European Journal of Clinical Nutrition
https://doi.org/10.1038/s41430-020-0673-9

ARTICLE

Carbohydrates, glycemic index and diabetes mellitus

Midlife susceptibility to the effects of poor diet on diabetes risk

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Received: 23 September 2019 / Revised: 19 May 2020 / Accepted: 23 June 2020

Abstract

Objective Type 2 diabetes mellitus (T2D) prevalence continues to increase, and age of incidence continues to decrease. More information is needed to target interventions to the ages where they can be most effective. The objective of this study was to explore the degree to which the association between diet and T2D incidence changes through adulthood.

Methods Participants were a large number ($N = 2818$) of community living adults in Canberra and Queanbeyan, Australia across three cohorts; young (20–24 followed to 32–36), mid-life (40–44 followed to 52–56) and late-life (60–64 followed to 72–76). Self-report dietary pattern scores at baseline and diabetes incidence across 12 years follow-up were measured, alongside confounders of caloric intake, sex, smoking status, years of education, hypertension, BMI and physical activity.

Results Cox proportional hazards indicated that neither Western nor Prudent dietary pattern scores were significantly associated with T2D incidence when confounders were included in the model. Unadjusted estimates suggested a positive association between Western dietary pattern scores and subsequent diabetes incidence (HR = 1.40, 95% CI [1.18, 1.64]). Compared with the mid-life cohort, a higher Western dietary pattern score posed a lower risk for incident T2D in the young cohort (unadjusted HR = 0.46, 95% CI [0.22, 0.96]), who also had significantly lower BMI and higher physical activity. No such significant effects were found for the late-life cohort.

Conclusions Our findings indicate that mid-life may be a period of heightened vulnerability to the effects of an unhealthy diet on diabetes risk, but this effect is attenuated when risk factors related to diet, such as adiposity, are taken into account.

Introduction

Poor diet is a major risk factor for the development of type 2 diabetes mellitus (T2D). A dietary pattern characterised by foods including lean meat, fish, unprocessed grains, fruit and vegetables ("Prudent") is associated with a lower risk of developing T2D, while a pattern characterised by foods high in sugars, fats and processed foods ("Western") is associated with a higher prevalence of T2D, even when total energy intake is taken into account [1–3]. Research in older adults (median age 50 or over) indicates that a Western dietary pattern is associated with higher T2D incidence [4], while a Prudent dietary pattern is associated with lower incidence [5, 6]. The link between diet quality and T2D does not occur in isolation.

Obesity and sedentary lifestyle are two of the primary modifiable risk factors for T2D incidence, due to their association with disrupted glucose metabolism (primarily via increasing insulin resistance) [7, 8]. Other important factors include sex (diet is less effective in achieving glycaemic control in women with diabetes [9]), smoking status (which can overwhelm the effects of good diet for glycaemic regulation [10]), years of education (where poorer

Supplementary information The online version of this article (https://doi.org/10.1038/s41430-020-0673-9) contains supplementary material, which is available to authorized users.

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Published online: 10 July 2020

education is associated with poorer glycaemic management and higher T2D prevalence [11]) and hypertension (which shares similar dietary risk factors as T2D [12], and is highly comorbid with T2D [13]).

Importantly, the impact of many of these risk factors depends on timing in life. T2D is diagnosed earlier in men than women [14]. The health risks posed by smoking or lack of glycaemic management associated with low levels of education are cumulative and thus are likely to compound throughout life, and co-morbidities with hypertension become more common with age [15]. Most notably, poor diet in combination with decline in physical activity is associated with increase in adiposity [16–18], which contributes substantially to increasing T2D incidence throughout adulthood [5, 19, 20].

At the population level, there is a gradual decrease of age at onset in T2D [21], highlighting the need to examine risk factors earlier in life. Although much of the literature focussing on diet and subsequent T2D incidence robustly accounts for many risk factors in cross-sectional settings, the importance of timing in life requires further investigation. The objective of this exploratory study was to establish the degree to which the association between diet and T2D incidence changes across adulthood. We use a survival analysis framework to explore the association between diet and diabetes incidence in a large community living sample, in the context of unmodifiable (such as sex) and modifiable factors (such as adiposity and physical exercise), with a specific focus on the role of age via the inclusion of three age cohorts across the adult lifespan.

Materials/subjects and methods

Participants were drawn from a population-based longitudinal study of ageing in Canberra and Queanbeyan, Australia, the PATH Through Life Project [22]. Seven thousand four hundred eighty-five participants were selected at random from the electoral roll and followed over 12 years to form three cohorts; young (aged 20–25 years in 1999–2000, followed to age 32–37), mid-life (aged 40–45 years in 2000–2001, followed to age 52–57) and late-life (aged 60–65 years in 2001–2002, followed to age 72–77). These age bands were selected a priori to from a longitudinal cross-sequential study, with cohort ages planned to overlap at 15 years follow-up. At the time of analysis, the average follow-up time between cohorts was 12 years (SD 0.43 years). Participants were selected on the basis of dietary data and T2D status availability. Following the sample selection steps outlined in Supplementary Table 1, 2818 participants were selected for analyses (young $n =$ 674, mid-life $n = 1018$, late-life $n = 1126$). All participants provided written informed consent. The ethical aspects of this study were approved by the Australian National University Human Research Ethics Committee.

The key outcome was whether participants self-reported T2D or adherence to treatment (diet plan, or medication) at any given wave of data collection. This was measured at all four waves of data collection. The key predictor was diet, which was measured at baseline using the self-reported Commonwealth Scientific and Industrial Research Organisation semi-quantitative Food Frequency Questionnaire [23]. Total caloric intake (kj/day), western and prudent dietary pattern z scores were derived in previous work undertaken by Jacka et al. using principal components analysis on the PATH dataset [24]. A Western dietary pattern was characterised by sausages, roast meat, chips and crisps and soft drinks. A Prudent dietary pattern was characterised by fresh fruit, vegetables, grilled fish and salad. See Supplementary Fig. 1 for a more detailed summary of constituent foods and loadings for each dietary pattern score, as derived in Jacka et al. [24]. The key modifier was age, as decomposed into time in study (years from baseline) and cohort (young, mid and late life). This deconstruction allows for investigation of the effects of time passing, and time in life (the focus of this study). The role of age will be explored by a cohort × dietary score interaction.

Covariates were selected on the basis of their reported links with the links between diet and diabetes incidence: total caloric intake, sex [9], smoking status [10], years of education [11], hypertension [13], BMI and physical activity [25]. Sex, smoking status and years of education were provided by self-report. Hypertension status was assigned on the basis of self-report of a medical diagnosis, self-reported medication use, or the average of two seated blood pressure measures exceeding 140 mmHg systolic and 90 mmHg diastolic. BMI was computed by the formula weight (kg)/height × height (m^2), based on self-report of weight and height at each assessment, and centred on 25 (the boundary between normal and overweight). Physical activity was measured in terms of metabolic equivalents (METs), from self-reported hours spent engaging physical using the formula (hours mild PA × 3) + (hours moderate PA × 6) + (hours vigorous PA × 9) [26].

Statistical analysis

Analysis consisted of linear models for demographic comparison across cohorts, and longitudinal Cox proportional hazards models for survival analysis (using the R package "Survival" [27]). Time was specified as wave of data collection (0 for baseline, and 1, 2, 3 for follow-up), paired with a Boolean indicator of diabetes (yes/no) at that time. Following STROBE guidelines, we fit unadjusted estimates and confounder-adjusted estimates with 95% confidence intervals. Models were fit for Western and

Prudent dietary variables separately and in combination, and comparison of coefficient performance and likelihood ratio tests were used to ascertain whether they could be modelled simultaneously. Notable coefficients are reported as hazard ratios in text to aid interpretability. Threats to model stability from nonlinearity, non-normality, non-informative censoring, non-proportional hazards and unmatched cohort sizes were also examined (See Supplementary materials for description of tests). Analysis code is available in the Supplementary materials. Alpha was set at 0.05.

Results

Sample characteristics are outlined in Table 1, which also includes differences between selected/non-selected participants from the broader PATH cohort. Western and Prudent dietary scores were included in the same cox survival hazard models due to very low correlation ($r < 0.01$), null likelihood ratio tests for no treatment effect in the survival process ($p > 0.05$) and minimal differences in coefficient magnitude, direction or significance (Supplementary Table 2). Cox survival hazard models are presented in Fig. 1 (see Supplementary Table 3 for precise numbers). Tests for non-informative censoring and assumption of proportional hazards indicated these factors did not bias results (see Supplementary Tables 4, 5).

In unadjusted models only, subsequent T2D incidence was positively associated with a Western dietary pattern score, negatively associated with a prudent dietary pattern score, negatively associated with being in the young cohort and positively associated with being in the late-life cohort (relative to mid-life cohort). The addition of a cohort by dietary score interaction indicated that the association between Western dietary score and diabetes incidence was significantly lower in the young than the mid-life cohort, though this was also significant in the unadjusted model only.

Linear models indicated that dietary patterns, body mass index and physical activity differed with age. Compared with the mid-life cohort, the young cohort had significantly higher Western dietary scores ($b = 0.29$, 95% CI [0.20, 0.38]), lower Prudent dietary scores ($b = -0.22$, 95% CI [−0.31, −0.13]), lower BMI ($b = -1.15$ units, 95% CI [−1.65, −0.65]) and more physical activity ($b = 6.041$ METs, 95% CI [2.2, 9.88]). Individuals in the late-life cohort had significantly lower Western dietary scores ($b = -0.19$, 95% CI [−0.27, −0.11]), higher Prudent dietary scores ($b = 0.34$, 95% CI [0.26, 0.42]), lower BMI ($b = -0.87$ units, 95% CI [−1.30, −0.43]) and more physical activity ($b = 11.27$ METs, 95% CI [7.93, 14.6]) than those in the mid-life cohort.

Table 1 Sample characteristics.

	Cohort (selected participants only)				Those not selected from PATH
	Young ($n = 674$)	Mid-life ($n = 1018$)	Late-life ($n = 1126$)	All ($n = 2818$)	($n = 4667$)
Age (at baseline)	23.18 (1.51)	43.17 (1.46)	62.98 (1.48)	46.27 (15.65)	41.76 (16.44)
Total person years at risk of diabetes incidence (PYAR)	8083	12,482	13,774	34,339	Unavailable
Diabetes incidence in study duration (absolute count)	10 (1.48)	32 (3)	105 (9)	147 (5)	Unavailable
Diabetes incidence in study duration (incidence rate accounting for PYAR)	0.001	0.002	0.007	0.004	Unavailable
Sex (Female)	438 (65)	595 (58)	568 (50)	1602 (56)	2193 (47%)
BMI	23.41 (3.96)	25.31 (4.36)	26.24 (4.15)	25.31 (4.36)	25.67 (5.01)
METs	44.45 (52.24)	36.52 (30.85)	40.63 (37.27)	40.09 (39.57)	40.76 (47.77)
Western dietary score (z)	0.24 (1.14)	−0.06 (0.94)	−0.25 (0.80)	−0.06 (1.14)	Unavailable
Prudent dietary score (z)	−0.31 (0.94)	−0.09 (0.95)	0.26 (0.97)	0 (0.98)	Unavailable
Total energy intake (kj/day)	9472.96 (3267.38)	8762.15 (2561.93)	8510.67 (2234.6)	8832.58 (2657.31)	Unavailable
Years education	15.02 (1.47)	14.84 (2.27)	14.27 (2.54)	14.66 (2.25)	14.1 (2.37)

For continuous variables, values reported are mean, values in brackets are standard deviation. For categorical variables, values reported are counts, values in brackets are percentages. Total person years at risk of diabetes incidence is calculated from exact time in study, so does not directly correspond to $n \times 12$ years. Extremely high standard deviation in METs is due to high skewness of variable. See Supplementary materials for participant selection (Supplementary Table 1). Inclusion required completeness in diabetes and dietary information, hence PYAR and dietary scores are unavailable for comparison between selected and non/selected.

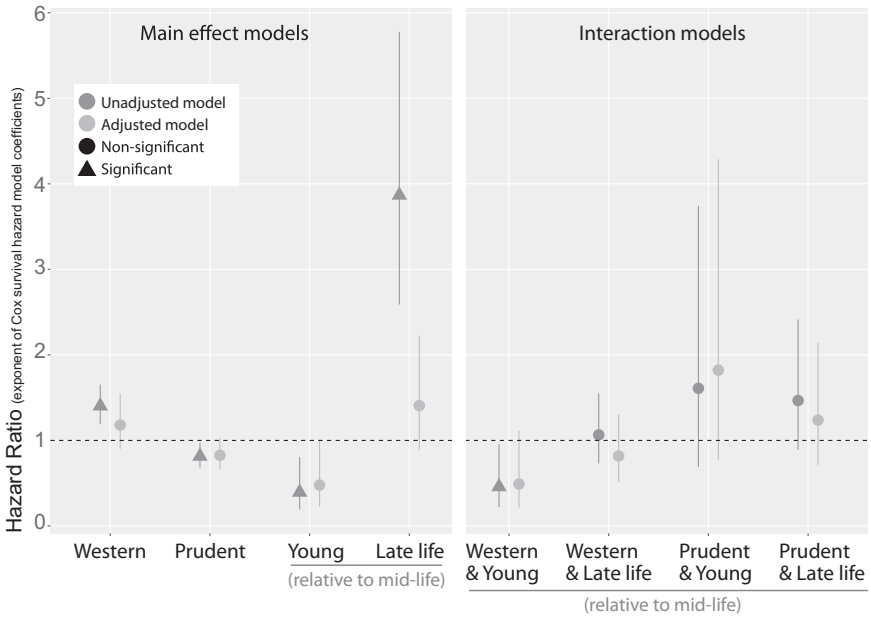

Fig. 1 Cox survival hazard model results. Coefficients are expressed as the exponent of the Cox survival hazard model coefficients. Unadjusted model contains only dietary pattern scores and age cohort. Adjusted model contain total caloric intake, sex, smoking status, years of education, hypertension, body mass index and physical activity. Significance is at $\alpha < 0.05$, demonstrated by 95% confidence intervals (shown by the coloured whiskers) not overlapping HR = 1 (denoted by the dotted horizontal line).

Assumptions for Cox models were met (Supplementary Tables 4, 5). To check the possibility that results were an artefact of sample size, Cox models in the 40s and 60s were repeated with a subsample reflecting the ratio of T2D diagnoses found in the 20s cohort; while significance was lost for the 60s cohort and confidence intervals widened due to the reduced in sample size, the magnitude of effects was similar (Supplementary Table 6).

Discussion

This exploratory study investigated the association between diet and subsequent diabetes incidence across different periods of the adult lifespan. In accord with previous literature, those in mid-life had a healthier diet (lower Western and higher Prudent dietary scores) but they also exercised less, had greater adiposity and a substantially higher risk of incident T2D in a 12 year period than those in the young cohort [16, 17, 19, 21]. Those in late life had the healthiest diet, exercised more than those in mid-life, had the greatest adiposity and the highest risk of incident T2D.

Results suggested that higher Western dietary intake were associated with an increased risk of incident T2D as elsewhere [1, 28], but only in unadjusted models. This association was less pronounced in young adulthood (ages 20–35), and more pronounced in later life (60–75). This may be taken to suggest an age-related increase of the impact of a Western dietary pattern on T2D risk, beginning in mid-life. However, the attenuation in magnitude and significance of these effects once confounders including age, adiposity and physical activity are included in analysis suggest further research is required to investigate the relative role of diet quality alone, versus as a contributor to adiposity.

Taking these tentative findings at face value, several candidate mechanisms can be proposed. One explanation is changes in glucose metabolism efficiency with age. Glucose metabolism becomes less efficient with advancing age [29]. Elevated blood glucose arising from consumption of Western dietary pattern may be metabolised efficiently in young adulthood, but inefficient metabolism in later life may allow chronically high levels to persist. Thus, the same level of consumption can pose differential risks for T2D incidence. Another possibility is cumulative, lifetime effects such as undesirable alterations to gut microbiota [30] and chronic inflammation [31] associated with a Western dietary pattern. A further possibility is that the current findings reflect a selection bias, as those in the younger cohort who did develop T2D may have a sufficiently high (possibly genetic [4]) predisposition that a Western diet cannot further contribute to T2D incidence. Further longitudinal research would be required to establish if this is the case, and also clarify whether undetected shifts in diet throughout the study may have impacted T2D incidence.

Although protective effects did not reach significance, higher Prudent dietary intake was consistently with lower T2D incidence across all cohorts. This could possibly be because, regardless of digestive or metabolic efficiency, properties such as high dietary fibre or low refined carbohydrate availability invariably result in lower postprandial blood glucose than foods typical in the Western dietary pattern [32].

The key strength of this study is the data drawn from a large, population-based sample of adults spanning a wide age range, rather than a specific demographic (e.g. only

female) or clinical group (e.g. the obese). Diabetes incidence in the current study was lower than may be expected, most likely due to the inclusion of the younger cohort, however it was on par with age-standardized estimates from Australian data collected at a similar time [33].

The key weaknesses were that diet was only measured at baseline (so the study is insensitive to changes in exposure over time) that analysis relied on self-report, and the possibility of survivorship bias. There is evidence that dietary patterns measured in a similar manner to the current study are stable over shorter periods (e.g. 14 months; [34] 2 years [35]), but stability over longer periods remains to be established. Current dietary and body mass results were similar to contemporary adult Australian samples (e.g. [36, 37]), indicating sample selection and self-report did not unduly effect key variables, though imprecision results may reflect imprecision in self-report. Survivorship bias may have arisen from lack of data prior to the age of 20 (for the younger cohort), and exclusion of individuals who had already developed T2D at baseline. More minor limitations include that the younger cohort was not followed into mid-life (where higher rate of incidence is anticipated), sample selection erred towards older and female participants within the population representative PATH study (possibly limiting generalizability).

In conclusion, our findings indicate that mid-life may be a period of heightened vulnerability to the effects of an unhealthy diet on diabetes risk, but current findings are imprecise, and attenuated when risk factors related to diet, such as adiposity, are taken into account.

Acknowledgements The authors are grateful to Anthony Jorm, Helen Christensen, Bryan Rogers, Keith Dear, Simon Easteal, Chantal Reglade Meslin, Jerome Maller, Patricia Jacomb, Karen Maxwell, Kristine Kuh and the PATH project interviewers.

Funding This work was supported the National Health and Medical Research Council [grant numbers 973302, 179805, 157125, 1063907 and 1100579]. At the time of writing, NC's Fellowship was funded by the Australian Research Council [grant number 120100227]. PB's Research Fellowship was funded by the Australian Research Council [FT13101444]], KJA's NHMRC Fellowship #1102694. FNJ was supported by an NHMRC Career Development Fellowship (2) [1108125].

Author contributions EW contributed to the design of the study, provided methodological input, conducted the statistical analyses and contributed to all aspects of manuscript preparation and submission. FNJ and PB generated the dietary patterns, provided methodological input and contributed to writing and editing of the manuscript. KJA contributed to the design of the study, provided methodological input and contributed to writing and editing of the manuscript. NC contributed to the design of the study, provided methodological input and theoretical expertise and contributed to all aspects of manuscript preparation and submission.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval This study was conducted according to the guidelines laid down in the Declaration of Helsinki and all procedures involving human subjects/patients were approved by the Australian National University Human Research Ethics Committee. Written informed consent was obtained from all participants.

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