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EAT-Lancet Healthy Reference Diet score and diabetes incidence in a cohort of Mexican women

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To improve the health of our planet and develop sustainable food policies, it is important to understand the health impact of a diet pattern that considers planetary and population health. We used data from the Mexican Teachers' Cohort (MTC) to estimate the association between the EAT-Lancet healthy reference diet (EAT-HRD) and type 2 diabetes (T2D) incidence. We included 74,671 women aged ≥25 years, free of T2D at baseline. A validated food frequency questionnaire (FFQ) was used to assess dietary intake. We created an EAT-HRD score based on 15 food groups recommended by the EAT-Lancet Commission (range from 0 to 15). T2D cases were identified through self-report and cross-linkage with clinical and administrative databases. We used Cox proportional hazards models to estimate the association between categories of the EAT-HRD score with T2D incidence. During a median follow-up of 2.16 y (IQR 1.8–4.3 y), we identified 3241 T2D incident cases. The median EAT-HRD score was 6 (IQR 5–7). In multivariable analyses, when comparing extreme categories, higher adherence to the EAT-HRD score was associated with lower T2D incidence (HR 0.90; 95% CI 0.75, 1.10), yet, the estimation was imprecise. Compared to those who did not meet the EAT-HRD recommendations, adhering to the red meat, legumes, and fish recommendations was associated with lower T2D incidence. Meeting the recommendation of dairy and added sugars was associated with an increased incidence of T2D. Higher adherence to a diet designed to promote environmental and human health may help prevent T2D incidence among women in a middle-income country.

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BACKGROUND

A suboptimal diet is an important preventable risk factor for chronic diseases. The proportion of type 2 diabetes (T2D) deaths attributable to unhealthy diets has been estimated to be 32.5% [1]. The risk of T2D incidence increases up to 30% when consuming food groups such as red and processed meats [2], and sugar-sweetened beverages [3, 4]. High intakes of fruits, [5, 6] green leafy vegetables [7, 8], and whole grains [9, 10] have shown to have a protective effect against T2D incidence. Increasing the consumption of whole fruits to 3 servings a day has also been associated with a reduced risk of T2D [6, 7]. Consistently, studies from the US and Europe have found that vegetable consumption is not associated with T2D risk, however, increasing one serving per day of leafy green vegetables reduces the risk of T2D by up to 13% [5, 7, 8, 11–13]. Diets are also a key link between population health and environmental sustainability. Evidence suggests that the food system contributes up to 29% of all greenhouse gas emissions [14–16]. Thus, reducing the environmental impact of diet and improving diet quality is a main global priority [17]. While multiple studies have assessed the impact of diet on environmental susceptibility [18–24] or the effects of diet on health, very few studies have studied the impact of a sustainable diet on health [19, 25–27].

In 2019, the EAT-Lancet commission published recommendations for a "Healthy Reference Diet" (EAT-HRD), promoting a plant-based diet, with low amounts of animal-based foods, unsaturated rather than saturated fat consumption, and with limited amounts of added sugars, refined grains, and processed foods [22]. Although the daily EAT-HRD recommendations were based on the health literature and the environmental impact of each food group [25], there is limited evidence on whether higher adherence to these recommendations prevents chronic diseases in different contexts [26, 28, 29]. This is especially true in low- and middle-income countries (LMIC), where the potential impact of climate change on health is higher, compared to high-income countries [30, 31]. As the economies of LMIC expand [32, 33], people tend to

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increase their meat consumption [34]. In fact, red meat is the highest contributor to dietary greenhouse gas emissions in many Latin American countries, including Mexico [35].

To improve the health of our planet and develop sustainable food policies, it is important to understand the health impact of a dietary pattern that considers planetary and population health. Thus, we aimed to estimate the association between the EAT-HRD and the incidence of T2D among Mexican women.

RESULTS

In 203,902 person-years of follow-up (median 2.2 years, IQR 1.8–4.3), we identified 3241 incident T2D cases (unadjusted incidence rate, 159 per 10,000 person-years). The median EAT-HRD score was 6 (IQR 5–7) and ranged from 0–13. Compared to women categorized in the lowest EAT-HRD category, women in the highest category were older, had higher physical activity levels, were less likely to be current smokers, more likely to be in the lowest SES category, and had a higher proportion of postmenopausal women (Table 1). From the 15 EAT-HRD food groups, the recommendations were met by 50% or more women for only 6 groups: other vegetables (57.7%); fruits (77.1%); tubers (80.5%); milk (59.2%); eggs (65.2%); and added fats (72.9%) (Supplementary Table 1).

Compared with the lowest EAT-HRD category (score 0–4), women in the highest EAT-HRD category (score 9–13) had a lower T2D incidence (HR = 0.90; 95% CI 0.75, 1.10), after adjusting for confounders. However, there was a lot of uncertainty in the point estimate (Table 2). We also found a lower incidence of T2D among women who met the EAT-HRD recommendation of red meat (HR = 0.79; 95% CI 0.63, 0.99), compared to those who did not meet the recommendation. A small protective association was also found for legumes (HR = 0.92; 95% CI 0.84, 0.99), and fish (HR = 0.92; 95% CI 0.85, 1.00). In contrast, meeting the EAT-HRD recommendation for dairy (HR = 1.12; 95% CI 1.04, 1.21), and added sugars (HR = 1.11; 95% CI 1.02, 1.21), versus not meeting the recommendation, was associated with an increased incidence of T2D (Table 3).

We conducted several sensitivity analyses and found that the results did not change when we used a different outcome definition: women who responded having a medical diagnosis of diabetes and who were under medical treatment (HR = 0.90; 95% CI 0.74, 1.11, comparing women with a score of 9–13 vs 0–4) (Supplementary Table 2). When we restricted our population to women whose health care provider was ISSSTE (HR = 0.95; 95% CI 0.76, 1.20, comparing women with a score of 9–13 vs 1–4) (Supplementary Table 3); or when we additionally adjusted for BMI (HR = 0.94; 95% CI 0.78, 1.14 comparing women with a score of 9–13 vs 0–4), the association was weaker and closer to the null (Supplementary Table 4). When additionally adjusting for hypertension (HR = 0.91; 95% CI 0.75, 1.10, comparing women with a score of 9–13 vs 0–4) our results did not change (Supplementary Table 4). With an observed hazard ratio of HR = 0.90, an unknown confounder that was associated with both the outcome and the exposure by a risk ratio of 1.46-fold each (E-value), conditional on the measured confounders, could explain away the estimate, but weaker joint unmeasured confounding could not.

DISCUSSION

In this cohort of Mexican women, we found a protective, but imprecise association with T2D incidence when comparing higher vs lower adherence to the EAT-HRD score. Additionally, women who met the EAT-HRD recommendations for red meat, legumes, and fish had a lower incidence of T2D. Contrary to what we expected, meeting the added sugars recommendation (<31 g/d) was associated with higher T2D incidence.

Different biological mechanisms may explain the benefits of plant-based diets for T2D prevention, such as improvement of postprandial glucose, enhanced glycemic control, improvement in insulin sensitivity, and decrease in chronic inflammation. Yet, to achieve these benefits, a diet must be rich in dietary fiber, antioxidants, unsaturated fatty acids, and micronutrients such as magnesium, and low in saturated fat [9, 36–38].

Few studies have evaluated the association between the EAT-HRD recommendations with health outcomes. Yet, evidence suggests that higher adherence to the EAT-Lancer diet may be beneficial to our health. In a cross-sectional analysis of the baseline characteristics of the ELSA-Brasil cohort (2008), those with high adherence to the EAT-Lancet recommendations had lower BMI (−0.50 kg/m^2; 95% CI −0.73, −0.27) and WC (−1.70 cm; 95% CI −2.28, −1.12) values, compared to participants with lower adherence, and were also less likely to be overweight or obese [39]. One recent study from a Swedish cohort, with ~20 years of follow-up, found a 25% decreased rate in all-cause mortality, 24% decreased rate in cancer mortality, and 32% decreased rate in cardiovascular disease mortality when comparing people with the highest adherence to the EAT-Lancet diet vs those with the lowest adherence [29]. Another study from the EPIC cohort estimated that up to 63% of deaths and 39% of incident cancers could be prevented in a 20-year risk period by fully adopting the EAT-Lancet diet, compared to not adopting the EAT-HRD diet [26]. Adherence to the EAT-Lancet diet in a Danish cohort has also been associated with a lower risk of subarachnoid hemorrhage stroke (HR = 0.30; 95% CI 0.12, 0.73) in a 15-year risk period [40]. Two previous studies have investigated the EAT-Lancet and T2D association. The EPIC-Oxford cohort study, with ~18-year follow-up found that people with the highest EAT-HRD score had a 59% decrease in the rate of T2D when compared to people with the lowest score [28]. Another study from the UK Biobank found that adherence to the EAT-Lancet diet was associated with a 19% decrease rate of T2D in a ~10-year risk period when comparing extreme tertiles [41]. Compared to these studies, our results show a smaller protective association for T2D. However, the difference in the magnitude of the association could be explained by differences in the way the score was created. Different scoring systems derived from different interpretations of the Eat-Lancet Commission recommendations could yield different results when studying the association between adherence to a plant-based diet and disease outcomes. Differences can also be attributed to the foods that make up each of the EAT-HRD food groups, and differences in the background diet of each population. Our results are also consistent with the protective associations observed in studies evaluating the relationship between a plant-based diet and T2D incidence [9, 36, 38].

Meeting the recommendation of limiting red meat consumption to ≤14 g/d was associated with lower T2D incidence, compared with those who did not meet this recommendation. Evidence from other cohort studies shows that lowering the consumption of red meat while adhering to a plant-based diet, was found to decrease T2D incidence [2, 36]. Meat production is the single most important source of methane and livestock production, and it is responsible for ~15% of all anthropogenic carbon dioxide emissions [42]. Thus, lowering red meat consumption worldwide will be key to meet the environmental sustainability goals. It is important to point out that only 2.7% of the MTC participants met the red meat recommendations, highlighting the opportunity to make the Mexican diet healthier, more sustainable, and climate-friendly. Fish consumption was also found to be protective against T2D incidence. While fish is an encouraged food by the EAT-Lancet, a potential conflict between dietary intake recommendations and sustainability may exist, depending on the type of fish consumed and the production methods used [43].

We also found that meeting the recommendations of dairy consumption was associated with an increased risk of T2D. This

Table 1. Baseline characteristics of women in the Mexican Teachers' Cohort by EAT-HRD score categories ($n = 74,671$)[a].

	EAT-HRD categories			
	0–4 ($n = 14,752$)	**5–6 ($n = 34,372$)**	**7–8 ($n = 20,904$)**	**9–13 ($n = 3091$)**
Age at baseline, years	40.9 (7.2)	42.0 (7.2)	42.9 (7.0)	43.7 (5.9)
Calorie intake, kcal/d	1816 (573)	1800 (635)	1836 (636)	1835 (642)
Alcohol consumption, g/d	1.0 (2.1)	1.1 (2.0)	1.1 (2.1)	1.1 (2.5)
Menopause status				
Pre-menopause	79.1	75.5	71.6	68.6
Post-menopause	12.1	15.1	17.4	20.3
Unknown	8.7	9.4	11.0	11.1
Family history of diabetes				
Yes	46.6	46.7	46.1	43.8
No	40.9	39.0	38.0	37.3
Missing	12.4	14.3	15.9	18.9.
Smoking				
Current	9.6	9.0	8.4	7.9
Past	12.9	12.0	11.2	10.3
Never	74.5	75.6	76.4	77.8
Missing	3.0	3.3	4.0	4.0
Physical activity, METs/wk[b]				
Tertile 1	33.0	31.4	29.3	28.8
Tertile 2	34.9	34.1	34.2	32.7
Tertile 3	31.3	33.5	35.3	36.9
Missing	0.8	1.0	1.2	1.6
SES[c]				
Tertile 1	22.6	26.8	29.7	31.9
Tertile 2	33.8	32.7	31.0	31.1
Tertile 3	43.6	40.4	38.3	37.0
Region[d]				
North	20.6	18.6	17.4	17.0
Center	18.3	23.8	28.1	29.4
South	36.3	33.4	33.1	31.7
CDMX	24.7	24.2	21.4	21.8
BMI, kg/m				
Normal	33.9	32.4	31.5	32.6
Overweight	37.0	38.2	38.9	39.0
Obesity	21.4	20.9	20.0	18.6
Missing	7.6	8.5	9.6	9.8
Hypertension[e]	13.4	13.6	14.2	15.4
Dietary intake, servings/day[f]				
Whole grains	0.2 (0.11)	0.2 (0.11)	0.2 (0.11)	0.2 (0.13)
Tubers	0.7 (0.82)	0.7 (0.85)	0.8 (0.90)	0.8 (1.0)
Legumes	0.8 (0.80)	1.1 (1.1)	1.5 (1.31)	2.0 (1.62)
Nuts and seeds	0.1 (0.17)	0.1 (0.08)	0.2 (0.23)	0.3 (0.4)
Dark green vegetables	0.2 (0.19)	0.1 (0.20)	0.2 (0.26)	0.6 (0.65)
Orange or red vegetables	0.3 (0.26)	0.3 (0.27)	0.6 (0.49)	0.9 (0.66)
Other vegetables	1.0 (0.85)	1.4 (1.17)	2.0 (1.5)	2.9 (2.2)
Fruits	1.7 (1.57)	2.2 (1.70)	2.7 (1.88)	3.4 (2.2)
Dairy	1.3 (1.00)	1.0 (0.93)	0.8 (0.81)	0.6 (0.62)
Eggs	1.7 (1.44)	1.2 (1.25)	0.8 (0.98)	0.6 (0.63)
Poultry	1.1 (0.68)	0.9 (0.68)	0.7 (0.64)	0.6 (0.68)

Table 1. continued

	EAT-HRD categories			
	0–4 ($n = 14,752$)	**5–6 ($n = 34,372$)**	**7–8 ($n = 20,904$)**	**9–13 ($n = 3091$)**
Red meat	4.5 (2.39)	4.2 (2.40)	3.9 (2.37)	3.2 (2.23)
Fish	0.7 (0.60)	0.9 (0.77)	1.1 (1.0)	1.4 (1.30)
Added sugars	2.0 (1.39)	1.8 (1.36)	1.6 (1.29)	1.3 (1.14)

[a]Values are mean ± SD or column percentages.
[b]Physical activity category; tertile 1: 9.00 (5.00–15.00), tertile 2: 22.00 (18.50–31.75), tertile 3: 54.50 (41.50–125.50).
[c]Household assets (phone, car, computer, vacuum cleaner, microwave oven, cell phone, and internet), SES category; tertile 1: 2.00 (1.00–3.00), tertile 2: 5.00 (4.00–5.00), tertile 3: 6.00 (6.00–7.00).
[d]North: Baja California, Durango, Nuevo León, Sonora; Center: Guanajuato, Hidalgo, Jalisco; South: Chiapas, Veracruz, Yucatán; CDMX: Mexico City and State of Mexico.
[e]Previously diagnosed cases. Self-reported at baseline.
[f]Serving per day of each group. Servings are calculated according to the EAT-HRD recommendations.

association has been widely studied [44–47]. The body of evidence suggests that low-fat dairy and yogurt consumption decreases the risk of T2D, while high-fat dairy increases T2D. The EAT-HRD score combines both, high and low-fat dairy, which could explain why dairy consumption increases the T2D incidence in our population. Contrary to what we expected, we also found that meeting the recommendation to limit added sugar consumption to 31 g/d increases T2D incidence. However, this result is consistent with the EAT-HRD-diabetes UK Biobank study, which also found that adhering to the added sugar recommendation was associated with an 85% increased rate of T2D [41]. Moreover, the association between T2D incidence and sugar consumption seems to be unclear in other studies; with two meta-analyses showing no association [4, 47].

One of the main goals of the EAT-HRD commission is to adapt the recommendations to the foods consumed at the regional and local levels. Efforts have been made in different countries such as India [48], Italy [49], Denmark [50], and Australia [51]. Except for India, these studies have successfully adapted available foods in their region to meet the EAT-HRD recommendations. When comparing national dietary guidelines from all countries, they have found that none of them consider the sustainability of food systems. To achieve the United Nations' Sustainable Development Goals, countries need to be able to deliver nutritious diets while minimizing the negative environmental impacts of diet. Recently, the EAT-Lancet recommendations were adapted to the Mexican diet [52], however, no study has evaluated whether this adaptation is also associated with a lower risk of chronic diseases.

When adapting the EAT-HRD recommendations, affordability also needs to be considered. Using data from 159 countries of different income levels, Hirvonen et al. analyzed the cost of adapting the EAT-HRD recommendations using the most affordable foods in the different countries [53]. In their study, the average cost for the EAT-HRD diet was US$2.84 per day. Although this cost is not affordable for the world's lower-income countries, for Mexico, on average, the cost of the EAT-HRD was 29% lower than the Mexican current food basket (main products and foods purchased by an average Mexican family). The cost of the EAT-HRD was USD 2.6 per day per person, while the Mexican current food basket costs USD 3.65 [54].

The strengths of the study include its prospective design, large sample size, adequate follow-up (83%), access to clinical data to identify T2D cases, and extensive information on risk factors for T2D. However, our study is not without limitations. Each component of the EAT-HRD score was constructed as a binary variable (adherence vs non-adherence) using the Eat-Lancet Commission recommended target as the cut-off. A limitation of this scoring system is that it categorizes participants who are almost adherent to the recommended targets together with participants who are far from adhering to the recommended targets. Additionally, our score did not consider the food group bounds reported in the original Eat-Lancet paper and it was not constructed to include proportional scores. However, perfect adherence to our score reflects a diet that is low in red meat, dairy, poultry, tubers, added sugars, and animal fat, and high in whole grains, fruits, vegetables, fish, and plant-based protein. Future studies should evaluate whether different scoring systems derived from the Eat-Lancet recommendations result in different associations with T2D incidence. As with every observational study, unmeasured confounding is always a possibility. However, we found that the minimum strength of association that an unmeasured (or residual) confounder would need to have with both, the exposure and the outcome, to fully explain the observed association was 1.46. Although diet was measured using a validated FFQ, measurement error of food groups is possible. However, given the prospective design, it is unlikely for this measurement error to be differential by disease status. Non-differential misclassification of diabetes is a possibility, especially if some cases are undiagnosed. Nevertheless, if the proportion of undiagnosed is small, the misclassification is likely to have only a minimal impact on the estimates. We excluded women who did not respond to the follow-up questionnaire (~10%), which could result in selection bias. However, we did not observe important differences in socio-demographic and lifestyle characteristics when we compared the baseline characteristics of our analytical sample to the entire cohort and to women who were lost to follow-up (Supplementary Table 5). Finally, all participants were female teachers, increasing internal validity, but may compromise generalizability and transportability to men. Nevertheless, it is unlikely that cohort participants differ biologically from the rest of the women in Mexico. Moreover, the distribution of characteristics and risk factors among cohort participants is similar to the general population of women in Mexico.

In conclusion, we found that higher adherence to the EAT-HRD score may help prevent T2D incidence among Mexican women. Moreover, meeting the EAT-HRD recommendations for red meat, legumes, and fish decreased the incidence of T2D. This study supports the need to reduce the consumption of foods that contribute to higher levels of greenhouse gas emissions for both, human and planetary health.

SUBJECTS AND METHODS

Study design and population

The Mexican Teachers' Cohort (MTC) is an ongoing prospective longitudinal cohort that was established between 2006 and 2008. MTC recruited 115,314 female public school teachers from 12 geographically and culturally diverse states in Mexico. Recruitment started in 2006 when 27,979 teachers aged ≥35 years from two Mexican states, responded to a baseline questionnaire. In 2008, the cohort was expanded by recruiting

Table 2. Eat-HRD score and diabetes incidence among Mexican women, MTC (n = 74,671).

	EAT-HRD category			
	0–4 (*n* = 14,752)	**5–6 (*n* = 35,924)**	**7–8 (*n* = 20,904)**	**9–13 (*n* = 3091)**
Person years	40,020	98,412	57,167	8303
Cases	602	1552	954	133
Crude incidence rate/10,000py	150	158	167	160
Age and energy adjusted model, HR (95% CI)[a,b]	1.00 (Ref)	0.99 (0.90–1.09)	0.99 (0.90–1.10)	0.89 (0.74–1.08)
Multivariable adjusted model, HR (95% CI)[a,c]	1.00 (Ref)	0.98 (0.90–1.08)	1.00 (0.90–1.10)	0.90 (0.75–1.10)

[a]Values are hazard ratios (95% confidence interval).
[b]Adjusted for age (continuous) and total energy intake (continuous, kcal/d).
[c]Further adjusted for SES (tertiles of number of assets); family history of diabetes (yes/no); region (categorical); smoking status (never/past/current); physical activity (tertiles of METs hours per week); menopausal status (pre-menopause/post-menopause/unknown); and alcohol intake (continuous, g/d).

Table 3. Eat-HRD food groups and diabetes incidence among Mexican women, MTC (*n* = 74,671).

EAT-HRD food groups/intake recommendation[a]	Multivariable adjusted model, HR (95% CI)[b,c]	Multivariable + food groups adjusted model, HR (95% CI)[b,d]
EAT-HRD food groups with an encouraged consumption recommendation/g/d		
Whole grains/≥232	0.91 (0.13–6.54)	0.71 (0.10–5.07)
Fish/≥28	0.92 (0.85–1.00)	0.92 (0.85–1.00)
Legumes/≥50	0.95 (0.88–1.02)	0.92 (0.84–0.99)
Nuts and seeds/≥50	0.90 (0.68–1.20)	0.91 (0.68–1.22)
Dark green vegetables/≥100	0.88 (0.74–1.05)	0.88 (0.73–1.07)
Orange or red vegetables/≥100	1.04 (0.92–1.18)	1.04 (0.91–1.19)
Other vegetables/≥100	1.03 (0.95–1.12)	1.06 (0.97–1.15)
Fruits/≥200	0.94 (0.86–1.03)	0.97 (0.88–1.06)
EAT-HRD food groups with a limited consumption recommendation/g/d		
Tubers/≤50	0.99 (0.91–1.10)	0.97 (0.89–1.07)
Dairy/≤250	1.13 (1.06–1.23)	1.12 (1.04–1.21)
Eggs/≤13	0.91 (0.84–0.98)	0.94 (0.87–1.01)
Poultry/≤29	0.97 (0.90–1.04)	0.98 (0.91–1.05)
Red meat/≤14	0.77 (0.61–0.96)	0.79 (0.63–0.99)
Added fats	1.07 (0.99–1.16)	1.07 (0.99–1.16)
Added sugars/≤31	1.13 (1.04–1.23)	1.11 (1.02–1.21)

[a]Intake recommendation in grams per day.
[b]Values are hazard ratios (95% confidence interval).
[c]Adjusted for age (continuous, years); total energy intake (continuous, kcal/d); SES (tertiles of number of assets); family history of diabetes (yes/no); region (categorical); smoking status (never/past/current); and physical activity (tertiles of METs hours per week); menopausal status (pre-menopause/post-menopause/unknown); and alcohol intake (continuous, g/d).
[d]Adjusted for age (continuous, years); total energy intake (continuous, kcal/day); SES (tertiles of number of assets); family history of diabetes (yes/no); region (categorical); smoking status (never/past/current); physical activity (tertiles of METs hours per week); menopausal status (pre-menopause/post-menopause/unknown); alcohol intake (continuous, g/d); and mutually adjusted for all food groups of the EAT-HRD score (continuous, g/day).

87,335 women aged ≥25 years from 10 additional states using a similar questionnaire [55]. We updated information on risk factors and disease diagnoses in the 2011–2013 cycle (83% follow-up rate).

For this study, we excluded women with self-reported T2D at baseline (*n* = 6018). We also excluded women with prevalent stroke, heart disease, or cancer (*n* = 2541) because these diagnoses may result in changes in diet. Women with implausible energy intake (<500 or >3500 kcal/d) or an incomplete food frequency questionnaire (FFQ, response to ≤70 items and/or missing staple food section, *n* = 20,274) were also excluded. Finally, we excluded women for whom no information was available after baseline (*n* = 11,483). The final analytical sample included 74,671 women. For women with a questionnaire on both, 2006 and 2008 waves (*n* = 13,165), 2006 was used as their baseline and 2008 as their first follow-up. The study was approved by the National Public Health Institute Research Committee. Response to the baseline questionnaire was considered informed consent.

Diet assessment and computation of the EAT-HRD score

Diet was measured using a 140-item FFQ. Every participant was asked how often, on average, they consumed a specified unit or portion size of each food item during the previous year. Ten frequencies of consumption were possible, ranging from never to ≥6 servings per day. The FFQ was previously validated using four 4-day 24-h recalls and two FFQ (at the beginning and end of the study) in 134 Mexico City female residents in a 12-month study [56]. Pearson correlation coefficients for total energy, carbohydrate, protein, and total fat intake between the FFQ and four 4-day 24-h recalls were 0.52, 0.57, 0.32, and 0.63, respectively.

We created an EAT-HRD score using the recommended targets of the food groups considered in the Eat-Lancet Commission as cut points [22]. We classified the 15 food groups either as encouraged to consume or food groups to limit [57]. Encouraged foods included whole grains, dark green vegetables; red or orange vegetables; other vegetables; fruits; legumes; nuts and seeds; and fish. Limited foods included tubers; dairy foods; eggs; chicken and other poultry; red meat, pork, and lamb; added fats; and added sugars (Supplementary Table 1) [57]. We converted the frequency of consumption of each FFQ item to servings per day and then to grams per day using predefined portion sizes. Participants were assigned one point for meeting each of the recommendations. For encouraged food groups, we assigned a point if women consumed the recommended target or more. In contrast, for food groups where consumption is discouraged, we assigned a point if women consumed equal or less of the recommended target, resulting in possible scores ranging from 0–15. A higher score reflects higher adherence to the EAT-HRD recommendations.

Since our FFQ does not capture the amount of fat added to foods, we used the type of fat used for cooking. We assigned a point when participants reported cooking with no added fat, vegetable, or olive oil, and no point if they cooked with butter, margarine, shortening, or lard. While the EAT-HRD considers the amount of sugar added to foods, such as honey, sugar, and other types of sweeteners, we estimated the total content of added sugars in each women's diet, as the Mexican diet includes a wide range of ultra-processed foods and desserts rich in sugar. We.used the Nutrient Profile Model of The Pan American Health Organization [58], which provides a guideline to determine the proportion of sugar that is added to processed foods. The total amount of sugar of every item in our FFQ was taken from the USDA food-composition database [59], and then estimated the proportion of added sugars. We assigned a point if women consumed less or equal to 31 grams of added sugar per day, and no points if consumption was greater than 31 grams/day.

Assessment of diabetes

Incident T2D cases were identified through self-report or cross-linkage with clinical and administrative databases. Participants were asked about physician-diagnosed diabetes, treatment, and year of diagnosis at baseline and follow-up. We defined an incident case of diabetes when women

responded yes (or provided the year of diagnosis) to two of the three following questions: (1) have you ever been diagnosed with diabetes or high blood sugar by a doctor (excluding pregnancy)?; (2) do you use diabetes treatment?; and (3) year of diabetes diagnosis. While the questionnaire does not specifically ask about T2D, given the age at diagnosis, it can be assumed that it is T2D. The validity of self-reported diabetes was evaluated in a subsample of 1222 women who responded to a supplementary T2D questionnaire. We found that 83.6% (95% CI: 81.3, 85.7) of women with self-reported T2D confirmed their diagnosis in the T2D supplementary questionnaire.

We also used clinical databases and mortality registries to identify additional diabetes cases. We cross-linked all MTC participants with two clinical databases from a healthcare provider (Institute for Social Security and Services for State Workers, ISSSTE) for which 78% of study participants are health insured and are social security policyholders. Beginning in 2007, patients with uncontrolled diabetes in ISSSTE were invited to a comprehensive diabetes management program (MIDE, Comprehensive Management of Diabetes by Stages). Also, in 2016 this institution implemented the Observatory of Diabetes and Chronic Disease (ODEC, for its Spanish acronym), a chronic disease registry that included diabetes (controlled and uncontrolled). We used a two-step cross-linkage procedure where we first identified perfect matches using a national identification number as the matching variable. We then used a probabilistic record linkage program (Registry Plus LinkPlus software Version 2.0, CDC, Atlanta) that uses an algorithm that works well for Hispanic names. Pairs matched on first and last names were manually reviewed and date of birth and state of residency was used to confirm matches. Deaths were identified using employer information (updated once a year), pension fund management databases, and next-of-kin reports. Cause of death was obtained through record linkage (following the same procedure as above) with two mortality registries (the Epidemiological and Death Statistics Subsystem and the National Institute of Statistics, Geography, and Informatics death record databases). We used the International Classification of Diseases 10th Revision's (ICD-10) code E11 to identify incident diabetes cases on the underlying cause of death and six contributing causes of death recorded in the death certificate. We additionally cross-linked all remaining MTC participants to these registries to identify deaths that had not been previously recorded (Supplementary Fig. 1). For the current analyses, we restricted deaths to those occurring between January 1, 2006, through April 30, 2014, the end of our follow-up.

Assessment of covariates

Baseline questionnaires collected information on age; household assets; smoking, physical activity, and other lifestyle habits; reproductive health; anthropometric measures, such as weight and height; family history of diabetes in first-degree relatives, and recently diagnosed diseases. The number of household assets (including access to car, telephone, cell phone, microwave, vacuum, computer, and internet) was used to create a socioeconomic status score (SES). Smoking was reported as never, past or current smokers. Physical activity was categorized into tertiles of total metabolic equivalent of task (MET) per week of multiple-choice frequencies of activity. The correlation between the MTC questionnaire and the International Physical Activity Questionnaire was 0.64 for moderate and vigorous physical activity [Pearson CC: 0.70 (95% CI 0.60,0.97); ICC: 0.780 (95% CI 0.67,0.88)] [60]. Total energy consumption was calculated by multiplying the energy of the specified predefined portion sizes by the frequency of consumption using the USDA food-composition database [59] supplemented with a database used in the National Health and Nutrition Survey in Mexico (personal communication). Alcohol consumption was calculated from the FFQ in grams per day. Menopausal status was defined using an algorithm that considered last menstruation, hot flushes, hormonal treatments, hysterectomy, and oophorectomy. When these data were unknown, an algorithm that used current age was used to determine menopausal status. BMI was calculated in kg/m^2 from self-reported weight and height by participants at baseline. The reproducibility and validity of self-reported anthropometry were evaluated in a subset of 3,413 MTC participants. Standardized technician measurements were well correlated with self-reported weight ($r = 0.92$) and height ($r = 0.86$) [61]. A medical diagnosis of hypertension was self-reported by participants at baseline.

Statistical analysis

We categorized women into 4 categories based on the distribution of the EAT-HRD score (1–4, 5–6, 7–8, and 9–13, with the lowest category as reference). Secondary exposures included the 15 individual food groups. Women were categorized as meeting or not the EAT-HRD food group recommendation.

Person-time was calculated for each woman from the date of response to the baseline questionnaire to the date of T2D diagnosis, date of death, or the date of response to their last questionnaire, whichever came first. For clinical or administrative databases, the end of follow-up was April 30, 2014. We assigned the date of diagnosis to the midpoint of the year in which a physician diagnosis was received. When the year of diagnosis was missing, the date was imputed to the midpoint between the date of response to the last questionnaire where they self-reported being free of T2D and the date of response to the questionnaire where they self-reported having T2D. We used Cox proportional hazard regression models to estimate HRs and 95% confidence intervals (CI) of the association between categories of the EAT-HRD score and T2D incidence. We also estimated the association between meeting the recommendation of each food group of the EAT-HRD score (secondary exposures) and incidence of T2D.

We used previous knowledge on biological mechanisms and variables that are associated with diet and are risk factors for T2D to decide which variables to include in the multivariable models [62]. Our first model was adjusted for age (continuous, years) and total energy intake (continuous, kcal/d). The second model was additionally adjusted for baseline socioeconomic, lifestyle, and reproductive factors, including the region of residence (north, center, Mexico City and the metropolitan area, south); SES (household assets, tertiles); family history of diabetes (yes, no, unknown); smoking (current, never, past), physical activity (METs/wk, tertiles), alcohol consumption (continuous, g/d), and menopausal status (premenopausal/postmenopausal/unknown). For the food group analyses (secondary exposures), we ran additional models with the food group (categorical) adjusted for all EAT-HRD food groups (continuous).

We used missing indicator variables to handle partially missing confounder information for family history of diabetes (14.5%), smoking (3.5%), and physical activity (1.2%) [63]. Additionally, we calculated the E-value, defined as the minimum strength of the association that an unmeasured (or residual) confounder would need to have with both, the exposure and the outcome, conditional on the measured covariates, to explain away a specific exposure-outcome association [64, 65].

We conducted several sensitivity analyses. First, we defined treated T2D as if women self-reported having medical treatment for T2D in addition to a medical diagnosis. For this definition, 87.0% (95% CI: 84.8, 89.0) of women who self-reported a medical diagnosis of T2D with medical treatment, confirmed their diagnosis or treatment in the T2D supplementary questionnaire. Second, given that the two clinical databases from the healthcare provider used to identify incident T2D cases only cover 78% of study participants, we restricted the analysis to this subgroup. Third, we categorized our primary exposure in quartiles instead of pre-specified categories. Fourth, BMI was not included in the main analysis because diet and BMI were measured at the same time, thus, BMI may act as a mediator of the EAT-HRD score and diabetes incidence. However, BMI may also be a confounder if BMI alters women's diet. Therefore, we ran an additionally adjusted model with baseline BMI (normal, overweight, obese). Finally, we further adjusted for hypertension at baseline (yes/no), to explore the potential confounding effect of hypertension in the association between the EAT-HRD score and T2D. Some studies have suggested that having hypertension increases the risk of T2D [66, 67], and people with a hypertension diagnosis can also modify their diet. All analyses were conducted using SAS software version 9.4.

DATA AVAILABILITY

The data that supports the findings of this study are available from the corresponding author (DS) upon request.

CODE AVAILABILITY

The code that supports the findings of this study is available from the corresponding author (DS) upon request.

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AUTHOR CONTRIBUTIONS

GEL and DS designed the work, GEL performed the analysis with supervision of DS, GEL and DS drafted the manuscript, and CB, CG, MC, ACV, RLR and ML revised the manuscript.

COMPETING INTERESTS

The authors declare no competing interests.

ADDITIONAL INFORMATION

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