonstrates that improving dietary quality may offer a cost-effective solution to reducing cataract risk. Improving diet quality appears to be a key modifiable risk factor, which could contribute to reducing incidenc3 of blindness and several visual disorders.

Refrences

1. Ghanavati M, Behrooz M, Rashidkhani B, Ashtray-Larky D, Zameni SD, Alipour M. Healthy Eating Index in Patients With Cataract: A Case-Control Study. Iranian Red Crescent medical journal. 2015;17(10):e22490-e.

2. Kaur A, Gupta V, Christopher AF, Malik MA, Bansal P. Nutraceuticals in prevention of cataract–An evidence based approach. Saudi Journal of Ophthalmology. 2017;31(1):30-7.

3. Chiu C-J, Taylor A. Nutritional antioxidants and age-related cataract and maculopathy. Experimental eye research. 2007;84(2):229-45.

4. Sedaghat F, Ghanavati M, Hajian PN, Hajishirazi S, Ehteshami M, Rashidkhani B. Nutrient patterns and risk of cataract: a case-control study. International journal of ophthalmology. 2017;10(4):586.

5. Ma Y, Gao W, Wu K, Bao Y. Flavonoid intake and the risk of age-related cataract in China's Heilongjiang Province. Food & nutrition research. 2015;59(1):29564.

6. Wu H, Zhang H, Li P, Gao T, Lin J, Yang J, et al. Association between dietary carbohydrate intake and dietary glycemic index and risk of age-related cataract: a meta-analysis. Investigative ophthalmology & visual science. 2014;55(6):3660-8.

7. Wu C, Han X, Yan X, Keel S, Shang X, Zhang L, et al. Impact of Diet on the Incidence of Cataract Surgery among Diabetic Patients: Findings from the 45 and Up Study. Current eye research. 2019;44(4):385-92.

8. Tan J, Wang JJ, Flood V, Kaushik S, Barclay A, Brand-Miller J, et al. Carbohydrate nutrition, glycemic index, and the 10-y incidence of cataract. The American journal of clinical nutrition. 2007;86(5):1502-8.

9. Theodoropoulou S, Samoli E, Theodossiadis PG, Papathanassiou M, Lagiou A, Lagiou P, et al. Diet and cataract: a case–control study. International ophthalmology. 2014;34(1):59-68.

10. Yari Z, Behrouz V, Zand H, Pourvali K. New Insight into Diabetes Management: from Glycemic Index to Dietary Insulin Index. Current diabetes reviews. 2019.

11. Anjom-Shoae J, Keshteli AH, Sadeghi O, Pouraram H, Afshar H, Esmaillzadeh A, et al. Association between dietary insulin index and load with obesity in adults. European journal of nutrition. 2019:1-13.

12. Azizi F. Dietary glycemic index, glycemic load, and cardiovascular disease risk factors: Tehran Lipid and Glucose Study. Archives of Iranian medicine. 2013;16(7):401.

13. Anderson C, Milne GL, Park Y-MM, Sandler DP, Nichols HB. Dietary Glycemic Index and Glycemic Load Are Positively Associated with Oxidative Stress among Premenopausal Women. The Journal of nutrition. 2017;148(1):125-30.

14. Nuttall FQ, Gannon MC. Plasma glucose and insulin response to macronutrients in nondiabetic and NIDDM subjects. Diabetes Care. 1991;14(9):824-38.

15. Bao Y, Nimptsch K, Wolpin BM, Michaud DS, Brand-Miller JC, Willett WC, et al. Dietary insulin load, dietary insulin index, and risk of pancreatic cancer–. The American journal of clinical nutrition. 2011;94(3):862-8.

16. Mvitu M, Longo-Mbenza B, Tulomba D, Nge A. Regular, high, and moderate intake of vegetables rich in antioxidants may reduce cataract risk in Central African type 2 diabetics. International journal of general medicine. 2012;5:489.

17. Christen WG, Liu S, Schaumberg DA, Buring JE. Fruit and vegetable intake and the risk of cataract in women. The American journal of clinical nutrition. 2005;81(6):1417-22.

18. Pastor-Valero M. Fruit and vegetable intake and vitamins C and E are associated with a reduced prevalence of cataract in a Spanish Mediterranean population. BMC ophthalmology. 2013;13(1):52.

19. Christen WG, Liu S, Glynn RJ, Gaziano JM, Buring JE. Dietary carotenoids, vitamins C and E, and risk of cataract in women: a prospective study. Archives of Ophthalmology. 2008;126(1):102-9.

20. Cooper AJ, Forouhi NG, Ye Za, Buijsse B, Arriola L, Balkau B, et al. Fruit and vegetable intake and type 2 diabetes: EPIC-InterAct prospective study and meta-analysis. European journal of clinical nutrition. 2012;66(10):1082.

21. Taylor A. Mechanistically linking age-related diseases and dietary carbohydrate via autophagy and the ubiquitin proteolytic systems. Autophagy. 2012;8(9):1404-6.

22. Bejarano E, Taylor A. Too sweet: problems of protein glycation in the eye. Experimental eye research. 2019;178:255-62.

23. Turati F, Filomeno M, Galeone C, Serraino D, Bidoli E, La Vecchia C. Dietary glycemic index, glycemic load and risk of age-related cataract extraction: a case–control study in Italy. European journal of nutrition. 2015;54(3):475-81.

24. Taylor H, Lee J, Wang F, Munoz B. A comparison of two photographic systems for grading cataract. Investigative ophthalmology & visual science. 1991;32(3):529-32.

25. Organization WH. The use and interpretation of anthropometry: report of a WHO expert committee. World Health Organ Tech Rep Ser. 1995;854:312-409.

26. Mirmiran P, Esfahani FH, Mehrabi Y, Hedayati M, Azizi F. Reliability and relative validity of an FFQ for nutrients in the Tehran lipid and glucose study. Public health nutrition. 2010;13(5):654-62.

27. Esfahani FH, Asghari G, Mirmiran P, Azizi F. Reproducibility and relative validity of food group intake in a food frequency questionnaire developed for the Tehran Lipid and Glucose Study. Journal of epidemiology. 2010;20(2):150-8.

28. Ghafarpour M, Houshiar-Rad A, Kianfar H. The manual for household measures, cooking yields factors and edible portion of food. Tehran: Keshavarzi Press; 1999.

29. Brinson MM, Eckles SD. US Department of Agriculture conservation program and practice effects on wetland ecosystem services: a synthesis. Ecological Applications. 2011;21(sp1):S116-S27.

30. Azar M, Sarkisian E. Food composition table of Iran. Tehran: National Nutrition and Food Research Institute, Shaheed Beheshti University. 1980;65.

31. Foster-Powell K, Holt SH, Brand-Miller JC. International table of glycemic index and glycemic load values: 2002. The American journal of clinical nutrition. 2002;76(1):5-56.

32. The University of Sydney glycemic index and GI database.

[Internet]. 2005. Available from: http://wwwglycemicindexcom.

33. Taleban F, Esmaeili M. Glycemic index of Iranian foods. National Nutrition and Food Technology Research Institute publication. 1999.

34. Holt S, Miller J, Petocz P. An insulin index of foods: the insulin demand generated by 1000-kJ portions of common foods. The American journal of clinical nutrition. 1997;66(5):1264-76.

35. De Jong V, Holt S, Brand-Miller J, editors. Insulin scores for single foods and their application to mixed meals. PROCEEDINGS-NUTRITION SOCIETY OF AUSTRALIA; 2000: Nutrition Society of Australia; 1998.

36. Chiu C-J, Robman L, McCarty CA, Mukesh BN, Hodge A, Taylor HR, et al. Dietary carbohydrate in relation to cortical and nuclear lens opacities in the Melbourne Visual Impairment Project. Investigative ophthalmology & visual science. 2010;51(6):2897-905.

37. Schaumberg DA, Liu S, Seddon JM, Willett WC, Hankinson SE. Dietary glycemic load and risk of age-related cataract. The American journal of clinical nutrition. 2004;80(2):489-95.

38. Klein BE, Klein R, Lee KE. Diabetes, cardiovascular disease, selected cardiovascular disease risk factors, and the 5-year incidence of age-related cataract and progression of lens opacities: the Beaver Dam Eye Study. American journal of ophthalmology. 1998;126(6):782-90.

39. Misra A, Singhal N, Khurana L. Obesity, the metabolic syndrome, and type 2 diabetes in developing countries: role of dietary fats and oils. Journal of the American College of Nutrition. 2010;29(3 Suppl):289s-301s.

40. Lipoeto NI, Geok Lin K, Angeles-Agdeppa I. Food consumption patterns and nutrition transition in South-East Asia. Public health nutrition. 2013;16(9):1637-43.

41. Rowe NG, Mitchell PG, Cumming RG, Wans JJ. Diabetes, fasting blood glucose and age-related cataract: the Blue Mountains Eye Study. Ophthalmic epidemiology. 2000;7(2):103-14.

42. Pollreisz A, Schmidt-Erfurth U. Diabetic cataract—pathogenesis, epidemiology and treatment. Journal of ophthalmology. 2010;2010.

43. Kinoshita JH, Fukushi S, Kador P, Merola LO. Aldose reductase in diabetic complications of the eye. Metabolism. 1979;28(4):462-9.

44. Harding JJ, Egerton M, Van Heyningen R, Harding R. Diabetes, glaucoma, sex, and cataract: analysis of combined data from two case control studies. British Journal of Ophthalmology. 1993;77(1):2-6.

45. Kador PF, Kinoshita JH, editors. Diabetic and galactosaemic cataracts. Ciba Found Symp; 1984: Wiley Online Library.

46. Oishi N, Morikubo S, Takamura Y, Kubo E, Tsuzuki S, Tanimoto T, et al. Correlation between adult diabetic cataracts and red blood cell aldose reductase levels. Investigative ophthalmology & visual science. 2006;47(5):2061-4.

47. Singh RP. Managing Diabetic Eye Disease in Clinical Practice: Springer; 2015.

48. Ye J, Lou L-X, He J-J, Xu Y-F. Body mass index and risk of age-related cataract: a meta-analysis of prospective cohort studies. PloS one. 2014;9(2):e89923.

49. Radzevičienė L, Ostrauskas R. Body mass index, waist circumference, waist–hip ratio, waist–height ratio and risk for type 2 diabetes in women: A case–control study. Public health. 2013;127(3):241-6.

50. Muoio DM, Newgard CB. Obesity-related derangements in metabolic regulation. Annu Rev Biochem. 2006;75:367-401.