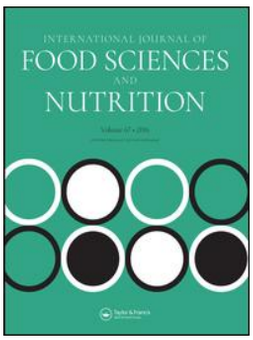

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RESEARCH ARTICLE

Dietary inflammatory index score, glucose control and cardiovascular risk factors profile in people with type 2 diabetes

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ABSTRACT

We examined the relationships between the dietary inflammatory index (DII®), dietary habits and cardiovascular risk factor profiles in people with type 2 diabetes mellitus (T2DM). Energy-adjusted DII (E-DII™) scores were calculated from a Food Frequency Questionnaire in 2568 T2DM patients from different parts of Italy. Analyses were conducted according to quartiles of sex-specific E-DII scores. Higher, more pro-inflammatory, (quartile 4) E-DII scores were associated with overall poor quality of the diet characterised by higher content of refined carbohydrates, added sugars, saturated fat and cholesterol and lower unsaturated fat, fibre and polyphenols compared to quartile 1. Higher E-DII scores also were associated with higher waist circumference (105.4 vs. 103.5 cm; $p = 0.002$), triglycerides (154.6 vs. 146.1 mg/dL; $p = 0.005$), diastolic blood pressure (80.05 vs. 78.6 mmHg; $p = 0.04$) and lower HDL-cholesterol (45.3 vs. 47.4 mg/dL; $p = 0.04$). In conclusion, E-DII is a potent marker of overall quality of the diet and is associated with an unfavourable cardiovascular risk factor profile.

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Dietary Inflammatory Index; cardiovascular risk factors; body weight; glucose control; plasma lipids

Introduction

Type 2 diabetes mellitus (T2DM) is a multifactorial condition characterised by a range of metabolic abnormalities including hyperglycaemia, insulin resistance, dyslipidemia, hypertension and abdominal obesity, which largely account for the high risk of cardiovascular diseases (CVD) associated with diabetes (Vaccaro et al. 2004). Increments in the production of pro-inflammatory cytokines further increase this risk, underlining the important link between obesity, diabetes and CVD (Ridker et al. 2002; Pearson et al. 2003). Diet is an important determinant of systemic inflammation and, not coincidentally, is one of the strongest lifestyle-related factors for the development of CVD (AMD and SID 2018).

The Dietary Inflammatory Index (DII®) has been developed as a means to assess the overall inflammatory properties of diet on a continuum from maximally anti- to pro-inflammatory (Shivappa et al. 2014). DII scores have been associated with indicators of systemic inflammation (Wirth et al. 2016; Shivappa et al. 2018a) and with several chronic, inflammation-related disabling conditions, including CVD (Garcia-Arellano et al. 2015; O'Neil et al. 2015; Ramallal et al. 2015; Ruiz-Canela et al. 2015; Neufcourt et al. 2016; Sokol et al. 2016). In a recent meta-analysis individuals with the highest DII scores, indicating the most pro-inflammatory diet, showed a 36% increased risk of CVD incidence and mortality, relative to those with the lowest DII scores (Shivappa et al. 2018b).

Despite the well-established relationship between inflammatory biomarkers and inflammation-related chronic disease endpoints, the relationship between the DII and intermediate biomarkers of cardiometabolic health is insufficiently explored. Several, but not all, studies in different populations have shown that the inflammatory properties of the diet are associated

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with hypertension, higher plasma triglyceride and lower high-density lipoprotein (HDL) cholesterol concentrations and abnormal lipoprotein composition (Neufcourt et al. 2016; Sokol et al. 2016). A few studies have evaluated the role of the DII as a risk factor for the development of diabetes, but to our knowledge no study to date has documented the DII score of habitual diet in people with diagnosed diabetes (Hébert et al. 2014; Denova-Gutiérrez et al. 2018; Hébert et al. 2019; King and Xiang 2019; Laouali et al. 2019; Mtintsilana et al. 2019; Shivappa et al. 2019). Thus, the evaluation of the DII in different population settings is important because dietary practices vary and might have an impact on both DII score and its relationship with cardiovascular health. In particular, the recommended diet for people with diabetes is low in saturated fat (SFA), refined carbohydrates and added sugars, high in fibre, and relatively rich in polyunsaturated (PUFA) fat and polyphenols (AMD and SID 2018). This dietary prescription is consistent with the Mediterranean dietary pattern, which has shown several health benefits, including protection against CV events and anti-inflammatory properties. Although adherence to the nutritional recommendations in people with diabetes is generally low (Rivellese et al. 2008; Vitale et al. 2016), it is expected that people with diabetes may have healthier eating behaviour than the general population.

The aim of this work is to document, in a clinic-based sample of patients with physician-diagnosed T2DM, the DII scores of the habitual diet, its relation with food components with known health effects (i.e. fibre, SFA, omega-3, PUFA, polyphenols) and its relation with glucose control and major cardiovascular risk factors.

Materials and methods

Study population

The study population consists of 2568 patients with T2DM diagnosed for at least 2 years, aged 50–75 years, participating in the TOSCA.IT study (NCT00700856), a clinical trial designed to compare the effects of sulfonylurea or pioglitazone, in addition to metformin, on cardiovascular events (Vaccaro et al. 2017). All study participants were insufficiently controlled while taking metformin at the dosage of 2 g per day and were randomly assigned to receive the addition of either pioglitazone or sulfonylurea. Participants were recruited between 2008 and 2014 at diabetes clinics all over the national territory (North 35%, Centre 26%, South 39%). For the purposes of the present analysis, we used data collected at the baseline visit, which was performed one to four weeks prior to randomisation to the study treatments. Patients with co-morbidities requiring a special dietary treatment were excluded from the analysis. The protocol of the TOSCA.IT study was approved by the Ethics Review Board of the Coordinating Centre and that of each participating centre. All participants signed a written informed consent. At baseline, a full clinical examination was performed by a physician; among other factors, sitting blood pressure was measured according to a standard protocol. Anthropometric data (weight, height, waist circumference) were collected by a registered dietitian in the morning after an overnight fast. Measured weight and height were used to compute body mass index [BMI = weight(kg)/height(m)^2]. Fasting blood samples were drawn on the same day; biochemical analyses were performed by a central laboratory. Total cholesterol, HDL-cholesterol and triglycerides were measured by standard methods, LDL-cholesterol was calculated according to the Friedewald equation (Friedewald et al. 1972) for people with triglycerides values <400 mg/dl. For people with triglyceride values >400 mg/dL (seven in total), the LDL-cholesterol was considered as a missing value. Glucose control was evaluated by glycated haemoglobin (HbA1c), measured with high-performance liquid chromatography (HPLC).

Personal and clinical information including education, marital status, smoking habits, and use of lipid lowering or anti-hypertensive medications were collected by questionnaire. The participants were classified as smokers if they were currently smoking one or more cigarettes per day on a regular basis.

Assessment of dietary habits

Dietary intakes were evaluated by the use of a self-administered semi-quantitative food frequency questionnaire, the Italian version of the European Prospective Investigation into Cancer and Nutrition (EPIC) questionnaire (Pisani et al. 1997; Pala et al. 2003). The questionnaire was administered in most cases on the same day of the baseline visit; in some cases, it was returned at the randomisation visit. The questionnaire contains 248 items with multiple possible answers, on 188 different foods, divided into 15 sections. The respondent indicates the frequency of consumption of each item in two ways: absolute frequency (per day, week, month or year) and relative frequency (or almost never, sometimes, about half of the time, most of the time, all the time). The amount

of the food consumed was assessed by a selection of pictures showing a small, medium and large portion size, with additional quantifiers (e.g. "smaller than the small portion" or "between the small and medium portion"). The questionnaires have been subjected to a quality check: those with more than 20 questions left unanswered were excluded from the analyses, as well as questionnaires with implausible data (i.e. daily caloric intake <800 or > 5000 Kcal). The questionnaires were electronically read and the energy and nutrient composition of the diet was calculated through the use of software developed by the Epidemiology and Prevention Unit, Fondazione IRCCS, Istituto Nazionale dei Tumori, Milan, and linked to the Food Composition Database for Epidemiological Studies in Italy of the European Institute of Oncology (Salvini et al. 1998), the Food Composition Tables of the National Institute of Research on Food and Nutrition (INRAN) (Carnovale and Marletta 2000) and the International Tables of Glycemic Index (Foster-Powell et al. 2002). The intake of polyphenols was evaluated using the "USDA Special Interest Database on Flavonoids" in combination with the "Phenol Explorer Database". The amount of advanced glycation end-products (AGEs) was estimated according to the database provided by Urribarri et al. (Uribarri et al. 2005), containing information on both foods and cooking methods. Dietary AGEs were calculated by multiplying the content of AGE by the daily consumption of each food and expressed as AGE kilounits/100 g food.

The adherence to the Mediterranean Diet was evaluated with the rMED score (relative Mediterranean Diet score), proposed by Buckland (Buckland et al. 2009). The details of this calculation have been described in detail in a prior publication (Vitale et al. 2018). Briefly, tertiles of the average daily intake of 9 key food groups (fruits, vegetables, legumes, cereals, fish, olive oil, meat and meat products, dairy products, and alcohol) expressed in grams/1000 Kcal (except alcohol) have been calculated. For the six food groups fitting the Mediterranean model (fruit, vegetables, legumes, cereals, fish and seafood and olive oil), we assigned a score of 0, 1 or 2 to the first, second or third tertile, respectively. For meat and dairy products, we assigned a score of 0, 1 or 2 to the third, second and first tertile, respectively. Alcohol was scored as dichotomous variable, therefore, we assigned 2 points for moderate intake (5–25 g/day for women and 10–50 g/day for men) and 0 points for a consumption at or below the sex-specific range. The rMED score in the study population ranged from 0 to 18.

Computation of the dietary inflammatory index (DII®)

The development of the DII has been described in detail elsewhere (Shivappa et al. 2014; 2018b). The inflammatory potential of the diet was calculated by computing the amounts of nutrients provided by the food frequency questionnaire. The mean intake of each food item was translated into statistical indices (*Z*-scores, which were converted to centred proportions), which were multiplied by their respective coefficients (overall food parameter-specific inflammatory effect scores) to calculate the DII score for each food parameter. Scores were summed to obtain the overall DII score for each participant. A positive DII score corresponds to a pro-inflammatory dietary habit and a negative DII score translates to an anti-inflammatory dietary habit. The following 22 food parameters were available to calculate the DII in the present study: total energy, carbohydrates, fat, protein, cholesterol, saturated fatty acids, monounsaturated fatty acids, polyunsaturated fatty acids, n-3 fatty acids, n-6 fatty acids, vitamin B12, vitamin B6, vitamin A, vitamin C, vitamin D, vitamin E, riboflavin, thiamine, iron, folic acid, zinc, niacin, fibre. Energy-adjusted DII (E-DII) scores were calculated using the density approach by calculating DII per 1000 kcal consumption, which entailed using the energy-adjusted global comparison database (Hébert et al. 2019). As energy was controlled by division, the E-DII relied on 21 food parameters (all those listed above minus energy) All results presented are based on the E-DII score.

Statistical analysis

Univariate statistics were computed as mean and standard deviation (M ± SD) for continuous variables or number and percentages for categorical variables, as appropriate.

Sex-specific quartiles were created for the E-DII. The relation of E-DII score with energy and nutrient composition of the diet, anthropometric variables, blood pressure, glucose control, and plasma lipids, was explored by stratifying the study population according to quartiles of sex-specific E-DII score (i.e. the score was lowest in the first quartile and highest in the last quartile). To compare variables across the quartiles, the analysis of variance and the χ^2 test was used to compare means and proportions, respectively. A *post-hoc* test that corrected for multiple comparisons (Bonferroni test) also was used in order to compare the highest quartile (4) vs. the lowest (1) quartile. Statistical analyses were performed with SPSS version

Table 1. General characteristics of the study participants according to sex-specific quartiles of Energy-adjusted Dietary Inflammatory Index (E-DII) scores.

DII score	Quartile 1 (N = 642) −2.0 ± 0.6 (−4.19; −1.12)	Quartile 2 (N = 642) −0.5 ± 0.3 (−1.11; 0.09)	Quartile 3 (N = 642) 0.6 ± 0.3 (0.09; 1.17)	Quartile 4 (N = 642) 2.0 ± 0.7 (1.17; 5.19)	*p* Value
Age (years)	62.2 ± 6.5	62.3 ± 6.4	62.4 ± 6.4	61.6 ± 6.6	.13
Gender					
Male (%)	303 (47.2)	361 (56.2)	417 (65.0)	453 (70.6)	<.0001
Female (%)	339 (52.8)	281 (43.8)	225 (35.0)	189 (29.4)	
Smokers (%)	94 (14.6)	107 (16.7)	109 (17.0)	134 (20.9)	.001
Education					
Secondary/university (%)	205 (31.9)	208 (32.4)	201 (31.3)	205 (31.9)	.79
None/primary (%)	437 (68.1)	434 (67.6)	441 (68.7)	437 (68.0)	
Marital status					
Married or cohabitants (%)	555 (86.5)	547 (85.2)	548 (85.2)	531 (82.8)	.39
Divorced, single or widowed (%)	87 (13.5)	95 (14.8)	95 (14.8)	111 (17.3)	

26.0. Statistical significance was assessed as $p < 0.05$ (two-sided).

Results

The study population consists of 2568 people with T2DM; 1534 men and 1034 women. Average age was 62.1 ± 6.5 years, BMI was 30.3 ± 4.5 kg/m^2 and duration of diabetes was 8.5 ± 5.7 years. All study participants were treated with metformin at the dosage of 2 g/day. The E-DII score ranged between −4.19 (maximally anti-inflammatory diet) and +5.19 (maximally pro-inflammatory diet). The general and socio-demographic characteristics of the study population are reported in Table 1 according to the quartiles of E-DII score. Age, educational level, and marital status was not significantly different across the quartiles of E-DII score whereas male gender and current smoking were significantly more frequent among people in the upper quartile of E-DII.

Table 2 describes the energy and nutrient composition of the diet across the quartiles of E-DII score. A higher inflammatory index score (Quartile 4) was associated with significantly higher energy intake, higher consumption of total carbohydrates, added sugars, saturated fat, cholesterol, alcohol, and a significantly lower intake of unsaturated fat (mono- and poly-unsaturated ω-3 and ω-6 fatty acids) fibre and total polyphenols compared to the lower quartile (Quartile 1). The intake of total proteins was not significantly different across quartiles of E-DII score, but the intake of plant proteins significantly decreased with increasing E-DII score. The AGE content of the diet increased significantly from Q1 to Q4, whereas the Mediterranean Dietary Score significantly decreased.

The analysis of food groups showed that people with a higher E-DII score (Quartile 4) consumed substantially less fruit, vegetables, legumes, fish, and olive oil, and more cereals, meat, dairy products, and alcohol, compared to the lower quartile (Quartile 1) (Table 3).

Regarding the cardiovascular risk factor profile, waist circumference, diastolic blood pressure, and plasma triglycerides progressively and significantly increased across quartiles of E-DII score, whereas a significant reduction was observed for HDL-cholesterol (Table 4); higher values for LDL-cholesterol and systolic blood pressure were also observed in the upper E-DII quartile, but the difference did not reach statistical significance. A high proportion of the cohort was taking lipid lowering medications (62.0%) and antihypertensive medications (92.5%), with no significant differences across quartiles of E-DII score.

Discussion

To our knowledge, this is the first study documenting the inflammatory potential of the diet in people with T2DM and its relationship with the cardiovascular risk factors profile. The E-DII score in the study population ranged from −4.19 to +5.19, which is similar to what was recently reported in one other Italian population (Shivappa et al. 2018a). Therefore, this suggests that, contrary to the expectations, the overall inflammatory potential of the habitual diet in people with T2DM is not different from that reported for the general population. Although in line with the general notion that adherence to the dietary recommendations in people with T2DM is low, the finding is nonetheless relevant. The detrimental effects of a proinflammatory diet on cardiovascular health have been largely attributed to its effects on systemic inflammation; however, a high inflammatory score also is a potent marker of a generally poor quality of the diet, which may further contribute to the

Table 2. Energy and nutrient composition of the habitual diet followed by study participants according to sex-specific quartiles of Energy-adjusted Dietary Inflammatory Index (E-DII) scores.

E-DII score	Quartile 1 (N = 642) −2.0 ± 0.6 (−4.19; −1.12)	Quartile 2 (N = 642) −0.5 ± 0.3 (−1.11; 0.09)	Quartile 3 (N = 642) 0.6 ± 0.3 (0.09; 1.17)	Quartile 4 (N = 642) 2.0 ± 0.7 (1.17; 5.19)	*p* Value
Total energy (kcal/day)	1648.3 ± 528.7	1785.9 ± 587.2	1992.5 ± 663.6	2208.7 ± 783.6[a]	<.0001
Proteins (% of TE)	18.3 ± 2.6	18.2 ± 2.5	18.3 ± 2.5	18.1 ± 2.4	.10
Animal protein (% of TE)	12.7 ± 3.1	12.4 ± 3.0	12.6 ± 3.1	12.6 ± 3.1	.77
Vegetable protein (% of TE)	5.7 ± 1.0	5.8 ± 1.1	5.7 ± 1.2	5.5 ± 1.2[a]	.004
Lipids (% of TE)	38.4 ± 6.1	36.1 ± 6.2	35.9 ± 5.6	36.2 ± 5.7[a]	<.0001
Saturated fat (% of TE)	11.6 ± 2.1	11.8 ± 2.5	12.3 ± 2.4	13.0 ± 2.7[a]	<.0001
Monounsaturated fat (% of TE)	19.6 ± 4.2	17.7 ± 3.7	17.2 ± 3.3	16.9 ± 3.1[a]	<.0001
Polyunsaturated fat (% of TE)	4.9 ± 1.4	4.5 ± 1.1	4.2 ± 0.9	4.1 ± 0.8[a]	<.0001
Omega 3 (% of ET)	0.60 ± 0.12	0.55 ± 0.10	0.53 ± 0.10	0.51 ± 0.09[a]	<.0001
Omega 6 (% of ET)	3.9 ± 1.3	3.6 ± 1.0	3.4 ± 0.8	3.2 ± 0.7[a]	<.0001
Cholesterol (mg/die)	284.2 ± 114.3	303.5 ± 131.6	340.5 ± 134.9	384.4 ± 169.1[a]	<.0001
Carbohydrates (% of TE)	43.2 ± 7.0	45.6 ± 7.4	45.7 ± 7.0	45.7 ± 7.1[a]	<.0001
Added sugars (% of TE)	2.1 ± 2.7	2.5 ± 3.5	2.5 ± 3.1	2.5 ± 3.5[a]	.03
Fibre (g/1000 kcal/day)	13.3 ± 2.5	11.5 ± 2.0	9.8 ± 1.7	8.3 ± 1.6[a]	<.0001
Alcohol (g)	6.7 ± 9.8	10.2 ± 14.2	13.0 ± 16.8	14.5 ± 19.8[a]	<.0001
Total polyphenols (mg/1000 kcal/day)	404.9 ± 147.2	350.0 ± 140.2	294.0 ± 117.8	242.2 ± 109.3[a]	<.0001
AGE[b] (KU/dl)	7432 ± 2134	10,674 ± 2099	17 987 ± 3212	23,215 ± 3567[a]	<.0001
rMED score	9.4 ± 2.6	8.9 ± 2.7	8.7 ± 2.8	8.1 ± 2.7[a]	<.0001

M ± SD or %.
[a]Post-hoc test corrected for multiple comparisons (Bonferroni test).
[b]Advanced glycated end-products.

Table 3. Food groups consumption (expressed as g/1000 kcal/day) according to the sex-specific quartiles of Energy-adjusted Dietary Inflammatory Index (E-DII) scores.

E-DII score	Quartile 1 (N = 642) −2.0 ± 0.6 (−4.19; −1.12)	Quartile 2 (N = 642) −0.5 ± 0.3 (−1.11; 0.09)	Quartile 3 (N = 642) 0.6 ± 0.3 (0.09; 1.17)	Quartile 4 (N = 642) 2.0 ± 0.7 (1.17; 5.19)	*p* Value
Fruit	233.8 ± 102.9	178.5 ± 77.1	137.7 ± 57.2	89.9 ± 47.0[a]	<.0001
Nuts	0.7 ± 1.6	0.6 ± 1.1	0.6 ± 1.3	0.4 ± 1.0[a]	.002
Vegetables	138.6 ± 52.8	100.0 ± 36.4	78.1 ± 32.5	61.4 ± 27.5[a]	<.0001
Legumes	19.1 ± 13.6	14.4 ± 11.1	11.2 ± 8.1	8.7 ± 6.9[a]	<.0001
Cereals	79.7 ± 29.8	95.7 ± 34.4	100.0 ± 35.2	104.8 ± 38.8[a]	<.0001
Fish	28.6 ± 19.3	22.8 ± 15.3	21.6 ± 16.2	17.2 ± 12.7[a]	<.0001
Olive oil	18.0 ± 6.3	14.0 ± 5.1	12.1 ± 4.5	10.4 ± 4.3[a]	<.0001
Meat	54.5 ± 26.4	51.5 ± 23.8	51.0 ± 23.8	50.6 ± 23.9[a]	.005
Processed meat	11.2 ± 7.7	13.8 ± 9.7	15.4 ± 9.9	17.9 ± 11.1[a]	<.0001
Dairy products	17.9 ± 11.9	19.5 ± 12.3	21.0 ± 12.7	22.5 ± 14.7[a]	<.0001
Alcohol	45.4 ± 63.3	64.0 ± 83.1	75.8 ± 91.1	74.6 ± 95.8[a]	<.0001

[a]*Post-hoc* test corrected for multiple comparisons (Bonferroni test).

Table 4. Cardiovascular risk factors profile according to the sex-specific quartiles of energy-adjusted Dietary Inflammatory Index (E-DII) score.

E-DII score	Quartile 1 (N = 642) −2.0 ± 0.6 (−4.19; −1.12)	Quartile 2 (N = 642) −0.5 ± 0.3 (−1.11; 0.09)	Quartile 3 (N = 642) 0.6 ± 0.3 (0.09; 1.17)	Quartile 4 (N = 642) 2.0 ± 0.7 (1.17; 5.19)	*p* Value
BMI (Kg/m^2)	30.2 ± 4.5	30.3 ± 4.2	30.5 ± 4.6	30.1 ± 4.3	.94
Waist circumference (cm)	103.5 ± 11.7	104.4 ± 10.6	105.0 ± 11.3	105.4 ± 11.1[a]	.002
Systolic blood pressure (mmHg)	132.4 ± 14.2	135.1 ± 24.9	134.3 ± 14.7	134.3 ± 14.1	.06
Diastolic blood pressure (mmHg)	78.6 ± 8.2	79.6 ± 8.1	80.0 ± 8.8	80.0 ± 8.9[a]	.04
HbA1c (%)	7.67 ± 0.50	7.63 ± 0.48	7.67 ± 0.51	7.69 ± 0.50	.26
LDL-cholesterol (mg/dL)	100.9 ± 30.6	104.9 ± 32.0	103.7 ± 30.8	103.3 ± 31.3	.06
HDL-cholesterol (mg/dL)	47.4 ± 12.4	46.0 ± 11.7	45.1 ± 11.5	45.3 ± 11.3[a]	.04
Non-HDL-cholesterol (mg/dL)	130.5 ± 37.2	135.0 ± 37.6	134.5 ± 34.1	133.7 ± 36.2	.15
Triglycerides (mg/dL)	146.1 ± 71.1	148.8 ± 72.5	153.5 ± 73.4	156.4 ± 81.3[a]	.005
% on lipid lowering medications	61.8	62.0	62.3	62.1	.66
% on antihypertensive medications	92.2	92.6	92.4	92.7	.72

M ± SD or %.
[a]*Post-hoc* test corrected for multiple comparisons (Bonferroni test).

detrimental health effects. In particular, in our population a diet with a high E-DII score is characterised by a high intake of saturated fat and added sugars and a low intake of fibre, polyunsaturated fat and

bioactive compounds with beneficial health properties such as polyphenols. We also document a positive association of E-DII with the AGE content of the diet, which suggests significant differences not only in the food choices, but also in the method of cooking. Furthermore, the higher proportion of smokers, and the lower Mediterranean dietary score in people with the highest E-DII highlight the association of the E-DII with a generally unhealthy lifestyle, which may further enhance the adverse metabolic effects of a proinflammatory diet. As for the relation of the E-DII with major cardiovascular risk factors profile we found that an increasing inflammatory potential of the diet was associated with the metabolic features commonly found with insulin resistance (i.e. higher waist circumference, triglycerides and blood pressure and lower HDL-cholesterol). These results are in line with most, though not all, previous studies in people without diabetes and highlight the concept that the effects of a proinflammatory diet may extend beyond the markers of systemic inflammation. The Supplémentation en Vitamines et Minéraux AntioXydants (SU.VI.MAX) study conducted in a cohort of 3726 subjects, demonstrated that a pro-inflammatory diet was associated with higher triglyceride and lower HDL-C levels (Neufcourt et al. 2016). Similarly, Sokol et al. reported an inverse relationship between the DII and HDL-C in a Polish population of 3862 participants, but no differences for the other metabolic parameters (Sokol et al. 2016). More recently, adverse metabolic features, including greater numbers of LDL-particles have been reported (Phillips et al. 2018). Our study confirms these findings and further expands knowledge by documenting in people with T2DM the relationship of the inflammatory dietary score with the overall quality of the diet, a generally unhealthy lifestyle and the cardiovascular risk factors profile, in particular the metabolic features associated with insulin resistance.

We do acknowledge that due to the cross-sectional design the temporality criterion of the Criteria for Judging Causality could not be met, thus limiting causal inference (Hill 1965). Second, as the dietary data used to calculate E-DII are self-reported, a certain degree of misclassification of exposure is possible and potential measurement error cannot be ruled out. However, we used validated tools in order to minimise the potential measurement error in the usual diet, and people with diets involving extreme energy intakes were excluded. In any case, any misclassification of exposure is likely to be non-differential and would result in an attenuation of associations. Furthermore, the E-DII score was calculated based on 22 of the 45 nutritional parameters on which the original E-DII was constructed. The dietary components not included, however, are not commonly consumed (Tabung et al. 2015). In addition, the heavy use of medications (antihypertensive and lipid lowering drugs) may have partly offset the impact of the DII on some CVD risk factors such as LDL-cholesterol).

Despite its weaknesses, the present study has several strengths. It is the first to document the inflammatory potential of diet, as measured by the DII, and its relationship with the metabolic profile in a large population with T2DM over and above the use of medications. Second, this study draws on a relatively large population. Third, it adds to the literature by examining this relationship in the context of a study with good exposure data for T2DM-related drugs.

In summary, these novel results provide evidence that the E-DII is a marker of the overall quality of the diet and with an unfavourable CVD risk factor profile, thus suggesting that the potential of a diet with a low inflammatory score extends beyond its anti-inflammatory effects and may be useful in achieving and maintaining a more favourable cardiovascular risk factors profile. This is particularly relevant in people with type 2 diabetes and insulin resistance who are at very high CVD risk.

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Disclosure statement

Dr. James R. Hébert owns controlling interest in Connecting Health Innovations LLC (CHI), a company that has licenced the right to his invention of the dietary inflammatory index (DII®) from the University of South Carolina in order to develop computer and smart phone applications for patient counselling and dietary intervention in clinical settings. Dr. Nitin Shivappa is an employee of CHI.

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