INTRODUCTION

Age-related cataract is the leading cause of blindness worldwide (1). Measures to delay or prevent the onset of cataract could have major economic and health effects. Hyperglycemia is thought to be a risk factor for cataract development, with considerable evidence linking diabetes or aberrant glucose metabolism to an increased cataract risk (2–6), particularly cortical cataract (4, 6, 7). Both animal and human studies have also shown that higher plasma glucose concentrations increase the risk of cataract (8, 9).

High carbohydrate intake has adverse effects on glucose metabolism (10). In addition, different carbohydrates can produce particular plasma glucose responses, as measured by the glycemic index (GI), which is a measure of carbohydrate quality. Glycemic load (GL), the mathematical product of a food’s GI and total available carbohydrate content, represents both the quantity and the quality of carbohydrates (11, 12). High GI and GL diets were also shown to have adverse metabolic effects (11, 13), whereas low GI diets were observed to reduce glycosylated hemoglobin concentrations in both persons with and without diabetes (14, 15). Given the potential role of hyperglycemia in cataractogenesis, higher carbohydrate intake or higher GI or GL diets could increase the risk of cataract development. A role for dietary carbohydrates in cataractogenesis was shown in animal models (16).

However, few epidemiologic studies have examined links between carbohydrate nutrition and cataract, particularly incident cataract (12, 17–20). We aimed in this report to assess the relation between carbohydrate nutrition measured at baseline, including dietary GI and GL, and the 10-y incidence of cataract in a large population-based study, the Blue Mountains Eye Study.

SUBJECTS AND METHODS

Study subjects

The Blue Mountains Eye Study is a population-based cohort study of vision, common eye diseases, and other health outcomes in an urban older Australian population. Study methods and procedures have been described (21). Briefly, baseline eye examinations of 3654 residents aged 49+ y were conducted (82.4% participation) during 1992–1994. Of these baseline participants,

ABSTRACT

Background: Although dietary carbohydrates are thought to play a role in cataractogenesis, few epidemiologic studies have examined links between carbohydrate nutrition and cataract.

Objectives: We investigated the associations between dietary glycemic index (GI), glycemic load (GL), total carbohydrate intake, and 10-y incident nuclear, cortical, and posterior subcapsular cataract.

Design: Of 3654 baseline participants in an Australian population aged ≥49 y (1992–1994), 933 were seen after 5 and/or 10 y, had completed a detailed semiquantitative food-frequency questionnaire, had no previous cataract surgery or baseline cataract, and had photographs taken to assess incident cataract with the Wisconsin Cataract Grading System. Dietary information was collected with a validated food questionnaire. GI was calculated from a customized database of Australian foods. GI, GL, and all other nutrients were energy adjusted. Hazard ratios (HRs) and 95% CIs were calculated with the use of discrete logistic models.

Results: After age, sex, diabetes, and other factors were controlled for, each SD increase in GI significantly predicted incident cortical cataract (HR: 1.19; 95% CI: 1.01, 1.39). Participants within the highest compared with the lowest quartile of GI were more likely to develop incident cortical cataract (HR: 1.77; 95% CI: 1.13, 2.78; P for trend = 0.035). These findings were similar after excluding participants with diabetes, although they were slightly attenuated and marginally nonsignificant (HR: 1.16; 95% CI: 0.98, 1.37, per SD increase in GI). No association was found between GI and nuclear or posterior subcapsular cataract and between GL or carbohydrate quantity and any cataract subtype.


KEY WORDS Cataract, cohort study, incidence, population, risk factors, Blue Mountains Eye Study, lens, nutrition, carbohydrate, glycemic index, glycemic load, glycation, humans, epidemiology, aging

INTRODUCTION

Age-related cataract is the leading cause of blindness worldwide (1). Measures to delay or prevent the onset of cataract could have major economic and health effects. Hyperglycemia is thought to be a risk factor for cataract development, with considerable evidence linking diabetes or aberrant glucose metabolism to an increased cataract risk (2–6), particularly cortical cataract (4, 6, 7). Both animal and human studies have also shown that higher plasma glucose concentrations increase the risk of cataract (8, 9).

High carbohydrate intake has adverse effects on glucose metabolism (10). In addition, different carbohydrates can produce particular plasma glucose responses, as measured by the glycemic index (GI), which is a measure of carbohydrate quality. Glycemic load (GL), the mathematical product of a food’s GI and total available carbohydrate content, represents both the quantity and the quality of carbohydrates (11, 12). High GI and GL diets were also shown to have adverse metabolic effects (11, 13), whereas low GI diets were observed to reduce glycosylated hemoglobin concentrations in both persons with and without diabetes (14, 15). Given the potential role of hyperglycemia in cataractogenesis, higher carbohydrate intake or higher GI or GL diets could increase the risk of cataract development. A role for dietary carbohydrates in cataractogenesis was shown in animal models (16).

However, few epidemiologic studies have examined links between carbohydrate nutrition and cataract, particularly incident cataract (12, 17–20). We aimed in this report to assess the relation between carbohydrate nutrition measured at baseline, including dietary GI and GL, and the 10-y incidence of cataract in a large population-based study, the Blue Mountains Eye Study.
2335 (75.1% of survivors) and 1952 (76.6% of survivors) returned to 5-y (1997–1999) and 10-y (2002–2004) follow-up examinations, respectively. Altogether, 2564 participants were followed at least once since the baseline examinations. Mean follow-up intervals were 5.1 y and 10.5 y, respectively, for the 5- and 10-y follow-up examinations.

Of the 2564 participants followed up at least once, we excluded participants at baseline who had cataract surgery performed (n = 100), with any type of cataract (n = 748), those with missing or ungradable photographs (n = 972), or those who did not complete or have a usable food-frequency questionnaire (FFQ) (n = 391). This gave 933 participants who were at risk of incident cataract. After further excluding participants with missing or ungradable photographs at the follow-up examinations to assess incident cases of each cataract subtype, there were 925, 920, and 923 participants at risk of developing cortical, nuclear, and posterior subcapsular cataract, respectively. These participants were used for our primary analyses. Most of the missing or ungradable photographs at baseline were caused by a random technical malfunction of the Topcon camera used to take nuclear cataract photographs; a problem ultimately repaired. As previously described (22), no significant differences were observed between the participants with and without Topcon photographs.

In supplementary analyses, we considered persons with one cataract subtype at baseline were at risk of developing the other 2 cataract subtypes during the 10-y follow-up. For example, participants with nuclear cataract at baseline were still considered at risk of incident cortical or posterior subcapsular cataract. For these analyses, excluding those with nuclear cataract at baseline gave 1094 participants at risk of developing nuclear cataract. Corresponding figures for cortical and posterior subcapsular cataract were 1535 and 1724, respectively.

This study followed recommendations of the Declaration of Helsinki and was approved by the Sydney West Area Health Service Human Research Ethics Committee. Written, informed consent was obtained from all participants.

Dietary assessment

Participants completed a 145-item, semiquantitative FFQ for Australian diet and vernacular modified from an early FFQ by Willett et al (23). This FFQ included questions about the types of breakfast cereals consumed, which were used to increase the accuracy of the GI and GL calculations. The FFQ was attempted and returned by 3267 participants at baseline (89.4%), with 2900 (88.9% of those who attempted the FFQ, 79.4% of those who attended examination) having usable FFQs. Characteristics of the FFQ respondents and exclusion criteria have been reported (20, 24, 25). Subjects were excluded if >12 FFQ questions or an entire page remained blank or if daily energy intakes were <2500 kJ or >18 000 kJ. The FFQ was found to be reliable in the population and to have reasonable concurrent validity compared with weighed food records collected during 1 y (energy-adjusted Spearman correlations > 0.5 for most nutrients). In particular, the FFQ showed reasonable agreement for carbohydrates, yielding a correlation coefficient of 0.57, and correctly classifying nearly 80% of participants within one quintile for carbohydrate intake (25).

A dietitian coded data from the FFQ into a customized database with the use of DBASE IV (Borland International, Scotts Valley, CA). This incorporated the Australian Tables of Food Composition 1990 (NUTTAB 90) (26) and published GI values with the scale of glucose = 100 (27). Additional GI data were obtained from the Sydney University Glycemic Index Research Service online database (www.glycemicindex.com). In total, 88.9% of GI values were obtained from published values, and 11.1% were interpolated from similar food items.

An overall dietary GI value for each participant was calculated by summing the weighted GI of individual foods, with the weighting proportional to the contribution of individual foods to total carbohydrate intake. The GL of each food was calculated by multiplying its GI by the amount of available carbohydrate (in g) per serving. The multiplication of each food’s GL by frequency of consumption, summed for all food items, gave the overall dietary GL.

Cataract grading and definition

At each examination, participants underwent a comprehensive eye examination. Cataract was documented from slit-lamp lens photographs (Topcon SL-7e camera; Topcon Optical, Tokyo, Japan) for nuclear cataract and from retroillumination lens photographs (Neitz CT-R cataract camera; Neitz Instruments, Tokyo, Japan) for cortical and posterior subcapsular cataract. Photographic grading closely followed the Wisconsin Cataract Grading System (28), as previously reported (21, 22), with good agreement found for assessments of both inter- and intragrader reliability (22). History of past cataract surgery was confirmed at both the examination and photographic grading. The presence of nuclear, cortical, and posterior subcapsular cataract was assessed for each eye. Presence and severity of nuclear cataract were defined on a 5-level scale by comparison with a set of 4 standard slit-lamp photographs; level 4 or 5 defined the presence of nuclear cataract. The extent of cortical or posterior subcapsular cataract was determined by estimating the lens area involved in segments of a grid overlying the photographs (28). Cortical opacity involving ≥5% of the total lens area or the presence of any posterior subcapsular opacity was used to define the presence of these 2 cataract types.

In primary analyses, incident cataract was defined as the first appearance at follow-up in either eye of each specific cataract type (nuclear, cortical, or posterior subcapsular) in bilaterally phakic participants without any cataract at baseline. In supplementary analyses, we considered participants with 1 cataract subtype at baseline to be at risk of developing the other 2 cataract subtypes. We then defined incident cataract as the first appearance at follow-up in either eye of each specific cataract type (nuclear, cortical, or posterior subcapsular) in bilaterally phakic participants without the corresponding type of cataract in either eye at baseline.

Assessment of confounders

Information on potential confounders was collected at baseline. A standardized interviewer-administered questionnaire was used to ascertain demographic information; family history; medications taken, including specific questions on use of oral or inhaled steroids; medical history, including self-reported diagnoses of diabetes, hypertension, acute myocardial infarction, angina, or stroke; and smoking and alcohol history. History of cardiovascular disease (stroke, angina, and myocardial infarction) was combined and termed any cardiovascular disease.

Weight, height, and blood pressure measurements were taken by a trained examiner. Fasting blood specimens were drawn for...
clinical biochemistry assessment. Body mass index (BMI; in kg/m²) was calculated. Obesity was defined as BMI ≥ 30. Diabetes was diagnosed from either medical history or fasting blood glucose ≥ 7.0 mmol/L. Of participants included in this study, 120 had diabetes at baseline, 6 of whom had type 1 diabetes. Hypertension was defined either in persons taking antihypertensive medications or in those found to have systolic blood pressure ≥ 160 mm Hg or diastolic blood pressure ≥ 95 mm Hg at the baseline examination. Sun-related skin damage was estimated on a 4-point scale (none, mild, moderate, and severe) by assessing the arms, hands, and face. Iris color was assessed by a clinical examiner as blue, hazel-green, tan-brown, or dark brown, by comparison with standard photographs taken before pupil dilation. The baseline refractive state was defined as the spherical equivalent refraction, calculated by algebraic addition of the best-corrected spherical refraction and half the cylindrical refraction. Myopia was defined as spherical equivalent refraction < −1.0 diopter. Higher educational achievement was defined as attainment of qualifications (certificate, diploma, or degree) after leaving school. Participants were also asked to self-rate their general health as excellent, good, fair, or poor (self-ranked health).

Statistical methods

SAS (version 9; SAS, Cary, NC) was used for all analyses. We examined the association of baseline dietary GI, GL, and carbohydrate intake (independent variables) with the 10-y incidence of nuclear, cortical, or posterior subcapsular cataract (outcome variables). Multivariate adjusted hazard ratios (HRs) and 95% CIs were calculated with the use of discrete linear logistic models (29). Discrete linear logistic models were implemented in SAS version 9.0 software with the use of the PHREG procedure with the ties = discrete option (30). Participants with incident cataract at the 5-y follow-up examination, or those who attended the 5-y but not the 10-y follow-up examination (n = 218), were censored at 5 y. Potential confounders considered included age (in y); sex (male, female); smoking (never, past, current); heavy alcohol consumption (≥4 drinks/d, <4 drinks/d); fasting white cell count (in 10⁹ cells/L); fibrinogen concentration (in g/L); platelet count (in 10⁹ cells/L); diabetes; BMI; obesity; dark brown iris color; myopia; skin damage; ever or current use of oral or inhaled steroids; higher educational achievement; self-ranked health; history of angina, acute myocardial infarction, stroke, or any cardiovascular disease; and hypertension (present, not present). Because different cataract types may have different risk factors, the final multivariate models varied by cataract type and retained factors that were significantly associated with each outcome.

Dietary intakes, GI, and GL were energy adjusted with the residual method described by Willett and Stampfer (31). Carbohydrate intake, dietary GI, and dietary GL were analyzed as continuous variables (per SD) and as quartiles. The lowest quartile was the referent group. We calculated the trend in relative risk across quartiles by modeling an ordinal variable corresponding to the median value for each quartile. We conducted analyses, initially controlling for and then excluding participants with diabetes. For the carbohydrate variables with a significant main effect, we additionally examined the effect modifications of age, sex, smoking, diabetes, BMI, and obesity by including product terms in the multivariable models.

RESULTS

Of the 2564 participants followed at least once, subjects without usable FFQ data (n = 391) tended to be older (66.3 y compared with 63.9 y), women (16.4% of women did not complete the FFQ compared with 13.6% of men), and less likely to have been past smokers (28.9% compared with 36.8%) or to have attained formal qualifications after leaving high school (42.2% compared with 51.6%). The prevalence of cortical, nuclear, and posterior subcapsular cataract at baseline was similar in subjects with and without FFQ data. The baseline characteristics according to quartiles of energy-adjusted dietary GI and GL for participants considered in our primary analyses (n = 933) are shown in Table 1. Dietary GI varied at baseline in the study population between an average 51.1 and 62.3 in the lowest and highest quartiles, respectively. Similarly, dietary GL varied between 103 and 161. Participants with the highest compared with the lowest GI quartile generally consumed less sugar, protein, fiber from cereals, fruit, or vegetables and more starch and alcohol. They were also more likely to be men, to have been smokers, and to have had hypertension. Participants with the highest compared with the lowest GLs generally consumed less alcohol, fat, and protein and more total carbohydrate, sugar, starch, breads and cereals, and fiber from cereals or fruit. They were also more likely to have had a history of cardiovascular disease and were less likely to have been smokers. GL and carbohydrate intake were highly correlated (correlation coefficient = 0.89, P < 0.0001). Trends in baseline characteristics across quartiles of carbohydrate intake were similar to those of GL. Baseline carbohydrate intake varied between an average 182 g and 280 g in the lowest and highest quartiles, respectively.

The association between dietary GI and incident cortical, nuclear, and posterior subcapsular cataract is shown in Table 2. After adjusting for age, sex, diabetes, cardiovascular disease, and obesity, participants in the top quartile of GI were significantly more likely to develop cortical cataract than participants in the lowest quartile. Each SD increase in GI was also significantly linked to cortical cataract incidence (HR: 1.19; 95% CI: 1.01, 1.39). These findings were similar after excluding participants with diabetes, although the association between each SD increase in GI and cortical cataract incidence was attenuated and became marginally nonsignificant (HR: 1.16; 95% CI: 0.98, 1.37). Further adjustment for fiber from bread and cereals, vegetables or fruit, dietary alcohol, antioxidants, folate, protein, fat, carbohydrate, and smoking showed similar results (data not shown). No significant interactions were observed between dietary GI and age, sex, smoking, diabetes, BMI, or obesity for cortical cataract. In addition, no significant associations were observed between GI and nuclear or posterior subcapsular cataract (Table 2) or between GL and carbohydrate intake and any cataract subtype (Table 3).

In supplementary analyses, when we considered that participants without cortical cataract but with nuclear or posterior subcapsular cataract at baseline were also at risk of developing cortical cataract, the association between the top quartile of GI and cortical cataract incidence, compared with the rest of the population, remained but was attenuated (HR: 1.31; 95% CI: 0.99, 1.72; P = 0.057). However, in supplementary analyses, we still found no associations between GI and the incidence of the other 2 cataract types. No significant associations were observed
between GL or total carbohydrate intake and the incidence of any cataract type (data not shown).

**DISCUSSION**

In this study, participants (with or without diabetes) in the top quartile of dietary GI had an increased risk of long-term incident cortical cataract than did participants in the lowest quartile. We found no association between GI and the incidence of either nuclear or posterior subcapsular cataract or between GL or total carbohydrate intake and any incident cataract subtype.

A role for carbohydrate nutrition in cataractogenesis is biologically plausible. Diabetes is an established risk factor for cataract (2, 3, 5, 6), and the level of hyperglycemia, as measured by glycosylated hemoglobin, was correlated with mean lens opacity (9) or an increased risk of nuclear and cortical cataract among persons with diabetes (3). Glucose is taken up relatively slowly from plasma into the aqueous humor, and then it passes freely into the lens, where it is slowly turned over (18). Prolonged exposure of the lens proteins to elevated glucose was observed to cause accumulation of polyol and glycation, which may then produce oxidation, cross-linking, aggregation, and precipitation of lens proteins (32–35). Because peak blood glucose concentrations and average blood glucose concentrations (as assessed by area under the curve) are higher after high GI foods (11), it is conceivable that higher GI foods could produce more damage to lens tissue than do lower GI foods. A potential role for oxidative damage in the association between high GI foods and cataract is further supported by studies showing that higher postprandial glucose concentrations result in greater depression of serum antioxidants (11, 36). Some evidence suggests a protective association between higher nutritional intake and serum concentrations of antioxidants and age-related cataract (37, 38). A model involving inflammation could also explain the observed association between carbohydrate nutrition and cataract. Higher GL was linked with higher plasma concentrations of the inflammatory marker C-reactive protein (39), and both inflammation and higher C-reactive protein concentrations have been linked to the development of cataract (40).

Studies also suggest that higher plasma glucose concentrations may have a greater effect on the lens cortex than on the nucleus (18). Nuclear magnetic resonance studies show that glucose concentrations remain higher in the cortex than in the nucleus (18, 42). Insolubilization of glycated lens proteins was observed to be initiated in the cortical

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**TABLE 1**

Baseline characteristics according to quartile (Q) of energy-adjusted dietary glycemic index (GI) and glycemic load (GL) in the Blue Mountains Eye Study

<table>
<thead>
<tr>
<th>Quartile score</th>
<th>Q1</th>
<th>Q2</th>
<th>Q3</th>
<th>Q4</th>
</tr>
</thead>
<tbody>
<tr>
<td>GI</td>
<td>51.1 ± 2.4</td>
<td>55.1 ± 0.7</td>
<td>57.7 ± 0.8</td>
<td>62.3 ± 2.9</td>
</tr>
<tr>
<td>GL</td>
<td>103 ± 14</td>
<td>125 ± 5</td>
<td>139 ± 4</td>
<td>161 ± 14</td>
</tr>
</tbody>
</table>

**Daily intake**

<table>
<thead>
<tr>
<th>Total energy (kJ)</th>
<th>8284 ± 2350</th>
<th>8654 ± 2661</th>
<th>8816 ± 2388</th>
<th>8800 ± 2817</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbohydrate (g)</td>
<td>234 ± 41</td>
<td>233 ± 37</td>
<td>237 ± 38</td>
<td>226 ± 42</td>
</tr>
<tr>
<td>Sugar (g)</td>
<td>135 ± 31</td>
<td>124 ± 28</td>
<td>122 ± 30</td>
<td>107 ± 38</td>
</tr>
<tr>
<td>Starch (g)</td>
<td>94 ± 23</td>
<td>106 ± 21</td>
<td>113 ± 24</td>
<td>118 ± 25</td>
</tr>
<tr>
<td>Fiber</td>
<td>8.1 ± 5.1</td>
<td>8.5 ± 5.8</td>
<td>7.4 ± 4.6</td>
<td>5.6 ± 4.0</td>
</tr>
<tr>
<td>Fruit</td>
<td>9.9 ± 6.4</td>
<td>7.2 ± 4.0</td>
<td>6.2 ± 3.7</td>
<td>3.8 ± 2.9</td>
</tr>
<tr>
<td>Vegetables (g)</td>
<td>10.7 ± 4.3</td>
<td>10.2 ± 4.0</td>
<td>9.5 ± 3.4</td>
<td>9.6 ± 4.8</td>
</tr>
<tr>
<td>Alcohol (g)</td>
<td>8.1 ± 13.6</td>
<td>9.3 ± 14.3</td>
<td>12.4 ± 17.6</td>
<td>19.6 ± 22.4</td>
</tr>
<tr>
<td>Total fat (g)</td>
<td>76 ± 15</td>
<td>77 ± 14</td>
<td>76 ± 14</td>
<td>75 ± 14</td>
</tr>
<tr>
<td>Protein (g)</td>
<td>93 ± 17</td>
<td>91 ± 13</td>
<td>87 ± 13</td>
<td>85 ± 15</td>
</tr>
<tr>
<td>Breads and cereals (g)</td>
<td>215 ± 130</td>
<td>240 ± 130</td>
<td>234 ± 136</td>
<td>211 ± 146</td>
</tr>
</tbody>
</table>

**Women (%)** | 72 | 61 | 48 | 36 | <0.0001 | 56 | 55 | 59 | 46 | 0.085 |

**Hypertension (%)** | 33 | 40 | 43 | 47 | 0.0025 | 37 | 39 | 45 | 41 | 0.22 |

**History of cardiovascular disease (%)** | 11.6 | 18.0 | 14.1 | 12.9 | 0.99 | 8.6 | 12.4 | 18.4 | 17.2 | 0.0019 |

**Diabetes (%)** | 5.1 | 6.4 | 3.4 | 4.3 | 0.37 | 6.9 | 4.3 | 4.3 | 3.9 | 0.15 |

**Obesity (%)** | 19.5 | 22.2 | 17.2 | 18.0 | 0.42 | 19.7 | 20.0 | 20.3 | 16.8 | 0.47 |

**Current smoker (%)** | 9.9 | 10.8 | 12.0 | 21.9 | 0.0002 | 20.2 | 12.5 | 9.0 | 12.9 | 0.011 |

1 For the 933 participants included in the primary analyses (those who attended follow-up at least once, completed a food-frequency questionnaire, did not have cataracts or prior cataract surgery at baseline, and had photographs available for cataract assessment).
2 Mantel-Haenszel chi-square test was used to compute P values for discrete variables; general linear models were used for continuous variables by modeling a variable corresponding to the median value for each quartile.
3 x ± SD (all such values).
4 Energy-adjusted except for total energy intake, breads and cereals, and alcohol.
5 Includes angina, myocardial infarction, or stroke.
### TABLE 2

Associations between baseline energy-adjusted dietary glycemic index (GI) and 10-y incident cataract in the Blue Mountains Eye Study

<table>
<thead>
<tr>
<th>GI</th>
<th>Quartile mean</th>
<th>Outcomes/ at risk</th>
<th>Adjusted HR (95% CI)</th>
<th>Adjusted HR (95% CI)</th>
<th>Adjusted HR (95% CI)</th>
<th>Outcomes/ at risk</th>
<th>Adjusted HR (95% CI)</th>
<th>Adjusted HR (95% CI)</th>
<th>Adjusted HR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Q1</td>
<td>51.1</td>
<td>42/233</td>
<td>1.00</td>
<td>1.00</td>
<td>66/229</td>
<td>1.00</td>
<td>1.00</td>
<td>12/231</td>
<td>1.00</td>
</tr>
<tr>
<td>Q2</td>
<td>55.1</td>
<td>55/233</td>
<td>1.35 (0.87, 2.09)</td>
<td>1.31 (0.84, 2.03)</td>
<td>66/230</td>
<td>0.97 (0.64, 1.45)</td>
<td>0.99 (0.65, 1.49)</td>
<td>15/233</td>
<td>1.19 (0.55, 2.60)</td>
</tr>
<tr>
<td>Q3</td>
<td>57.7</td>
<td>56/231</td>
<td>0.89 (0.55, 1.44)</td>
<td>0.86 (0.53, 1.40)</td>
<td>68/234</td>
<td>0.99 (0.65, 1.49)</td>
<td>1.03 (0.68, 1.56)</td>
<td>18/231</td>
<td>1.58 (0.74, 3.39)</td>
</tr>
<tr>
<td>Q4</td>
<td>62.3</td>
<td>58/228</td>
<td>1.77 (1.13, 2.78)</td>
<td>1.77 (1.13, 2.78)</td>
<td>70/227</td>
<td>1.23 (0.81, 1.88)</td>
<td>1.26 (0.82, 1.95)</td>
<td>11/228</td>
<td>1.01 (0.43, 2.37)</td>
</tr>
</tbody>
</table>

P for trend 0.037 0.035 0.32 0.28 0.86 0.84

1 HR, hazard ratio; Q, quartile.
2 Adjusted for age and sex.
3 Adjusted for age, sex, diabetes, cardiovascular disease, and obesity.
4 Adjusted for age, sex, diabetes, smoking, myopia, skin damage, and ever use of inhaled steroids.
5 Adjusted for age, sex, diabetes, ever use of steroids, and myopia.

### TABLE 3

Associations of baseline energy-adjusted dietary glycemic load (GL) and carbohydrate intake (in g) with 10-y incident cataract in the Blue Mountains Eye Study

<table>
<thead>
<tr>
<th>Dietary variable</th>
<th>Quartile mean</th>
<th>Outcomes/ at risk</th>
<th>Adjusted HR (95% CI)</th>
<th>Adjusted HR (95% CI)</th>
<th>Adjusted HR (95% CI)</th>
<th>Outcomes/ at risk</th>
<th>Adjusted HR (95% CI)</th>
<th>Adjusted HR (95% CI)</th>
<th>Adjusted HR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>GL</td>
<td>Q1</td>
<td>103</td>
<td>41/230</td>
<td>1.00</td>
<td>63/327</td>
<td>1.00</td>
<td>12/330</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Q2</td>
<td>125</td>
<td>51/232</td>
<td>1.17 (0.75, 1.82)</td>
<td>58/227</td>
<td>0.83 (0.54, 1.27)</td>
<td>14/230</td>
<td>1.10 (0.50, 2.43)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Q3</td>
<td>139</td>
<td>62/228</td>
<td>1.40 (0.91, 2.16)</td>
<td>78/229</td>
<td>1.13 (0.76, 1.69)</td>
<td>14/234</td>
<td>1.06 (0.48, 2.33)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Q4</td>
<td>161</td>
<td>37/229</td>
<td>0.88 (0.55, 1.42)</td>
<td>71/232</td>
<td>1.18 (0.79, 1.76)</td>
<td>16/229</td>
<td>1.35 (0.63, 2.92)</td>
<td></td>
</tr>
</tbody>
</table>

P for trend 0.28 0.23 0.28 0.23 0.46 0.45

| Carbohydrate    | Q1            | 182               | 48/230               | 1.00                 | 57/320               | 1.00              | 10/230               | 1.00                 |
|                 | Q2            | 223               | 50/232               | 0.91 (0.59, 1.40)    | 72/320               | 1.09 (0.72, 1.66)  | 14/232               | 1.29 (0.56, 2.97)    |
|                 | Q3            | 245               | 50/232               | 0.88 (0.57, 1.37)    | 75/320               | 1.19 (0.79, 1.80)  | 15/230               | 1.41 (0.62, 3.20)    |
|                 | Q4            | 280               | 43/231               | 0.78 (0.50, 1.22)    | 60/320               | 1.01 (0.66, 1.54)  | 17/231               | 1.64 (0.74, 3.66)    |

P for trend 0.28 0.23 0.28 0.23 0.22 0.21

1 HR, hazard ratio; Q, quartile.
2 Adjusted for age and sex.
3 Adjusted for age, sex, diabetes, cardiovascular disease, and obesity.
4 Adjusted for age, sex, diabetes, smoking, myopia, skin damage, and ever use of inhaled steroids.
5 Adjusted for age, sex, diabetes, ever use of steroids, and myopia.
region in diabetic cataract (33). Epidemiologic studies also support a more consistent link between diabetes and cortical rather than nuclear cataract (3, 4, 6, 7).

Only a few epidemiologic studies have investigated the association between carbohydrate nutrition and cataract, mostly cortical and nuclear cataract, or cataract surgery. The findings to date have been inconsistent, particularly for studies of GI and cataract. Only 3 previous studies have examined this link. The Age-Related Eye Disease Study found that higher GI predicted nuclear, but not cortical, cataract (17). Neither the Nutrition and Vision Project, which examined cortical and nuclear opacities (18), nor the Nurses’ Health Study and Health Professionals Follow-up Study, which examined cataract extractions and nuclear and posterior subcapsular cataract subtypes (12), found an association with GI.

A more consistent link between carbohydrate quantity or GI, which tend to be highly correlated (18), and cortical cataract is emerging in recent data. The Nutrition and Vision Project reported an association between dietary carbohydrate quantity or GI and increased risk of cortical, but not nuclear, opacities (18). In our previous cross-sectional report (20), we found that the risk of cortical cataract was 40% higher for participants in the fifth quintile (>268.2 g) of daily carbohydrate intake than with participants in the first quintile (<172.6 g). However, we did not find any association between carbohydrate quantity or GI and incident cataract in our study sample. Further longitudinal studies are needed to clarify the association between carbohydrate nutrition and cataract.

Confounding by diabetes is a concern in assessing the association between carbohydrate nutrition and cataract. Our findings, however, persisted after excluding participants with diabetes, most of whom had type 2 diabetes. Hence, the observed associations found between GI and cortical cataract are not explained by the presence of diabetes.

Strengths of our study include its long follow-up of a population-based sample, a reasonably good follow-up (~75% of surviving participants were examined), and cataract diagnosis based on detailed grading of lens photographs, shown to have high reproducibility (43). Photographic graders were also masked to other subject characteristics, such as nutritional data. Limitations of our study include the possibility of confounding from unknown confounding factors, the relatively high number of participants with missing FFQ data, which is frequent in older populations, as well as missing nuclear photographs, caused by a random camera error. However, ungradable lens photographs occurred nondifferentially among subjects with and without cataract. As previously reported (22), participants with and without gradable nuclear photographs were similar with respect to smoking history, age, sex, history of diabetes, hypertension, steroid use, and alcohol intake. Although bias from healthy behaviors among participants with low GI or carbohydrate intakes cannot be ruled out, the dietary measures were made at a time before widespread publicity about the potential benefits of low GI diets was common knowledge.

In summary, this appears to be one of few studies to examine the association between carbohydrate nutrition and incident cataract, and it appears to be the first population-based cohort study to examine the link between GI or GL and cataract. In an Australian cohort, we found poorer dietary carbohydrate quality, measured by high GI, predicted incident cortical cataract but not nuclear or posterior subcapsular cataract. Neither carbohydrate quantity nor GL were associated with incident cataract. Because carbohydrate foods represent the main dietary component for humans (17) and cataract entails such a significant health and economic burden, further studies to clarify whether aspects of dietary carbohydrate intake may affect the risk of cataract development are warranted.

The author’s responsibilities were as follows—PM, JJW, VF, and JT: design and conduct of the study; PM, JJW, VF, and AB: collection and management of the data; JT and SK: analysis of the data; PM, JJW, VF, JT, JB-M, AB, and SK: interpretation of the data; JT: preparation of the first draft manuscript; PM, JJW, VF, JBM, AB, SK, and JT: review and approval of the manuscript. None of the authors had a personal or financial conflict of interest.

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