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Association Between the EAT-Lancet Diet Pattern and Risk of Type 2 Diabetes: A Prospective Cohort Study

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Background: The EAT-Lancet Commission has promulgated a sustainable dietary guideline and recommended that it was designed to improve the human health and support environmental sustainability.

Objective: This research was designed to explore the association between this healthy diet pattern (EAT-Lancet diet pattern, EAT-LDP) and risk of type 2 diabetes (T2D).

Methods: Between 2006 and 2010, a total of 59,849 participants from the UK Biobank without diabetes, cardiovascular disease, or cancers were included at baseline. The EAT-LDP score was constructed on the sum of 14 food components and then categorized into three tertiles. Multivariable Cox proportional hazards regression models were conducted to explore the association between EAT-LDP score and the risk of incident T2D. A mediation analysis was also implemented to disentangle the role of body mass index (BMI) and waist circumference in the relationship between EAT-LDP score and T2D.

Results: During a median follow-up of 10 years, 2,461 incident T2D cases were recorded. In analyses that compared tertile 3 of the EAT-LDP score (highest) with tertile 1 (lowest), the hazard ratio (HR) for T2D was 0.81 (95% CI: 0.72–0.90) after adjusting for sociodemographic status and health-related factors. Participants who reported a one-point increase in the diet score were associated with a 6% decrease in risk of T2D (HR: 0.94, 95% CI: 0.91–0.97). A significant indirect association was observed between the EAT-LDP score and T2D (β: 0.66, 95% CI: 0.65–0.67), indicating that 44% of the association of EAT-LDP score with T2D was mediated by BMI. Additionally, 40% of the association of EAT-LDP score with T2D was mediated by waist circumference was also observed.

Conclusions: Our findings indicate that a higher adherence to EAT-LDP contributes to lower risk of T2D. Further independent validation is needed to be conducted before applying the EAT-LDP to inform dietary guidelines.

Keywords: EAT-Lancet diet pattern, type 2 diabetes, nutrition, UK biobank, metabolism

INTRODUCTION

Diabetes mellitus, characterized by chronic hyperglycemia, refers to a metabolic condition that results from an interaction of genetic and environmental factors (1, 2). The prevalence of diabetes in the UK is up to 3.8 million people, accounting for about 9% of the adult population (3). Evidence has shown that suboptimal diet could be the driver of the obesity pandemic, which is the leading risk factor of preventable death and disability (4–6). Numerous studies have evaluated the associations between the specific food type and nutrient intake with type 2 diabetes (T2D) risk (7–9).

With the emphasis on overall diet quality, the diet pattern integrates potentially interactive and cumulative associations of different dietary components, which facilitate translation of findings into dietary recommendations. Diet patterns could reflect the numerous and multifaceted combinations of nutrient and food consumption in the real world (10). Multiple studies have found a beneficial effect of higher adherence to the plant-based Mediterranean (Medi) diet pattern on the risk of diabetes (11–13). In a comparative study, researchers concluded that both Healthy Eating Index (HEI) and Alternate Healthy Eating Index (AHEI) showed influences on T2D (14) in the US adults. A meta-analysis including 15 cohort studies showed that diets of the high quality are associated with a significant risk reduction for T2D and other chronic diseases ($p < 0.05$) (15). A prospective study among Taiwanese has found a protective effect of vegetarian diet on diabetes risk (16). The low levels of triglyceride to high-density lipoprotein cholesterol ratio diet pattern may reduce the incident T2D that has also been ascertained (17).

Here, we examined a new healthy and sustainable diet pattern, named EAT-Lancet diet pattern (EAT-LDP) raised by the EAT-Lancet Commission, which was designed to nurture human health and support environmental sustainability (18). It consists of whole grains, fruits, vegetables, legumes, nuts, unsaturated oils, low-to-moderate amount of seafood and poultry, and includes no or a low quantity of red meat, processed meat, added sugar, refined grains, and starchy vegetables (19). To date, few prospective studies have investigated the association between the EAT-LDP and adverse health conditions (20, 21). In this study, we further explored the association between EAT-LDP and incident T2D among the UK adults over a more than 10-year follow-up.

MATERIALS AND METHODS

Data Source and Study Population

The UK Biobank is a large-scale biomedical database started from 2006. The aim of the program is to investigate the influence of genetic and environmental factors on disease development. It has recruited more than 500,000 volunteers aged 40 to 69 across the UK and will follow them over the next 30 years. Participants were invited to the assessment centers to complete a series of lifestyle, health and socioeconomic interviews, and physical measurements. In addition, biological samples were also collected. All disease conditions, prescription drug use, and deaths of them during the whole study period will be recorded through the centrally managed UK National Health Service system (22).

The initial 502,507 participants were invited to provide information on their food consumption in the past year through a touch questionnaire at the assessment centers. Subsequently, a sub-sample of about 20,000 population was also invited to repeat the questionnaire at assessment centers every 4 years, to examine the possible changes in participants' responses to the questionnaire and their dietary intake over time. Meanwhile, in order to gather more detailed information about the actual amount of food or food groups actually consumed by the participants, the UK Biobank also adopted an online 24-h dietary assessment tool named Oxford WebQ. It was developed for use in large population studies and had been validated in previous studies (23, 24). It collected additional detailed dietary intake information of 210,970 participants at least once through the 24-h recall. Participants were asked whether they have consumed the predefined 206 foods or 32 drinks in the past 24 h (24). After that, some of them were also invited to repeat the online 24-h questionnaire for a total of four times through emails between February 2011 and June 2012 every 3–4 months. At the same time, participants were also asked whether they consumed over the previous 24 h were fairly typical for their daily life (25).

Participants who reported consumption of at least 7 foods included in the EAT-LDP based on the 24-h dietary assessment tool were included in the analysis first ($N = 69,686$). Later, who reported history of any cancer (2,953), cardiovascular disease (1,923), and diabetes (1,269) at the baseline were also excluded. We also excluded participants with abnormal total energy intakes (<2,093 or >14,650 kJ/day in female and <3,349 or >16,743 kJ/day in male participants). Finally, we excluded participants who were followed for <1 year ($N = 236$) to minimize the potential for reverse causality bias. As a result, a total of 59,849 participants were considered for inclusion in the following main analysis (**Figure 1**). All participants had given written informed consent.

Definition of EAT-LDP Score

The EAT-LDP score used in this study was designed by Knuppel et al. (20). The distribution of the diet score can be seen in **Supplementary Table 1**. Definition of portion size and food items used in this study can be seen in **Supplementary Table 2**. The EAT-LDP score is consisted of 8 main dietary components, including whole grains, tubers and starchy vegetables, vegetables, fruits, dairy foods, protein sources, added fats, and added sugars. Participants were assigned with a point for meeting each of the recommendations. Each dietary component contributed 0 or 1 point resulting in a total score ranging from 0 to 14 points. The higher dietary scores indicated a greater adherence to the individual healthy eating patterns.

The online 24-h dietary assessment tool did not record the concrete weight of consumed food, but the number of predefined portion size was defined using the UK's standard

Abbreviations: EAT-LDP, the EAT-Lancet diet pattern; T2D, type 2 diabetes; BMI, body mass index; PA, physical activity; MET, metabolic equivalent; HR, hazard ratio.

TABLE 1 | Sociodemographic characteristics of the study population by the EAT-LDP score group.

Characteristic	Total	Tertiles of EAT-LDP scor			*P*-value
		T1	T2	T3	
Total, *n* (%)	59,849	27,527 (45.99)	15,188 (25.38)	17,137 (28.63)	
Total energy intake (kJ/day), mean (SD)	8,463.00 (2,442.31)	8,218.17 (2,517.47)	8,547.62 (2,400.66)	8,781.33 (2,311.17)	<0.001
Age (years), mean (SD)	55.91 (8.14)	55.08 (8.24)	56.21 (8.09)	56.96 (7.89)	<0.001
Sex, *n* (%)					<0.001
Female	34,512 (57.67)	14,547 (52.85)	9,013 (59.34)	10,952 (63.92)	
Male	25,337 (42.33)	12,980 (47.15)	6,175 (40.66)	6,182 (36.08)	
Ethnicity, *n* (%)					<0.001
White	56,049 (93.65)	25,420 (92.35)	14,362 (94.56)	16,267 (94.94)	
Asian or Asian British	452 (0.76)	226 (0.82)	105 (0.69)	121 (0.71)	
Black or black background	1,284 (2.15)	674 (2.45)	276 (1.82)	334 (1.95)	
Chinese	1,114 (1.86)	709 (2.58)	214 (1.41)	191 (1.11)	
Mixed background	176 (0.29)	93 (0.34)	36 (0.24)	47 (0.27)	
Others	774 (1.29)	405 (1.47)	195 (1.28)	174 (1.02)	
Townsend deprivation index, *n* (%)					<0.001
1 (least deprived)	11,972 (20.04)	5,048 (18.37)	3,196 (21.08)	3,728 (21.79)	
2	11,964 (20.05)	5,300 (19.29)	3,044 (20.07)	3,620 (21.16)	
3	11,944 (19.99)	5,493 (19.99)	3,034 (20.01)	3,417 (19.97)	
4	11932 (19.97)	5475 (19.93)	3072 (20.26)	3385 (19.78)	
5 (Most deprived)	11,936 (19.98)	6,159 (22.42)	2,818 (18.58)	2,959 (17.29)	
Education attainment, *n* (%)					<0.001
College or university degree	23,617 (39.46)	9,846 (35.77)	6,314 (41.57)	7,457 (43.52)	
Professional qualifications	2,894 (4.84)	1,234 (4.48)	759 (5.00)	901 (5.26)	
Others	33,338 (55.70)	16,447 (59.75)	8,115 (53.43)	8,776 (51.22)	
Smoking status, *n* (%)					<0.001
Never	34,432 (57.53)	15,089 (55.00)	8,940 (59.01)	10,403 (60.84)	
Previous	20,369 (34.13)	9,416 (34.32)	5,145 (33.96)	5,808 (33.96)	
Current	4,884 (8.18)	2,930 (10.68)	1,065 (7.03)	889 (5.20)	
Drinking status, *n* (%)					0.005
Never	2,194 (3.67)	1,020 (3.71)	558 (3.67)	616 (3.60)	
Previous	1,891 (3.16)	939 (3.41)	457 (3.01)	495 (2.89)	
Current	55,700 (93.07)	25,535 (92.76)	14,151 (93.17)	16,014 (93.46)	
Obesity-related markers					
BMI, mean (SD)	26.97 (4.64)	27.43 (4.74)	26.85 (4.60)	26.36 (4.44)	<0.001
Waist circumference	88.77 (13.20)	90.33 (13.37)	88.34 (13.14)	86.66 (12.65)	
Total PA (MET-min/week), mean (SD)	2,668.23 (2,604.84)	2,601.35 (2,643.52)	2,687.34 (2,597.38)	2,756.06 (2,547.09)	<0.001

BMI, body mass index; EAT-LDP, EAT-Lancet diet pattern; PA, physical activity; MET, metabolic equivalent.

was mediated by BMI as mediator variable. The total effect was significant ($\beta = 0.56$, 0.54–0.57, $p < 0.0001$), with the natural direct effects ($\beta = 0.85$, 0.82–0.88, $p < 0.0001$) and the natural indirect effect ($\beta = 0.66$, 0.65–0.67, $p < 0.0001$). Similar results were found within sex-stratified analyses. The relationship between the EAT-LDP score and risk of T2D was 36% mediated by BMI among male and 53% among female participants.

Table 3 also shows that the mediating role of the waist circumference in the association between the EAT-LDP score and incident of T2D (40%). Similarly, the relationship between the EAT-LDP score and risk of T2D was 35% mediated by waist circumference among male and 50% among female participants.

Sensitivity Analysis

We observed similar results in the analysis that included participants who were followed for <3 years, which reinforced the robustness of our findings (**Supplementary Table 5**). The results did not significantly change when the multiple imputation was conducted (**Supplementary Table 6**). We also explored the associations between the EAT-LDP score and incident T2D stratified by age, sex, ethnicity, Townsend deprivation index, education attainment, smoking status, and drinking status. In the stratified analysis, compared tertile 3 of the EAT-LDP score with tertile 1, the hazard ratio (HR) for T2D in female participants was 0.72 (95% CI: 0.61–0.86). The protective effects of adherence to higher EAT-LDP score can be seen in the white,

TABLE 2 | HRs (95% CIs) for the associations between EAT-LDP score and incidence of T2D (n = 59,849).

	No. of participants (%)	Cases of T2D	Incidence rate per 1,000 person-year (95% CI)	Model 1[a]	Model 2[b]	Model 3[c]	Model 4[d]
Tertiles of EAT-LDP score							
T1	27,527 (45.99)	1,302	4.74 (4.49, 5.00)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)
T2	15,188 (25.38)	567	3.72 (3.43, 4.04)	0.79 (0.71, 0.87)	0.82 (0.75, 0.91)	0.85 (0.76, 0.95)	0.90 (0.81, 1.01)
T3	10,986 (18.36)	592	3.44 (3.17, 3.73)	0.72 (0.66, 0.80)	0.77 (0.70, 0.85)	0.81 (0.72, 0.90)	0.95 (0.81, 1.06)
P for trend				<0.0001	<0.0001	<0.0001	0.249
1-point increment in diet score	59,849	2,461	4.11 (3.95, 4.27)	0.90 (0.88, 0.93)	0.92 (0.90, 0.95)	0.94 (0.91, 0.97)	0.99 (0.96, 1.02)

HRs, hazard ratios; EAT-LDP, the EAT-Lancet diet pattern; T2D, type 2 diabetes. [a]*Crude model.* [b]*Adjusted for age, sex, Townsend deprivation index, qualifications, and ethnicity.* [c]*Adjusted for age, sex, Townsend deprivation index, qualifications, ethnicity, smoking, drinking, physical activity, and energy.* [d]*Adjusted for age, sex, Townsend deprivation index, qualifications, ethnicity, smoking, drinking, physical activity, energy, BMI, and waist circumference.*

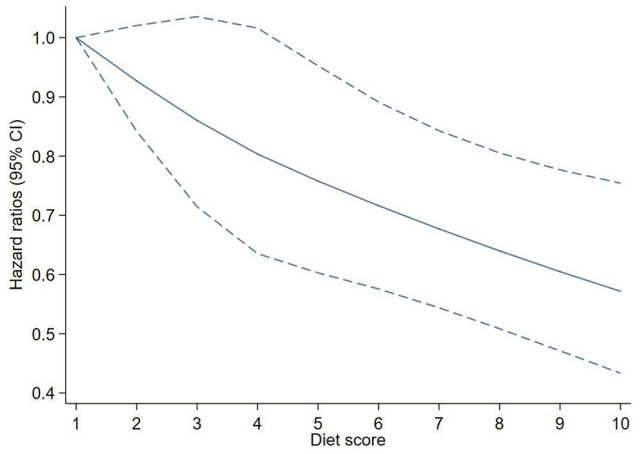

FIGURE 2 | Adjusted hazard ratios (HRs) of T2D events risk according to EAT-LDP score. The figure shows HRs for T2D adjusted for age, sex, Townsend deprivation index, qualifications, ethnicity, smoking, drinking, physical activity, energy, BMI, and waist circumference. BMI, body mass index; EAT-LDP, the EAT-Lancet diet pattern; T2D, type 2 diabetes.

never and previous smoking, and current drinking participants (**Supplementary Table 7**). Meanwhile, the effect modification by these covariates is not significant.

DISCUSSION

This study explored the association between a healthy diet score and risk of T2D in a large population of 59,849 middle-aged adults in the UK. During a median 10 years of follow-up, 2,461 participants developed T2D. Our results show that greater adherence to EAT-LDP was associated with lower T2D risk over time.

Our findings appear to be consistent with the previous studies (20, 31), reporting the EAT-LDP shows beneficial associations for diabetes. With data from the EPIC-Oxford study, Knuppel et al. simultaneously investigated the associations of the EAT-LDP with major health outcomes (including ischemic heart disease, stroke, diabetes, and all-cause mortality) (20). They have drawn the conclusion that the EAT-Lancet reference diet shows beneficial associations for incidence of ischemic heart disease and diabetes, which is consistent with our research. Every individual component of the EAT-LDP has been separately investigated in this study. According to our exploratory results, the adherence to the recommended intake of potato, vegetables, fruits, dairy foods, beef, lamb, and pork were associated with lower T2D incident risk. The specific food components, like fruits, vegetables, legumes, olive oil and fish, have been verified to be associated with better health status (32–34). Consumption of these foods are related to lower body weight, hemoglobin A1c, low-density lipoprotein (LDL) and oxidative stress, and improved high-density lipoprotein (HDL), which are beneficial to the improvement of the prevention and prognosis of T2D (35, 36). Some specific food components were not associated with T2D risk, and this might indicate that the synergistic effects that occur in the EAT-LDP bring superior benefits compared with those from each isolated nutrition (37). Our result also shows that not adhering to the recommendation of the added sugars (≤31 g/day) was associated with a greater risk for T2D. It has been assumed that excess sugar can promote weight gain through extra calories intake, thus T2D (38). In 2015, based on 17 cohorts prespecified information, Imamura et al. concluded that the habitual consumption of sugar sweetened beverages was associated with a greater incidence of T2D (39).

Adhering to the recommendation amount of grains (≤464 g/day) was not associated with T2D risk ($p > 0.675$), which was not consistent with previous studies (40, 41), as both whole grain and refined grains were included in this research. Previous study has found an increased risk of colorectal cancer in those with high intakes of red and processed meat (42). Our study found that people who were consuming an average of ≤28 g/day beef, lamb, and pork had a 40% (95% CI: 0.40–0.90) lower risk of T2D. This means higher beef intake is associated with increased T2D risk. Proportion of the study population adhering to the protein foods is relatively small. Currently, recommendations of protein intake are based on individual assessment and the consideration of health issues (43). We need to further explore the underlying associations between the specific food component and risk of the T2D.

TABLE 3 | Adjusted direct and indirect associations of T2D with EAT-LDP score mediated *via* BMI, and waist circumference.

	Overall ($N = 59,849$)		Male ($N = 25,337$)		Female ($N = 34,512$)	
Measures	**β (95% CI)**	***P*- value**	**β (95% CI)**	***P*-value**	**β (95% CI)**	***P*-value**
BMI						
Marginal total association	0.56 (0.54, 0.57)	<0.0001	0.70 (0.67, 0.73)	<0.0001	0.42 (0.40, 0.44)	<0.0001
Natural direct association	0.85 (0.82, 0.88)	<0.0001	1.12 (1.07, 1.17)	<0.0001	0.61 (0.58, 0.65)	<0.0001
Natural indirect association *via* BMI	0.66 (0.65, 0.67)	<0.0001	0.62 (0.61, 0.63)	<0.0001	0.68 (0.67, 0.69)	<0.0001
Proportion mediated (%)	44	36	53			
Waist circumference						
Marginal total association	0.55 (0.53, 0.56)	<0.0001	0.69 (0.66, 0.72)	<0.0001	0.41 (0.39, 0.43)	<0.0001
Natural direct association	0.90 (0.87, 0.93)	<0.0001	1.13 (1.08, 1.18)	0.020	0.63 (0.61, 0.67)	<0.0001
Natural indirect association *via* waist circumference	0.61 (0.53, 0.56)	<0.0001	0.61 (0.60, 0.62)	<0.0001	0.64 (0.63, 0.65)	<0.0001
Proportion mediated (%)	40	35	50			

BMI, body mass index; EAT-LDP, the EAT-Lancet diet pattern; T2D, type 2 diabetes. Adjusted for age, sex, Townsend deprivation index, qualifications, ethnicity, smoking, drinking, physical activity, and energy.

Much of the existing literature has considered obesity indicators such as BMI, waist circumference, or waist–hip ratio (WHR) as confounders and adjusted them in the Cox model (44, 45). However, we conducted the mediation analysis and observed that the association between the EAT-Lancet diet adherence and the risk of T2D was 44% mediated by BMI, or 40% by waist circumference. We also observed a direct effect of the healthy diet, suggesting that EAT-LDP can prevent T2D even if it does not lead to change to BMI or waist circumference. Our results have strong biological plausibility. Laouali et al. found that a higher anti-inflammatory potential of the diet is associated with a lower risk of T2D with BMI as a mediator factor in a France population (46). Fan et al. prospectively followed 10,419 Chinese adults and concluded that the waist circumference and its change were strongly associated with the risk of T2D (47). Previous studies have observed that weight loss among overweight or obese patients with T2D was consistently associated with a reduction of hemoglobin A1c, insulin resistance, and leptin levels, which involved in the pathogenesis of T2D (48, 49).

Strengths and Limitations

This study has the advantages of prospective design and large sample size of diet habits to explore the association between the healthy and sustainable EAT-LDP and incident of T2D. Our results of the main analysis are shown to be consistent with the sub-analyses. There are some potential limitations warrant consideration. First, each component of EAT-LDP score was constructed as a binary variable (adherence to the target intake levels vs. non-adherence). This may lead to the loss of some dietary information. A more refined scoring method should be developed to investigate its association with the possible health status. Second, participants in our study can only represent middle-aged and elderly people. And 93% of people are white. Target participants included in our main analysis were those who reported consumption of at least 7 foods according to the EAT-LDP. Therefore, the obtained results could not be generalized to other population with different characteristics. At the same time, the dietary information used in this analysis mainly comes from the baseline assessment, which may not reflect the potential changes in participants' eating habits. Third, patients diagnosed early were followed for a longer period of time than patients diagnosed in recent years. Longer follow-up time would allow the increase of the duration between nutritional assessment and assessment of the T2D. Fourth, T2D in this research was diagnosed through inpatient medical records. Although doctors' diagnosis is a more common and precise way, the actual incidence of T2D could be underestimated. Last but not least, although we have adjusted for different confounding factors, there may be residual of unmeasured confounding factors that cannot be excluded in the observational studies. More validation is needed for reliable estimation of the associations between EAT-LDP and the possible adverse health outcomes.

Conclusions

In light of the increasing global burden of diabetes, our results seem to be clinically relevant for diabetes prevention, and the EAT-LDP is an achievable and sustainable objective that should be promoted.

DATA AVAILABILITY STATEMENT

Details of the UKB data are available upon reasonable request (https://bbams.ndph.ox.ac.uk/ams/resApplications).

ETHICS STATEMENT

The studies involving human participants were reviewed and approved by NHS National Research Ethics Service (NW/0382). The patients/participants provided their written informed consent to participate in this study.

AUTHOR CONTRIBUTIONS

YW contributed to the conception and ideas. YW, XW, CX, and ZC contributed to the study design. YW and ZC had full access to all of the data in the study. CX and ZC performed the statistical analysis, results interpretation, and assisted by HY and YH. CX and ZC

wrote the first and successive drafts of the manuscript. All authors contributed to the article and approved the submitted version.

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SUPPLEMENTARY MATERIAL

The Supplementary Material for this article can be found online at: https://www.frontiersin.org/articles/10.3389/fnut.2021.784018/full#supplementary-material

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