



# The relationships between macronutrient and micronutrient intakes and type 2 diabetes mellitus in South Asians: A review

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## ABSTRACT

**Background:** South Asians (SA) have increased prevalence of type 2 diabetes mellitus (T2DM). The role of nutrient intakes in T2DM in SA is not well understood, however.

**Objective:** The paper reviewed the relationship between macronutrient and micronutrients intakes and T2DM in SA.

**Methods:** The MEDLINE database was searched for relevant papers on this topic in SA.

**Results:** There was some evidence that dietary fiber and linoleic acid intake may reduce but carbohydrates may increase the risk of T2DM. Some studies found higher energy from protein and fat in subjects with T2DM versus controls. Other studies, however, found lower carbohydrate intake among those with T2DM or no relationship between diet composition and T2DM. Several vitamins and minerals were also inversely related to T2DM.

**Conclusions:** The data were limited to a few epidemiological studies. Most studies did not distinguish between undiagnosed and known T2DM. Subjects with known T2DM are more likely to have changed their diet. Prospective cohort or randomized controlled studies examining the role of diet composition, using precise image-assisted dietary assessment method and blood biomarkers, in the development of T2DM among migrant and native SA are needed. Lastly, a more complete nutrient database for foods consumed by SA is needed.

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## 1. Introduction

South Asians (Indians, Pakistanis, Bangladeshis, Nepalese, and Sri Lankans) have a higher prevalence of type 2 diabetes mellitus (T2DM) compared to other populations. This paper reviews the epidemiology of T2DM in South Asians (SA), examines the role of nutrient intakes in T2DM in this population, and identifies gaps in the literature. This review also describes the mechanisms by which intake of various nutrients contributes to the risk for T2DM. In addition, dietary intake assessment methodology is briefly reviewed before the section on the

role of nutrient intakes in T2DM. Lastly, it identifies areas of future research and provides an overall perspective.

The literature search on the relationship between nutrient intakes and T2DM was conducted by searching the MEDLINE database (National Library of Medicine, Bethesda, MD) for relevant articles from the year of MEDLINE's inception through November 2018. Key phrases such as dietary intake and type 2 diabetes, nutrient intake and type 2 diabetes, and correlates of type 2 diabetes with and without the terms SA or Asian Indians were used to identify relevant articles.

## 2. Epidemiology of T2DM in SA

SA living in South Asia or western countries have a higher prevalence of T2DM than other races. Hills et al.<sup>1</sup> have conducted an extensive review on the epidemiology of T2DM in south Asia including data from the Chennai Urban Rural Epidemiology Study (CURES),<sup>2</sup> Indian Council of Medical Research–India DIABetes (ICMR-INDIAB) study,<sup>3</sup> the National Urban Diabetes Survey (NUSDS),<sup>4</sup> two household surveys in India,<sup>5</sup> and the International Diabetes Federation (IDF) report.<sup>6</sup> Mohan et al.<sup>2</sup> reported a 14.3% age-standardized prevalence of T2DM among a representative sample of 2350 Asian Indians, aged 20 years or older, recruited in 2003–2004 from Chennai, India. More recently, Anjana

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et al.<sup>3</sup> reported an overall prevalence of diabetes of 7.3% (including both type 1 and 2 diabetes) and pre-diabetes of 10.3% among 57,117 individuals, aged 20 years or older, in the ICMR-INDIAB cross-sectional study conducted in 15 out of 28 Indian states from 2008 to 2015. The prevalence varied by state and was higher in urban compared to rural areas (11.2% vs. 5.2%). A higher prevalence was observed among those in the low socioeconomic status living in the urban areas of the more economically developed states. However, in rural areas, T2DM was more prevalent in individuals of higher socioeconomic status. Ramachandran et al.<sup>4</sup> reported an age-standardized prevalence of T2DM of 12.1% in the NUDS study among 11,216 participants, aged 20 or older, from 6 major Indian cities including Chennai, Bangalore, Hyderabad, Calcutta, Mumbai and New Delhi and completed in 2000. Geldsetzer et al.<sup>5</sup> reported a crude prevalence of T2DM of 7.5% among 1,320,555 adults, aged 18 years or older, from two household surveys conducted in 2012–2014 in all 29 Indian states except Jammu and Kashmir and Gujarat. The IDF report<sup>6</sup> showed an age-adjusted prevalence of T2DM of 8.4% for Bangladesh, 10.4% for India, 7.3% for Nepal and 10.7% for Sri Lanka for 2017 among adults aged 20–79 years.

Data from two cross-sectional studies conducted in the United States (U.S.) show that the age-adjusted prevalence of T2DM, among individuals 44–84 years, is 23% in SA, 6% in whites, 18% in African Americans, 17% in Latinos, and 13% in Chinese Americans.<sup>7</sup> The data on SA were collected in 2010–2013 among 799 participants and those on the other races and ethnicities were collected in 2000–2002 and included 2611 whites, 1879 African Americans, 1493 Latinos, and 801 Chinese Americans.

Data from The Study of Health Assessment and Risk in Ethnic Groups Study conducted in 3 cities (Hamilton, Toronto, and Edmonton) in Canada, in 1996–1998, among 985 subjects aged 35–75 y, showed that the prevalence of known T2DM (those who were being treated for this condition) was nearly 3 times higher among SA migrants compared to Europeans (6.2% vs. 2.2%, respectively).<sup>8</sup> After excluding the subjects with known T2DM, the same study reported a prevalence of newly diagnosed T2DM of 10% in SA and 6% in Europeans.<sup>8</sup> Data from the administrative health and immigration records in 2005 showed that the prevalence of T2DM among adults, aged 20 years or older, was about 12% in immigrants from south Asia ( $n = 217,367$ ) compared to <6% in those from Western Europe and North America ( $n = 98,931$ ) living in Ontario, Canada.<sup>9</sup>

The 1981 census data from the Southhall Diabetes Survey in west London, United Kingdom (U.K.) showed that the age-adjusted prevalence of known T2DM was >8% in Asians aged 50–59 ( $n = 2290$ ) and >12% in those aged 60–69 ( $n = 1035$ ), and the corresponding percentages for Europeans were <2% ( $n = 3625$ ) and <3% ( $n = 3205$ ), respectively.<sup>10</sup> More recent data from the U.K. showed that the crude prevalence estimates of T2DM among individuals 16 years or older in 2010 was 14% in SA compared to 9.8% in blacks and 6.9% in white, mixed, and other groups.<sup>11</sup>

The prevalence of T2DM in SA living in the U.S. is higher than that of SA living in Canada and the U.K. possibly because the data in the U.S. were collected several years later when the overall prevalence of obesity and T2DM were higher in the U.S. population. The prevalence of T2DM in SA living in the U.S. is also different from that in India possibly due to the effect of acculturation and differences in socioeconomic status. Nevertheless, the prevalence of T2DM is higher in SA compared to whites. In addition, to the high prevalence of T2DM, SA generally develop it a decade earlier<sup>1</sup> and at body mass index that is lower than that of whites.<sup>1,12</sup> Patients who are diagnosed with early onset T2DM are more likely to have earlier onset of cardiovascular disease and more nephropathy compared to those diagnosed with later onset T2DM.<sup>1,13</sup>

### 3. Dietary assessment methodology

Because the focus of this paper is to examine the relationship between nutrient intakes and T2DM, a description of the methods<sup>14,15</sup>

used to assess dietary intake in various studies is given in this section. Also provided are the strengths and limitations of each method.<sup>14,15</sup> All the methods used were valid.

Majority of the studies in this area used a food frequency questionnaire (FFQ) to assess dietary intake. A FFQ consists of a set of foods with response categories to determine the usual frequency of consumption of the foods over a period of time (typically the past month or year). It is usually self-administered, inexpensive to use, does not affect eating behavior, and is often optically scanned for data entry. It is, however, limited by the inability to specify food preparation methods and combination of foods in meals. This is problematic in SA since many prepare their own meals. Moreover, a FFQ may not include all possible foods eaten. The FFQ is also limited by the difficulty that participants have in estimation of frequency of different foods consumed and serving sizes. In addition, the subjects have to remember what was consumed over a long period of time, the questionnaire is cognitively challenging to complete, literacy of the respondent is required, and the responses are affected by season.

Another method used was a 1-day or 3-day dietary recall in which the subjects are asked to recall all the foods consumed, including the type and amount, over the past day or 3 days. The strengths of this method are that the intake is quantified, literacy of the respondent is not required, and it does not affect eating behavior. Limitations include difficulty in estimating portion sizes and underreporting or not remembering all the foods eaten. In addition, the 1-day food recall may not be representative of the usual dietary intake.

The 7-day weighed intake method was used to assess dietary intake in one study. The subjects are asked to weigh and record all the food items consumed over 7 days. The advantage is that the food intake is precisely quantified. The disadvantage includes a high respondent burden and investor cost because of the number of days recorded. In addition, the subjects may change their diet on the day that the diet is recorded.

The last method that was used is the image-assisted dietary assessment method over 3 days. In this method, the subjects are asked to take pictures of the food and drink consumed including second helpings and left-overs on 3 days (two week days and one weekend day). A ruler is placed in front of the plate before the pictures are taken. The researchers then review the pictures and interview the subjects to clarify portion sizes and preparation methods. This method addresses the limitations of the FFQ method. It also reduces underreporting compared to a dietary recall without the images.<sup>15</sup> The limitations of the image-assisted dietary assessment method include having to remember to take photos of meals and snacks, and subjects may change their eating behavior on the day the photos are being taken. In addition, a cell phone or a camera would be needed to implement this method.

None of the studies reviewed below provided objective measures of dietary intake such as blood nutrient values. The Institute of Medicine, the National Institutes of Health, and the Center for Disease Control in United States recognize the issues associated with subjective assessment of dietary intake and the critical need to assess nutritional biomarkers that would serve as objective measures for dietary intake and disease such as T2DM.<sup>16</sup> Nutrient biomarkers are, however, expensive to assess especially in large epidemiologic studies.

### 4. Relationship between nutrient intakes and T2DM in non-SA populations

Many prospective observational studies conducted among whites, Hispanics, African-Americans, and/or East Asians have shown that macronutrient (protein, carbohydrate, and fat) and micronutrient (vitamins and minerals) intakes determine the risk for T2DM. Studies in these populations have shown that total dietary fiber, cereal fiber, and fats derived from plants reduce the risk whereas high intakes of carbohydrate and protein, especially animal protein, enhance the risk for T2DM.<sup>17–22</sup> Other studies, in these populations, have found that micronutrients such as vitamin C, 25-hydroxyvitamin D,  $\beta$ -carotene,  $\alpha$ -tocopherol,

calcium, magnesium, potassium, and zinc, either in the diet and/or blood, are inversely related to T2DM.<sup>23–29</sup> The above studies used a FFQ to assess dietary intake.

## 5. Applicability of findings from non-SA to SA

Very little is known about the role of macronutrient and micronutrient intakes in T2DM in SA. Data from studies in other populations may not be applicable to SA because of differences in macronutrient and micronutrient intakes and consumption of very ethnic specific diets among SA. Based on a number of studies conducted in India and western countries, SA tend to consume more percent energy from carbohydrate and less percent energy from fat and saturated fat compared to the general American population.<sup>30–37</sup> They are also more likely to meet the recommendations for dietary fiber, vitamin C, vitamin E, vitamin K, and magnesium compared to Americans.<sup>35,37,38</sup>

The differences in nutrient intakes may be due to the fact that SA diets tend to be rich in plant foods<sup>39</sup> and include many ethnic specific foods that are usually not found in western diets. SA diets typically consist of curries made with vegetables, beans, or lentils and consumed with rice, chapatti (flat bread made with mostly wheat flour), dosa (a shallow fried pancake made with parboiled rice and black gram dhal batter), or idli (fermented steamed cake made with parboiled rice and black gram dhal batter).<sup>35,40,41</sup> The curries are made with ghee (clarified butter) or vegetable oils. Other foods commonly included are milk, yogurt, savory fried snacks, fruit, nuts, and desserts.<sup>35,40,41</sup> Chicken, fish, or meat are also consumed by SA who are not vegetarians.

## 6. Relationship between nutrient intakes and T2DM in SA populations

The section below presents data from epidemiologic studies (there are no randomized studies) on the relationship between macronutrient and micronutrient intakes and T2DM in SA. This section will also identify gaps in the literature in this population.

The studies on SA migrants in western countries will be presented in a separate section from those on native SA because of the effects of acculturation on lifestyle. Talegawkar et al.<sup>42</sup> have found that length of stay in the U.S. is associated with a generally worsening diet including an increase in consumption of saturated and *trans* unsaturated fats and a decrease in protein, dietary fiber, folate and potassium intake. In addition, Venkatesh et al.<sup>43</sup> have reported that dietary acculturation of SA migrants in the U.S. increases their risk for T2DM.

### 6.1. Studies on SA living in India

#### 6.1.1. Macronutrients

Three cross-sectional studies in India have examined the relationship between macronutrient intake and T2DM in native SA. Shobana et al.<sup>30</sup> examined the relationship between dietary intake and glycemic status in a representative sample of 900 urban SA in Chennai (formerly Madras), India. The researchers found that those with known T2DM ( $n = 55$ ) compared to controls ( $n = 762$ ) had lower energy intake (1557 and 1911 kcal/d, respectively) and percent energy from carbohydrates (61.6% and 66.5%, respectively) and higher percent energy from fat (22.4% and 19%, respectively) and protein (15.9% and 14%, respectively) (Fig. 1). The same study, however, did not find any differences in energy and macronutrient intake between those with newly diagnosed T2DM ( $n = 26$ ) and controls,<sup>30</sup> indicating that diagnosis of diabetes may lead to some dietary changes. This study had several limitations, however. The data on the number of macronutrients were limited and micronutrient intakes were not assessed. In addition, dietary data which were collected using a single 24-h recall, may not be representative of usual intake. The study was also limited to SA living in an urban area. Urbanization is linked to a decline in nutrition quality.<sup>1</sup> In addition, this study design does not permit inference of causation. The study also

did not assess the relationship between nutrient intake and T2DM by dietary pattern such as vegetarian and non-vegetarian even though 42% of the SA households in India are vegetarian (no meat, fish, poultry, or egg consumption) and another 58% are less strict vegetarian or non-vegetarian.<sup>39</sup> Sridhar et al.<sup>44</sup> have reported that vegetarians consumed more dietary fiber, iron, calcium, folate, and vitamin C but less protein and vitamin B12, and had lower risk for cardiovascular disease compared to non-vegetarians living in India. Agrawal et al.<sup>45</sup> have shown that vegetarian diets in India are associated with a lower risk for T2DM compared to non-vegetarian diets.

Mohan et al.<sup>46</sup> evaluated the relationship between macronutrient intake and risk for T2DM in a representative sample ( $n = 1843$ ) of the population from the Chennai Urban Epidemiology Study 59. T2DM was diagnosed from fasting blood glucose and an oral glucose tolerance test. Subjects with a self-reported history of T2DM were excluded. Data from this study showed that carbohydrate intake increased the risk whereas fiber intake decreased the risk for newly diagnosed T2DM. The odds ratio for newly diagnosed T2DM for the highest versus lowest quartile of carbohydrate intake, adjusted for age, sex, body mass index (BMI), income, physical activity, family history of T2DM, smoking, alcohol, and dietary fiber, was 4.55 (95% confidence interval: 2.49, 8.29). The odds ratio for newly diagnosed T2DM for the highest versus lowest quartile of fiber intake, adjusted for age, sex, body mass index (BMI), income, physical activity, family history of T2DM, smoking, alcohol, and dietary carbohydrate was 0.31 (0.15, 0.62). This study had a number of limitations. Data on the relationship between micronutrient, energy, and the remaining macronutrient intakes and T2DM were not shown. Dietary data were collected using a semi-quantitative food frequency questionnaire (FFQ). The strengths and limitations of a FFQ have been described earlier. The dietary database used to calculate nutrient intakes and the food labels were considered limited by the authors. Because the study was cross-sectional, it has the same issues related to study design as described earlier. It is not clear if all the subjects were from an urban background. Lastly the relationships between nutrient intakes and T2DM by dietary pattern were not elucidated.

Joshi et al.<sup>31</sup> evaluated dietary intake of SA with and without T2DM. The subjects were recruited from routine outpatient clinic visits from 10 sites located in north, east, west, south, and central parts of India. The subjects with T2DM had been diagnosed with this condition for at least 12 months and were not on any dietary plan or advice. Dietary intake was measured using a FFQ and 3-day dietary recall. The results showed that individuals with T2DM ( $n = 385$ ) compared to controls ( $n = 409$ ) had lower energy intake (1548 and 2132 kcal/d, respectively), % energy from total carbohydrate (64.1% and 66.8%, respectively), and % energy from simple carbohydrate (7.1% and 13.9%, respectively) but higher % energy from complex carbohydrate (57.0% and 52.9%, respectively) and protein (14.3% and 12.0%, respectively) (Fig. 1). There was no difference in % energy from fat (21.6% and 21.2%, respectively). This study also had several limitations, however. It was cross-sectional design and the authors did not try to recruit a representative sample of the population. It was not clear which dietary intake assessment method was used to generate the results. There were no data on micronutrients and a number of macronutrients. The subjects with T2DM were not newly diagnosed and could have altered their diet after diagnosis even though the authors excluded subjects who were on any current diet plan. Differences by dietary pattern were not examined.

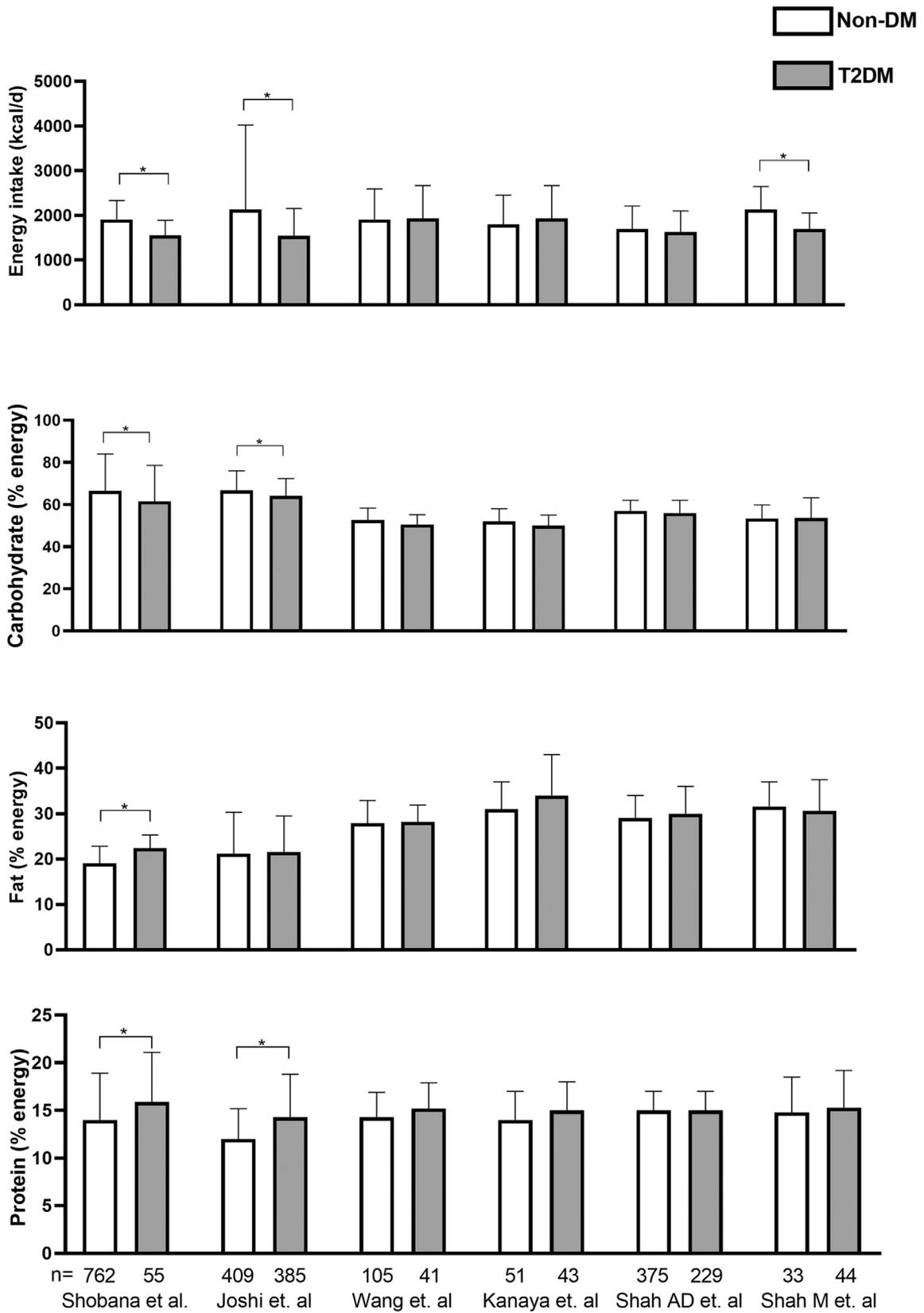
#### 6.1.2. Micronutrients

There are no studies from South Asia that have evaluated the relationship between micronutrient intake and T2DM in SA.

### 6.2. Studies on SA living in western countries

#### 6.2.1. Macronutrients

A large population of SA live in western countries such as the U.K., Canada, and the U.S. According to the 2011 U.K. Census data, there are



**Fig. 1.** Energy intake and percent energy from carbohydrate, fat, and protein in various studies. Each bar indicates mean energy intake and the whisker standard deviation (SD). T2DM: type 2 diabetes mellitus (gray bars); Non-DM: Non-Diabetes Mellitus (white bars). The numbers at the bottom of each bar refer to sample size. The energy intake reported for non-diabetics by Joshi et al. had an unusually large SD compared to mean.

3,078,374 (4.9% of the total population) SA, excluding other Asian groups and people of mixed ethnicity, living in the U.K.<sup>47</sup> The 2016 Census data from Canada show that 1,963,330 SA are residing in that country.<sup>48</sup> Based on the 2010 U.S. Census data, nearly 4 million SA live in the U.S.<sup>49</sup>

Two studies in Britain have examined the relationship between macronutrient intakes and T2DM in SA. Peterson et al.<sup>50</sup> collected dietary data on 48 SA randomly recruited from a diabetes clinic in London. They found that the subjects consumed a diet that was low in carbohydrate (42% of total energy intake) and high in fat (46% of total energy intake) and they commonly consumed saturated fat rich dairy foods and fried snacks.<sup>50</sup> Data on macronutrients were limited and no data were given on micronutrients. The authors noted that they did not provide information on dietary fiber intake because their nutrient database did not have dietary fiber data for many SA foods. The dietary data were collected using only a single 24-h recall. SA with undiagnosed T2D were not examined. In addition, there was no control group in this study or analysis by dietary patterns.

Sevak et al.<sup>32</sup> compared the energy and macronutrient intake of non-diabetic SA British men with high (above the 75th percentile;  $n = 76$ ) and low (below the 28th percentile;  $n = 93$ ) insulin concentrations recruited for the Southhall Study on diabetes and coronary heart disease in west London. They found that the former group consumed less energy from alcohol (1.5% versus 5.2% of total energy) compared to the latter group.<sup>32</sup> There was no difference in dietary fiber and energy intake, and percent energy from carbohydrate, protein and total, saturated, monounsaturated, and polyunsaturated fat intake between the two groups. The authors also compared the data on SA to that of European men recruited for the same study, and found that the former group consumed less energy (9.5 vs 10.8 MJ/d) and percent energy from total fat (36.5% vs 39.2%) (due to less saturated and monounsaturated fat) but more energy from carbohydrate ( $46.4 \pm 6.6$  vs  $40.9 \pm 6.62$ ) (due to starch), and polyunsaturated fatty acids (8.2% vs 7.0% of energy), and consumed more dietary fiber (3.2 vs 2.0 g/MJ) compared to the latter group. The dietary intake in this study was assessed by a rigorous method, the 7-d weighed intake. The authors noted issues with incomplete dietary fatty acid information with the UK food databases. No data were given on micronutrient intakes. The study design was cross-sectional and the information was not given by dietary patterns.

Only three studies, 2 cross-sectional studies and one case-control study, in the U.S., have examined the relationship between nutrient intakes and T2DM among SA in the U.S. Wang et al.<sup>33</sup> and Kanaya et al.<sup>51</sup> published cross-sectional analyses of data, collected between August 2006 and October 2007 for the Metabolic Syndrome and Atherosclerosis in South Asians Living in America Study, on 150 SA Indian migrants living in the San Francisco Bay area. Wang et al.<sup>33</sup> found that SA subjects with T2DM ( $n = 41$ ) consumed less energy from carbohydrate (50.5% versus 52.7%) and more absolute protein intake adjusted for energy intake (74 g versus 69 g) compared to SA without T2DM ( $n = 105$ ) but there was no difference in energy intake or % energy intake from fat or protein between the two groups (Fig. 1). Higher level of protein intake was associated with a 70% increase in the odds of T2DM per standard deviation in g of protein intake/d (standardized odds ratio: 1.70 [95% CI 1.08, 2.68]).<sup>33</sup> Using the same database, Kanaya et al.<sup>51</sup> compared dietary intake between those with normal glucose tolerance ( $n = 51$ ), prediabetes ( $n = 56$ ) or T2DM ( $n = 43$ ) and found no difference in the intake of energy, cereal fiber, and percent energy from fat, carbohydrate and protein among the three groups (Fig. 1). The same group of investigators, Shah AD et al.,<sup>34</sup> published cross-sectional data collected between October 2010 and March 2013 for the Mediators of Atherosclerosis in South Asians Living in America Study on nearly 900 SA subjects recruited from the San Francisco Bay area and the greater Chicago area. The researchers<sup>34</sup> found no difference in energy and macronutrient intake among SA with normal glucose tolerance ( $n = 375$ ), prediabetes ( $n = 295$ ), or T2DM ( $n = 229$ ) (Fig. 1). All of these studies assessed dietary intake using a FFQ. Data on different types of fatty acids, dietary

fiber, and micronutrients were not reported. The data in the first two papers were limited to Indian SA.<sup>33,51</sup> None of the studies evaluated differences in nutrient intakes between subjects with and without T2DM by dietary patterns. Causation may not be inferred from these studies.

More recently, Shah M et al.<sup>35</sup> conducted a case-control study comparing the nutrient intakes of 44 SA with T2DM and 33 age-and-sex matched controls recruited from Dallas-Fort Worth, Texas. Dietary intake was assessed using the image-assisted dietary assessment method, an objective and valid measure.<sup>15</sup> Subjects with T2DM compared to controls reported lower intake of energy (mean difference: 499 kcal/d;  $p < 0.0001$ ), linoleic acid (3.6 g/d;  $p = 0.003$ ), dietary fiber (8.6 g/d;  $p < 0.0001$ ) but no difference in percent energy from carbohydrate, protein, total fat, *trans* fatty acid, saturated fat, polyunsaturated fat, and *cis-monounsaturated* fat, and  $\alpha$ -linolenic acid, eicosapentaenoic acid, and docosahexaenoic acid. They were also more likely not to meet the requirements for linoleic acid and dietary fiber compared to controls. The data on energy intake and percent energy from carbohydrate, fat, and protein are shown in Fig. 1. Differences in nutrient intakes between those with and without T2DM were not evaluated based on dietary pattern. The study did not include subjects with undiagnosed diabetes. The authors noted issues with the nutrition analysis databases and explained the process by which they addressed these issues. They noted that they used a nutrition software package that contained >80,000 foods and about 400 SA foods from various databases across the world but the only database that had generally the most complete set of nutrients was the United States Department of Agriculture (USDA) Standard Reference database. Because of some missing nutrients in the other databases, the authors selected the USDA database to calculate nutrient content of foods whenever possible. For SA foods that were not available in the program, had incomplete nutrient information, the preparation methods were not specified by the databases, or subjects prepared them differently from what was in the database, recipes using raw ingredients from the USDA database were added to the program. More than 60 recipes were added. One of the investigators prepared many of these recipes and weighed the food before adding them to the database.

## 6.2.2. Micronutrients

The study by Shah M et al.,<sup>35</sup> described above, reported that SA with T2DM also had lower intake of vitamin A (262  $\mu$ g/d), vitamin E (2.7 mg/d), calcium (133 mg/d), magnesium (116 mg/d), zinc (1.4 mg/d), potassium (754 mg/d), and  $\beta$ -carotene (1761  $\mu$ g/d) compared to controls. They were also more likely not to meet the requirements for vitamin E, calcium, magnesium, zinc, and potassium compared to controls. The limitations of this study are already described in the previous paragraph. Because the study did not include subjects with undiagnosed diabetes, it raises the possibility that the subjects with T2DM reduced their energy intake after diagnosis which could have affected their nutrient intakes. Nevertheless, this was the only study that has evaluated the relationship between micronutrient as well as macronutrient intakes and T2DM in SA.

## 7. Summary and mechanisms for role of nutrient intakes in T2DM

### 7.1. Dietary fiber intake

Among the studies in SA that reported data on dietary fiber, two studies<sup>35,46</sup> found an inverse correlation between dietary fiber and T2DM and two studies found no relationship.<sup>32,51</sup> According to a meta-analysis of prospective cohort studies in other populations, total dietary fiber and cereal fiber are inversely related to T2DM.<sup>17</sup> In a review paper, Gulati and Misra<sup>52</sup> noted that SA in India are increasingly replacing foods rich in fiber such as whole wheat flour chapattis, millet chapattis, and pancakes made from gram flour with foods low in fiber content such as noodles, vermicelli and refined flour breads. Increasing fiber intake improves glycemic response in SA. Mohan et al.<sup>53</sup> found that

replacing white rice with brown rice reduced 24-h glucose and fasting insulin responses in overweight Asian Indians. Recently, Boers et al.<sup>54</sup> evaluated the effect of increasing the soluble fiber content of a flatbread flour mix, by adding guar gum and chick pea flour to the mix, on glycemic response in healthy SA, and found that the addition of soluble fiber improved both postprandial glucose and insulin responses compared to just the flatbread flour mix.

There are several mechanisms by which soluble fiber may control the glycemic response to a meal. Soluble fiber, also known as viscous or fermentable fiber, improves the postprandial glycemic response by delaying gastric emptying and transit in the small bowel, and reduces glucose diffusion and access of  $\alpha$ -amylase to the carbohydrate substrate due to the viscosity in the gut content caused by the viscous fiber.<sup>55</sup> Soluble fiber may also improve glycemic control by increasing the production of an incretin hormone, glucagon-like peptide-1,<sup>55</sup> and increasing the expression of glucose transporter type 4 in skeletal muscle and peripheral glucose uptake.<sup>55</sup> Another mechanism for improved insulin sensitivity with soluble fiber may be related to the production of short-chain fatty acids (SCFAs) through the bacterial action in the gut on fermentable fiber.<sup>56</sup> Some of these SCFAs reduce the mobilization of free fatty acids from adipose cells by acting on G protein-coupled receptor 43/free fatty acid receptor 2 in these cells.<sup>56</sup> Robertson et al.<sup>57</sup> found that resistant starch reduced subcutaneous abdominal adipose tissue free fatty acid release and increased insulin sensitivity. Insoluble fiber may also be involved in improved glycemic response. Weikert et al.<sup>58</sup> reported that compared to consuming just white bread, test meals containing bread with highly purified insoluble fibers accelerated the early postprandial insulin and glucose-dependent insulinotropic polypeptide (GIP) responses and reduced the postprandial glucose response the following day in healthy women.

### 7.2. Dietary carbohydrate intake

The studies on SA reviewed above have shown mixed findings on energy intake from carbohydrate and glycemic status possibly because those with T2DM may have changed their diet. However, mixed results were also found among the 5 studies that compared subjects with newly diagnosed or unknown T2DM or prediabetes or those with high plasma insulin levels with controls.<sup>30,32,34,46,51</sup> Of these, 3 studies<sup>30,34,51</sup> found no difference and 2 studies found a higher percent energy from carbohydrates among those with newly diagnosed T2DM<sup>46</sup> or hyperinsulinemic subjects.<sup>32</sup> In a systematic review and meta-analysis of prospective cohort studies conducted in the U.S. and Europe, Alhazmi et al.<sup>18</sup> also found a higher risk for T2DM with higher total carbohydrate intake. This may be related to the high glycemic index (GI) and glycemic load (GL) associated with high carbohydrate diets rich in starch and added sugars. Both high GI and high GL are linked to T2DM.<sup>59,60</sup> Willett et al.<sup>59</sup> have reported that diets with high GI or GL lead to higher postprandial glucose and insulin responses. A higher demand for insulin may impair beta cell function and cause T2DM in individuals with low beta cell reserve.<sup>61</sup>

### 7.3. Dietary protein intake

Majority of the studies on SA, reviewed above, did not find any difference in percent energy from protein between those with and without T2DM<sup>32,34,35,51</sup> or did not report any findings on protein intake.<sup>46</sup> Two studies<sup>30,31</sup> reported a higher energy from protein in the SA subjects with T2DM compared to controls. One of these studies, Shobana et al.,<sup>30</sup> however, found no difference in percent energy from protein intake between subjects with just newly diagnosed T2DM and controls. The studies in SA did not separate protein intake into animal and plant protein even though many SA are vegetarian.<sup>39</sup> Malik et al.<sup>22</sup> found higher risk for T2DM with higher animal protein intake and a modestly lower risk with plant protein in the Nurses' Health and Health Professionals Follow-up cohorts in the U.S. The association between

animal protein and T2DM may be due to red and processed meat. Heme iron from red and processed meat may cause oxidative damage to the pancreas.<sup>62</sup> Nitrites from red meat are converted to nitrosamines which may damage the beta-cells in the pancreas.<sup>62</sup> Advanced glycation end products from red and processed meat may also be a mechanism linking red and processed meat with T2DM.<sup>62</sup> A diet rich in plant protein, specifically nuts, legumes, and wholegrains, will contain more fiber and have a lower GI and GL. These have been shown previously to improve glycemic response.

### 7.4. Dietary fat intake

Majority of the studies in SA reported no difference in dietary fat intake between those with T2DM and controls. Shobana et al.<sup>30</sup> found a higher percent energy from fat in SA with T2DM compared to controls but this difference disappeared when the subjects with known T2DM were excluded. Shah M et al.<sup>35</sup> found a lower percent energy intake from linoleic acid in subjects with T2DM compared to controls. A systematic and meta-analysis of cohort studies on diet composition and T2DM in other populations showed that a high vegetable fat intake was linked with a lower risk for T2DM in women.<sup>18</sup> The major fat in vegetable fat is polyunsaturated fatty acids.<sup>18</sup> Forouhi et al.<sup>19</sup> evaluated the relationship between dietary fat and T2DM in the European Prospective Investigation into Cancer and Nutrition (EPIC) study across 8 European countries and found an inverse relationship between circulating linoleic acid and T2DM but no convincing association between marine-derived omega-3 fatty acids and T2DM. Gulati and Misra<sup>52</sup> have reported that SA in India are increasingly replacing *cis*-monounsaturated and polyunsaturated fatty acids with *trans*-unsaturated and saturated fatty acids through consumption of partially hydrogenated vegetable oils and palmolein oil. Moreover, Bhardwaj et al.<sup>63</sup> have shown that constantly heating oils or fats for frying, a common practice among SA, results in more *trans*-unsaturated and saturated fats and less *cis*-unsaturated fats. Gulati et al.<sup>64</sup> have shown that incorporating almonds (rich in *cis*-monounsaturated fat, polyunsaturated fat, and soluble fiber) in a healthy diet for 24 months along with increased physical activity improved glycemic control in SA with T2DM living in New Delhi. According to a systematic review and meta-analysis of randomized controlled feeding trials by Imamura et al.,<sup>65</sup> replacing saturated fat with polyunsaturated fat lowers fasting glucose and HbA1c, and improves insulin resistance and insulin secretion capacity.<sup>65</sup> Replacing saturated fat with *cis*-monounsaturated fat only improved HbA1c and insulin resistance.<sup>65</sup> The authors attributed the improvement in glycemic control with polyunsaturated fat to omega-6 but not omega-3 fatty acids.<sup>65</sup> Linolenic acid within cell membranes in phospholipids may modulate insulin receptor activity by increasing membrane fluidity.<sup>66</sup>

### 7.5. Energy intake

Among the studies that reported data on energy intake, three studies found no difference in energy intake between those with and without T2DM<sup>33</sup> or between those with normal glucose tolerance, prediabetes or T2DM,<sup>34,51</sup> or between those with high or low 2-h postprandial insulin levels.<sup>32</sup> The remaining 3 studies found a lower energy intake among SA with T2DM compared to controls.<sup>30,31,35</sup> Of these 3 studies, one study<sup>30</sup> reported that the difference in energy intake between those with and without T2DM disappeared when the diabetic subjects were limited to just newly diagnosed T2DM indicating that the lower energy intake in subjects with T2DM may be due to changes in the diet following diagnosis. Although the majority of the above studies indicated no difference in energy intake between those with and without T2DM, decreasing energy intake and body weight among those with T2DM has been shown to improve glycemic control irrespective of diet composition.<sup>67</sup>

## 7.6. Micronutrient intake

None of the studies in SA, except for the study by Shah M et al.,<sup>35</sup> evaluated the relationship between micronutrient intake and T2DM. Shah M et al.<sup>35</sup> found that SA with T2DM consumed less vitamin A, vitamin E, calcium, magnesium, zinc, potassium, and  $\beta$ -carotene compared to controls. Studies in other populations including whites, Hispanics, African-Americans, and/or East Asian have also found an inverse relationship between dietary and/or serum vitamin C, 25-hydroxyvitamin D,  $\beta$ -carotene,  $\alpha$ -tocopherol, calcium, magnesium, potassium, and zinc and T2DM.<sup>23–29</sup> The inverse relationship between these nutrients and T2DM may be mediated via enhancement of insulin mediated glucose uptake or beta cell function.<sup>68–72</sup>

## 8. Future research

The current literature in the area of nutrient intakes in type 2 diabetes in SA is limited by a number of methodological issues discussed above. Identified below are the types of future studies that are needed in SA based on the methodological constraints of the current literature.

- There are very few epidemiologic studies examining the role of nutrient intakes in T2DM in SA. Most of the previous case-control and cross-sectional studies were conducted in those with known T2DM which may affect dietary intake. Future case-control and cross-sectional studies need to compare the nutrient intakes of SA with undiagnosed T2DM or pre-diabetes with controls. There are no prospective cohort studies examining the role of nutrient intakes in T2DM in SA. Prospective cohort studies are needed to examine the relationship between nutrient intake and the development of T2DM in those with normal blood glucose and prediabetes. The following recommendations should be considered when designing these epidemiologic studies:
  - Because of the effect of acculturation on dietary intake, data needs to be collected in SA living in both South Asia and western countries as well as urban and rural areas.
  - Most studies examined the role of just macronutrients in T2DM. Comprehensive dietary assessment including the role of macronutrient and micronutrient intakes in T2DM should be undertaken.
  - A large segment of the SA population is vegetarian. Epidemiologic studies examining the role of vegetarian versus non-vegetarians diets in T2DM are needed.
  - Because a FFQ or 24-h recall have a number of limitations identified earlier, other methods such as the image-assisted dietary intake assessment method and blood nutrient values should be considered.
- There are very few randomized controlled trials examining the effect of dietary and nutrient intakes on prevention and treatment of T2DM in SA. Some specific areas to focus when designing these studies in SA are:
  - Examining the effect of high fiber versus moderate fiber diets on prevention and treatment of T2DM.
  - Examining the effect of varying carbohydrate (starch) and fat (*cis-monounsaturated* fat) intake while controlling protein and fiber intake on prevention and treatment of T2DM.
  - Comparing the effect of plant versus animal protein diets on prevention and treatment of T2DM.
  - Comparing the effect of varying amount of dietary fatty acids such as polyunsaturated, saturated, and *cis-monounsaturated* fatty acids on prevention and control of T2DM.
  - Examining the effect of specific micronutrients identified above on prevention and treatment of T2DM.
- The current nutrient databases have incomplete nutrient values for many SA foods. There is a pressing need to develop a more complete

nutrient database for foods consumed by SA in collaboration with the USDA.

## 9. Overall perspective

The review showed that certain nutrients are related to T2DM in SA and similar findings were also noted from studies conducted in non-SA populations. The literature on nutrient intakes and T2DM in SA is, however, limited by a small number of studies, lack of prospective cohort and randomized controlled studies, issues with dietary assessment methodology, assessing SA with known T2DM rather than those with undiagnosed T2DM or prediabetes, and examining only a few nutrients. Future research using more rigorous study design and methods and examining a comprehensive set of nutrients will help to further elucidate the role of nutrient in T2DM in SA. SA diets may be different by country and rural/urban settings suggesting that studies in this population should be conducted in different locations. A large segment of the SA population is vegetarian justifying examining how dietary patterns and changing the composition of the diet affects the prevention and treatment of T2DM. Lastly, physical activity levels, obesity, genetic predisposition, intrauterine and early life exposures, environmental pollutants, stress, and socio-economic status, although not reviewed in this paper, may be other contributing factors in the development of T2DM in SA.

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## References

- Hills AP, Arena R, Khunti K, et al. Epidemiology and determinants of type 2 diabetes in south Asia. *Lancet Diabetes Endocrinol* 2018;6:966–78.
- Mohan V, Deepa M, Deepa R, et al. Secular trends in the prevalence of diabetes and impaired glucose tolerance in urban South India—the Chennai Urban Rural Epidemiology Study (CURES-17). *Diabetologia* 2006;49:1175–8.
- Anjana RM, Deepa M, Pradeepa R, et al. Prevalence of diabetes and prediabetes in 15 states of India: results from the ICMR-INDIAB population-based cross-sectional study. *Lancet Diabetes Endocrinol* 2017;5:585–96.
- Ramachandran A, Snehalatha C, Kapur A, et al. High prevalence of diabetes and impaired glucose tolerance in India: National Urban Diabetes Survey. *Diabetologia* 2001;44:1094–101.
- Geldsetzer P, Manne-Goehler J, Theilmann M, et al. Diabetes and hypertension in India: a nationally representative study of 1.3 million adults. *JAMA Intern Med* 2018;178:363–72.
- International Diabetes Federation. IDF diabetes atlas teBIDF. <http://www.diabetesatlas.org/resources/2017-atlas.html> 2017. Accessed April 4, 2018.
- Kanaya AM, Herrington D, Vittinghoff E, et al. Understanding the high prevalence of diabetes in U.S. south Asians compared with four racial/ethnic groups: the MASALA and MESA studies. *Diabetes Care* 2014;37:1621–8.
- Anand SS, Yusuf S, Vuksan V, et al. Differences in risk factors, atherosclerosis, and cardiovascular disease between ethnic groups in Canada: the Study of Health Assessment and Risk in Ethnic groups (SHARE). *Lancet* 2000;356:279–84.
- Creatore MI, Moineddin R, Booth G, et al. Age- and sex-related prevalence of diabetes mellitus among immigrants to Ontario, Canada. *Canadian Medical Association Journal* 2010;182:781–9.
- Mather HM, Keen H. The Southall Diabetes Survey: prevalence of known diabetes in Asians and Europeans. *Br Med J (Clin Res Ed)* 1985;291:1081–4.
- Holman N, Forouhi NG, Goyder E, et al. The Association of Public Health Observatories (APHO) Diabetes Prevalence Model: estimates of total diabetes prevalence for England, 2010–2030. *Diabet Med* 2011;28:575–82.
- Snehalatha C, Viswanathan V, Ramachandran A. Cutoff values for normal anthropometric variables in asian Indian adults. *Diabetes Care* 2003;26:1380–4.
- Yeung RO, Zhang Y, Luk A, et al. Metabolic profiles and treatment gaps in young-onset type 2 diabetes in Asia (the JADE programme): a cross-sectional study of a prospective cohort. *Lancet Diabetes Endocrinol* 2014;2:935–43.
- Coulston AM, Boushey CJ. *Nutrition in the prevention and treatment of disease*. 2nd ed. Burlington, MA: Elsevier Academic Press; 2008.
- Gemming L, Utter J, Ni MC. Image-assisted dietary assessment: a systematic review of the evidence. *J Acad Nutr Diet* 2015;15:64–77.
- Pfeiffer CM, Sternberg MR, Schleicher RL, et al. The CDC's Second National Report on Biochemical Indicators of Diet and Nutrition in the U.S. Population is a valuable tool for researchers and policy makers. *J Nutr* 2013;143:938S–47S.

17. InterAct C. Dietary fibre and incidence of type 2 diabetes in eight European countries: the EPIC-InterAct Study and a meta-analysis of prospective studies. *Diabetologia* 2015;58:1394–408.
18. Alhazmi A, Stojanovski E, McEvoy M, et al. Macronutrient intakes and development of type 2 diabetes: a systematic review and meta-analysis of cohort studies. *J Am Coll Nutr* 2012;31:243–58.
19. Forouhi NG, Imamura F, Sharp SJ, et al. Association of plasma phospholipid n-3 and n-6 polyunsaturated fatty acids with type 2 diabetes: the EPIC-InterAct Case-Cohort study. *PLoS Med* 2016;13, e1002094.
20. Yary T, Voutilainen S, Tuomainen TP, et al. Serum n-6 polyunsaturated fatty acids, Delta5- and Delta6-desaturase activities, and risk of incident type 2 diabetes in men: the Kuopio Ischaemic Heart Disease Risk Factor Study. *Am J Clin Nutr* 2016;103:1337–43.
21. AlEssa HB, Bhupathiraju SN, Malik VS, et al. Carbohydrate quality and quantity and risk of type 2 diabetes in US women. *Am J Clin Nutr* 2015;102:1543–53.
22. Malik VS, Li Y, Tobias DK, et al. Dietary protein intake and risk of type 2 diabetes in US men and women. *Am J Epidemiol* 2016;183:715–28.
23. Villegas R, Gao YT, Dai Q, et al. Dietary calcium and magnesium intakes and the risk of type 2 diabetes: the Shanghai Women's Health Study. *Am J Clin Nutr* 2009;89:1059–67.
24. Fang X, Wang K, Han D, et al. Dietary magnesium intake and the risk of cardiovascular disease, type 2 diabetes, and all-cause mortality: a dose-response meta-analysis of prospective cohort studies. *BMC Med* 2016;14:210.
25. Arnlov J, Zethelius B, Riserus U, et al. Serum and dietary beta-carotene and alpha-tocopherol and incidence of type 2 diabetes mellitus in a community-based study of Swedish men: report from the Uppsala Longitudinal Study of Adult Men (ULSAM) study. *Diabetologia* 2009;52:97–105.
26. Vashum KP, McEvoy M, Shi Z, et al. Is dietary zinc protective for type 2 diabetes? Results from the Australian longitudinal study on women's health. *BMC Endocr Disord* 2013;13:40.
27. Harding AH, Wareham NJ, Bingham SA, et al. Plasma vitamin C level, fruit and vegetable consumption, and the risk of new-onset type 2 diabetes mellitus: the European prospective investigation of cancer—Norfolk prospective study. *Arch Intern Med* 2008;168:1493–9.
28. Ekmekcioglu C, Haluza D, Kundi M. 25-Hydroxyvitamin D status and risk for colorectal cancer and type 2 diabetes mellitus: a systematic review and meta-analysis of epidemiological studies. *Int J Environ Res Public Health* 2017;14.
29. Chatterjee R, Davenport CA, Svetkey LP, et al. Serum potassium is a predictor of incident diabetes in African Americans with normal aldosterone: the Jackson Heart Study. *Am J Clin Nutr* 2017;105:442–9.
30. Shobana R, Snehalatha C, Latha E, et al. Dietary profile of urban south Indians and its relations with glycaemic status. *Diabetes Res Clin Pract* 1998;42:181–6.
31. Joshi SR, Bhansali A, Bajaj S, et al. Results from a dietary survey in an Indian T2DM population: a STARCH study. *BMJ Open* 2014;4, e005138.
32. Sevak L, McKeigue PM, Marmot MG. Relationship of hyperinsulinemia to dietary intake in south Asian and European men. *Am J Clin Nutr* 1994;59:1069–74.
33. Wang ET, de Koning L, Kanaya AM. Higher protein intake is associated with diabetes risk in South Asian Indians: the Metabolic Syndrome and Atherosclerosis in South Asians Living in America (MASALA) study. *J Am Coll Nutr* 2010;29:130–5.
34. Shah AD, Vittinghoff E, Kandula NR, et al. Correlates of prediabetes and type II diabetes in US South Asians: findings from the Mediators of Atherosclerosis in South Asians Living in America (MASALA) study. *Ann Epidemiol* 2015;25:77–83.
35. Shah M, Vasandani C, Adams-Huet B, et al. Comparison of nutrient intakes in South Asians with type 2 diabetes mellitus and controls living in the United States. *Diabetes Res Clin Pract* 2018;138:47–56.
36. Wright JD, Wang CY. Trends in intake of energy and macronutrients in adults from 1999–2000 through 2007–2008. *NCHS Data Brief* 2010:1–8.
37. McGuire S. Scientific Report of the 2015 Dietary Guidelines Advisory Committee. Washington, DC: US Departments of Agriculture and Health and Human Services, 2015. *Adv Nutr* 2016;7:202–4.
38. Millen BE, Abrams S, Adams-Campbell L, et al. The 2015 Dietary Guidelines Advisory Committee scientific report: development and major conclusions. *Adv Nutr* 2016;7:438–44.
39. FAOSTAT. Food and Agriculture Organization of the United Nations, statistical database. Available from: <http://faostat.fao.org/>. Accessed June 6, 2018.
40. Misra A, Singhal N, Sivakumar B, et al. Nutrition transition in India: secular trends in dietary intake and their relationship to diet-related non-communicable diseases. *J Diabetes* 2011;3:278–92.
41. Radhika G, Sathya RM, Ganesan A, et al. Dietary profile of urban adult population in South India in the context of chronic disease epidemiology (CURES-68). *Public Health Nutr* 2011;14:591–8.
42. Talegawkar SA, Kandula NR, Gadgil MD, et al. Dietary intakes among South Asian adults differ by length of residence in the USA. *Public Health Nutr* 2016;19:348–55.
43. Venkatesh S, Conner T, Song WO, et al. The relationship between dietary acculturation and type 2 diabetes risk among Asian Indians in the U.S. *J Immigr Minor Health* 2017;19:294–301.
44. Shridhar K, Dhillon PK, Bowen L, et al. The association between a vegetarian diet and cardiovascular disease (CVD) risk factors in India: the Indian Migration Study. *PLoS One* 2014;9, e110586.
45. Agrawal S, Millett CJ, Dhillon PK, et al. Type of vegetarian diet, obesity and diabetes in adult Indian population. *Nutr J* 2014;13:89.
46. Mohan V, Radhika G, Sathya RM, et al. Dietary carbohydrates, glycaemic load, food groups and newly detected type 2 diabetes among urban Asian Indian population in Chennai, India (Chennai Urban Rural Epidemiology Study 59). *Br J Nutr* 2009;102:1498–506.
47. 2011 census: ethnic group, local authorities in the United Kingdom. Office for National Statistics, 11 October 2013. Retrieved 25 April 2015.
48. Statistics Canada. 2016 Census. <https://www12.statcan.gc.ca/census-recensement/2016/dp-pd/prof/index.cfm?Lang=E> Retrieved 10 March 2016.
49. A demographic snapshot of South Asians in the United States. [http://saalt.org/wp-content/uploads/2016/01/Demographic-Snapshot-updated\\_Dec-2015.pdf](http://saalt.org/wp-content/uploads/2016/01/Demographic-Snapshot-updated_Dec-2015.pdf) December 2015. Accessed February 20, 2017.
50. Peterson DB, Dattani JT, Baylis JM, et al. Dietary practices of Asian diabetics. *Br Med J (Clin Res Ed)* 1986;292:170–1.
51. Kanaya AM, Wassel CL, Mathur D, et al. Prevalence and correlates of diabetes in South Asian Indians in the United States: findings from the metabolic syndrome and atherosclerosis in South Asians living in America study and the multi-ethnic study of atherosclerosis. *Metab Syndr Relat Disord* 2010;8:157–64.
52. Gulati S, Misra A. Abdominal obesity and type 2 diabetes in Asian Indians: dietary strategies including edible oils, cooking practices and sugar intake. *Eur J Clin Nutr* 2017;71:850–7.
53. Mohan V, Spiegelman D, Sudha V, et al. Effect of brown rice, white rice, and brown rice with legumes on blood glucose and insulin responses in overweight Asian Indians: a randomized controlled trial. *Diabetes Technol Ther* 2014;16:317–25.
54. Boers HM, MacAulay K, Murray P, et al. Efficacy of fibre additions to flatbread flour mixes for reducing post-meal glucose and insulin responses in healthy Indian subjects. *Br J Nutr* 2017;117:386–94.
55. Papatheanasopoulos A, Camilleri M. Dietary fiber supplements: effects in obesity and metabolic syndrome and relationship to gastrointestinal functions. *Gastroenterology* 2010;138:65–72 e61–62.
56. Sleeth ML, Thompson EL, Ford HE, et al. Free fatty acid receptor 2 and nutrient sensing: a proposed role for fibre, fermentable carbohydrates and short-chain fatty acids in appetite regulation. *Nutr Res Rev* 2010;23:135–45.
57. Robertson MD, Bickerton AS, Dennis AL, et al. Insulin-sensitizing effects of dietary resistant starch and effects on skeletal muscle and adipose tissue metabolism. *Am J Clin Nutr* 2005;82:559–67.
58. Weickert MO, Mohlig M, Koebnick C, et al. Impact of cereal fibre on glucose-regulating factors. *Diabetologia* 2005;48:2343–53.
59. Willett W, Manson J, Liu S. Glycemic index, glycemic load, and risk of type 2 diabetes. *Am J Clin Nutr* 2002;76:274S–80S.
60. Krishnan S, Rosenberg L, Singer M, et al. Glycemic index, glycemic load, and cereal fiber intake and risk of type 2 diabetes in US black women. *Arch Intern Med* 2007;167:2304–9.
61. Fung TT, McCullough M, van Dam RM, et al. A prospective study of overall diet quality and risk of type 2 diabetes in women. *Diabetes Care* 2007;30:1753–7.
62. Pan A, Sun Q, Bernstein AM, et al. Red meat consumption and risk of type 2 diabetes: 3 cohorts of US adults and an updated meta-analysis. *Am J Clin Nutr* 2011;94:1088–96.
63. Bhardwaj S, Passi SJ, Misra A, et al. Effect of heating/reheating of fats/oils, as used by Asian Indians, on trans fatty acid formation. *Food Chem* 2016;212:663–70.
64. Gulati S, Misra A, Pandey RM. Effect of almond supplementation on glycaemia and cardiovascular risk factors in Asian Indians in North India with type 2 diabetes mellitus: a 24-week study. *Metab Syndr Relat Disord* 2017;15:98–105.
65. Imamura F, Micha R, Wu JH, et al. Effects of saturated fat, polyunsaturated fat, mono-unsaturated fat, and carbohydrate on glucose-insulin homeostasis: a systematic review and meta-analysis of randomised controlled feeding trials. *PLoS Med* 2016;13, e1002087.
66. Kroger J, Jacobs S, Jansen EH, et al. Erythrocyte membrane fatty acid fluidity and risk of type 2 diabetes in the EPIC-Potsdam study. *Diabetologia* 2015;58:282–9.
67. Heilbronn LK, Noakes M, Clifton PM. Effect of energy restriction, weight loss, and diet composition on plasma lipids and glucose in patients with type 2 diabetes. *Diabetes Care* 1999;22:889–95.
68. Vincent HK, Bourguignon CM, Weltman AL, et al. Effects of antioxidant supplementation on insulin sensitivity, endothelial adhesion molecules, and oxidative stress in normal-weight and overweight young adults. *Metabolism* 2009;58:254–62.
69. Jayawardena R, Ranasinghe P, Galappaththy P, et al. Effects of zinc supplementation on diabetes mellitus: a systematic review and meta-analysis. *Diabetol Metab Syndr* 2012;4:13.
70. Gagnon C, Daly RM, Carpentier A, et al. Effects of combined calcium and vitamin D supplementation on insulin secretion, insulin sensitivity and beta-cell function in multi-ethnic vitamin D-deficient adults at risk for type 2 diabetes: a pilot randomized, placebo-controlled trial. *PLoS One* 2014;9, e109607.
71. Zillich AJ, Garg J, Basu S, et al. Thiazide diuretics, potassium, and the development of diabetes: a quantitative review. *Hypertension* 2006;48:219–24.
72. Song Y, He K, Levitan EB, et al. Effects of oral magnesium supplementation on glycaemic control in Type 2 diabetes: a meta-analysis of randomized double-blind controlled trials. *Diabet Med* 2006;23:1050–6.